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Post-traumatic stress disorder symptom remission and cognition in a large cohort of civilian women‡

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Abstract

Background. Post-traumatic stress disorder (PTSD) is associated with cognitive impairments. It is unclear whether problems persist after PTSD symptoms remit.

Methods. Data came from 12 270 trauma-exposed women in the Nurses' Health Study II. Trauma and PTSD symptoms were assessed using validated scales to determine PTSD status as of 2008 (trauma/no PTSD, remitted PTSD, unresolved PTSD) and symptom severity (lifetime and past-month). Starting in 2014, cognitive function was assessed using the Cogstate Brief Battery every 6 or 12 months for up to 24 months. PTSD associations with baseline cognition and longitudinal cognitive changes were estimated by covariate-adjusted linear regression and linear mixed-effects models, respectively.

Results. Compared to women with trauma/no PTSD, women with remitted PTSD symptoms had a similar cognitive function at baseline, while women with unresolved PTSD symptoms had worse psychomotor speed/attention and learning/working memory. In women with unresolved PTSD symptoms, past-month PTSD symptom severity was inversely associated with baseline cognition. Over follow-up, both women with remitted and unresolved PTSD symptoms in 2008, especially those with high levels of symptoms, had a faster decline in learning/working memory than women with trauma/no PTSD. In women with remitted PTSD symptoms, higher lifetime PTSD symptom severity was associated with a faster decline in learning/working memory. Results were robust to the adjustment for sociodemographic, biobehavioral, and health factors and were partially attenuated when adjusted for depression.

Conclusion. Unresolved but not remitted PTSD was associated with worse cognitive function assessed six years later. Accelerated cognitive decline was observed among women with either unresolved or remitted PTSD symptoms.

Introduction

Post-traumatic stress disorder (PTSD) is a debilitating mental disorder that can occur in people who have experienced traumatic events such as sexual assault, natural disasters, or terrorist attacks. The lifetime prevalence of PTSD is estimated to be 3.9% globally and 6.8% in the United States (U.S.), and it is approximately twice as common in women than men (Kessler et al., 2005; Koenen et al., 2017). Compared to men, women with PTSD tend to be exposed to traumatic events at a younger age, may experience more severe symptoms, and are more likely to have comorbid mental disorders, such as depression and anxiety disorders (Olff, 2017; Olff, Langeland, Draijer, & Gersons, 2007b).

Accumulating evidence has linked PTSD with an increased risk of developing various chronic diseases in both men and women, including cardiovascular diseases (CVD) (Dyball, Evans, Boos, Stevelink, & Fear, 2019; Kubzansky, Koenen, Spiro, Vokonas, & Sparrow, 2007; Sumner et al., 2015), type 2 diabetes (Roberts et al., 2015; Vancampfort et al., 2016), and dementia (Desmarais et al., 2020), as well as all-cause mortality (Boscarino, 2006; Roberts, Kubzansky, Chibnik, Rimm, & Koenen, 2020a). There is also a growing body of literature linking PTSD with poor cognitive function and accelerated cognitive aging, including

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our previous research in the Nurses' Health Study II (NHS II) (Clouston et al., 2019; Greenberg, Tanev, Marin, & Pitman, 2014; Lawn et al., 2022; Roberts et al., 2022; Rosen et al., 2022; Schuitevoerder et al., 2013; Scott et al., 2015; Sumner et al., 2017).

While some individuals experience chronic or recurrent PTSD symptoms, others' symptoms may resolve over time, with or without treatment (Galatzer-Levy et al., 2013; Steinert, Hofmann, Leichsenring, & Kruse, 2015). A meta-analysis of 42 prospective studies on the natural course of PTSD found that, on average, nearly half (44%) of patients with PTSD experienced remission after three or more years from their initial assessment (Morina, Wicherts, Lobbrecht, & Priebe, 2014). However, the estimated remission rate largely varied across studies, likely due to heterogeneity in study design, including the time between the traumatic event and the first assessment, follow-up length and frequency, PTSD assessment (interview-based or self-reported), the definition of remission, types of traumatic events, and whether the participants sought for or received treatment (Morina et al., 2014; Steinert et al., 2015).

While PTSD is linked to many adverse health outcomes, much less is known about whether the elevated health risks persist after PTSD symptoms resolve. A few studies have investigated health trajectories after PTSD remission, with mixed results across outcomes. For example, in previous research, we found that women with remitted PTSD symptoms did not show significant differences in the risk of CVD compared to individuals with no trauma exposure, while elevated CVD risks were found in those with ongoing PTSD symptoms ($N = 49\,859$) (Gilsanz et al., 2017). That study also found a positive association between PTSD symptom duration and CVD incidence (Gilsanz et al., 2017). Studies in Veterans Health Affairs (VHA) patients ($N = 979$–1598) found that clinically meaningful reductions in PTSD symptoms were associated with a lower risk of incident type 2 diabetes (Scherrer et al., 2019) and hypertension (Scherrer et al., 2020a). Small prospective psychotherapy studies in patients with PTSD ($N = 37$–235) reported improvements in quality of life (Schnurr & Lunney, 2016), sleep quality (Rousseau et al., 2021), and blood pressure (Schubert et al., 2019) as PTSD symptoms reduced and/or remitted. However, some evidence suggests that individuals may still experience adverse health events after PTSD remission. For example, a study of 25 patients with PTSD treated with integrative trauma-focused cognitive behavioral therapy found that hypothalamic–pituitary–adrenal (HPA) axis feedback sensitivity remained unchanged after PTSD symptom improvement (Schubert et al., 2019); another study in 1034 traumatically injured patients found that patients with resolved PTSD symptoms had poorer quality of life in various domains compared to those who never developed PTSD (Bryant et al., 2016). In addition, VHA studies ($N = 1079$–1330) found that patients with a clinically meaningful reduction in PTSD symptoms within 12 months of the initial assessment were no different from those with less or no symptom reduction with regard to subsequent risks of CVD, ischemic heart disease, hyperlipidemia, and weight loss over a 2–7 year follow-up (Scherrer et al., 2020a, 2020b).

It remains unclear whether and to what extent cognitive deficits ameliorate with the remission of PTSD symptoms, with findings inconsistent across studies and cognitive measures (Ben-Zion et al., 2018; Clouston et al., 2016; Eren-Koçak, Kılıç, Aydın, & Hızlı, 2009; Fani et al., 2009; Haaland, Sadek, Keller, & Castillo, 2016; Nijdam, Martens, Reitsma, Gersons, & Olff, 2018; Vermetten, Vythilingam, Southwick, Charney, & Bremner, 2003; Walter, Palmieri, & Gunstad, 2010). One study of 813 World Trade Center responders found that current but not remitted PTSD was associated with cognitive impairment (Clouston et al., 2016). However, other studies have been limited by small samples ($N < 100$) of relatively younger individuals and failure to control adequately for potential confounders, such as age and education. In addition, most studies were conducted in clinical settings and assessed cognitive change in relation to PTSD symptom improvement over the course of treatment, thus providing limited information on how post-remission cognition compares to that of individuals who never had PTSD after trauma exposure.

The present study leverages the NHS II, a large prospective cohort of U.S. women, to examine how prior endorsement of PTSD symptoms, remitted or unresolved, are associated with cognitive function and changes over time measured six years after the PTSD assessment. We hypothesized that (a) unresolved PTSD symptoms would be associated with poorer cognitive function and worse changes in cognitive function over a 2-year follow-up compared to those who never had PTSD after trauma exposure, and (b) women whose PTSD symptoms remitted would have similar cognitive function and cognitive changes as women who did not have PTSD after trauma exposure.

Methods

Study participants

Data came from 116 429 U.S. female nurses enrolled in the NHS II in 1989, with biennial follow-up ongoing. Women responded to biennial questionnaires about lifestyle and health. In 2008, 60 804 women who returned a 2007 biennial questionnaire were invited to complete a supplemental questionnaire on trauma exposure and PTSD history; of these, 54 224 (89%) responded. In 2014, 43 957 of these responders with known email addresses were invited to an initial (henceforth 'baseline') cognitive assessment. A total of 15 138 women completed this assessment, with 14 994 passing data quality checks as previously described (Roberts et al., 2022). We excluded 2724 women who reported no trauma exposure, a requirement for PTSD and subsequent remission, leaving 12 270 participants.

Following the baseline cognitive assessment, women were asked to complete additional cognitive assessments every 6 or 12 months for up to 24 months. Those who did not complete a cognitive assessment at one time point were not invited to the next session. Thus, women completed up to five assessments; over a median follow-up of 1.0 year, 7937 (64.7%) completed at least two assessments, with 625 (5.1%) completing all five. The study was approved by the Institutional Review Board of Brigham and Women's Hospital. Completion of assessments implied consent.

Trauma and PTSD assessment

In 2008, women reported lifetime exposure to 15 traumatic events and any other traumatic event not listed from a modified version of the Brief Trauma Questionnaire (Morgan et al., 2001; Schnurr, Vielhauer, Weathers, & Findler, 1999). Trauma-exposed women were asked to indicate their worst event. Lifetime (ever) and past-month experience of seven PTSD symptoms with reference to their worst trauma was assessed using the Short Screening Scale for DSM-IV PTSD (Breslau, Peterson, Kessler, & Schultz, 1999). PTSD symptom severity (lifetime and past-month) was defined

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as the number of symptoms reported. See online Supplementary Materials for more details.

We defined remission as reporting lifetime but no past-month PTSD symptoms; women who reported past-month symptoms were identified as having unresolved symptoms at the time of the assessment. Trauma-exposed women were classified into three groups indicating PTSD status as of 2008: (a) no PTSD: trauma exposed but no PTSD symptoms at any point in their lifetime, (b) remitted PTSD: any lifetime symptoms, but no past-month PTSD symptoms, and (3) unresolved PTSD: any past-month PTSD symptoms. In addition, we utilized a cutoff score of four on the PTSD screener to create more granular categories that incorporate symptom severity. In a validation study of this screener, a score of ⩾4 identified PTSD cases with sensitivity = 85% and specificity = 93% (Breslau et al., 1999). Based on this cut-off, we further classified trauma-exposed women into five groups: (a) trauma/no PTSD symptoms, (b) remitted mild (1–3) PTSD symptoms, (c) remitted moderate-severe (4–7) PTSD symptoms, (d) unresolved mild (1–3) PTSD symptoms, (e) unresolved moderate-severe (4–7) PTSD symptoms.

Cognitive assessment

The Cogstate Brief Battery was used to assess cognitive function beginning in 2014. Cogstate is a self-administered online tool comprised of four tasks measuring psychomotor speed, attention, visual learning, and working memory (Fredrickson et al., 2010; Koyama et al., 2015). This computerized instrument has good construct and criterion validity (Hammers et al., 2012; Maruff et al., 2009), test-retest reliability (Fredrickson et al., 2010; Lim et al., 2013a; Maruff et al., 2013), acceptability and efficiency in large community-based studies of older individuals (Fredrickson et al., 2010; Koyama et al., 2015), and clinical utility in identifying cognitive impairments and dementia (Hammers et al., 2012; Lim et al., 2012; Maruff et al., 2013, 2009). Standardized z scores were calculated at all assessments using means and standard deviations at baseline for each task. Two composite scores were generated and analyzed as primary outcomes: psychomotor speed/attention composite and learning/working memory composite. For both composites, higher scores reflect better cognitive function. These two composites have been previously validated in our sample using confirmatory factor analysis (Sumner et al., 2017) and have been found to be sensitive measures of cognitive decline (Lim et al., 2013b; Maruff et al., 2013). See online Supplementary Materials for more details.

Covariates

Analyses were adjusted for potential confounding factors for the association between PTSD status and cognition, including sociodemographic factors, biobehavioral factors, and health conditions. These included age at Cogstate baseline, self-identified race/ethnic identity (Non-Hispanic White, Black, Hispanic, Asian, other), parental education at participant's birth (high school or less, some college, 4 + years of college), and participant's highest education (associate's, bachelor's, master's, doctorate). Biobehavioral factors included body mass index [BMI, kg/m^2, calculated from self-reported height and weight (Rimm et al., 1990)], cigarette smoking (nonsmoker, former smoker, current smoker of 1–14, 15–24, or 25 + cigarettes/day), alcohol consumption (0, 1 − <5, 5 − <10, 10 − 20, 20 + grams/day), diet quality measured using the Alternative Healthy Eating Index (excluding alcohol consumption) (Chiuve et al., 2012), ascertained in 2007, and physical activity (<3, 3 − <9, 9 − <18, 18 − <27, 27 + metabolic equivalent hours/week), ascertained in 2005. Lifetime history of physician-diagnosed hypertension, type 2 diabetes, stroke, and myocardial infarction was ascertained based on women's reports on biennial questionnaires from 1989–2007. While the assessment of biobehavioral factors and health conditions predated the PTSD assessment in 2008, some of these factors may be on the pathway between PTSD remission and cognition, as the exact timing of PTSD remission is not known. For example, good diet quality may positively impact the remission of PTSD symptoms, or it may be the result of PTSD remission. It was not possible to determine the sequence of PTSD remission and these health factors; thus, we cannot conclusively distinguish if they are serving as confounders or as mediators between the PTSD status in 2008 and cognition assessed six years later.

Past-week depressive symptoms and history of physician-diagnosed depression were analyzed in a secondary analysis, as they could be a cause or consequence of unresolved PTSD. Past-week depressive symptoms were assessed in 2008 using the 10-item Center for Epidemiologic Studies Depression (CES-D-10) scale. The CES-D-10 has excellent psychometrics and performs similarly to the 20-item CES-D (Andresen, Malmgren, Carter, & Patrick, 1994). History of physician-diagnosed depression was ascertained from biennial questionnaires in 2003–2007.

Less than 5.0% of all covariates were missing, except for participant's education (24.4%), which was assessed by a supplemental 2018 questionnaire in a subset of participants. We addressed missing values in all covariates using multiple imputations (MI) with a fully conditional specification method (Brand, 1999; van Buuren, 2007), generating 10 imputed datasets. See online Supplementary Materials for more details.

Statistical analysis

For descriptive analyses, we summarized sociodemographic and health characteristics across PTSD groups using means and standard deviations and frequencies where appropriate. To comprehensively examine the relationship between PTSD status, symptom severity, and cognition, we modeled PTSD in three different ways in the association analysis. Firstly, we used the 3-level categorical variable to compare remitted and unresolved PTSD with those who had no PTSD ever (reference group). Secondly, we used a continuous score of lifetime and past-month PTSD symptoms to assess the linear relationship between symptom severity and cognition among women with remitted and unresolved PTSD symptoms, respectively. Thirdly, we utilized a 5-level categorical variable that combined both PTSD status and symptom severity, with trauma/no PTSD symptoms as the reference group.

To estimate the association between PTSD in 2008 and baseline cognition in 2014 (henceforth 'cross-sectional analysis,' with prospective measures of outcome), we used multivariate linear regression (PROC GLM in SAS v9.4). The beta coefficients for PTSD were reported, representing the estimated differences in baseline cognition compared to the reference group or per one unit increase in symptom severity. We also estimated cognitive differences associated with one year of aging by fitting linear regressions on cognitive composites with age as the predictor, adjusted for race/ethnicity (Roberts et al., 2020b; Sumner et al., 2017). To estimate the association between PTSD in 2008 and the rate of cognitive change during follow-up (henceforth 'longitudinal analysis'), we used linear mixed-effects models (PROC MIXED in

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SAS v9.4), including time since baseline in years, PTSD, the interaction between time and PTSD, and covariates. Dependence between repeated measurements within study participants was accounted for via the inclusion of random intercepts and random slopes for time. The beta coefficients for the time-PTSD interaction terms were reported, representing the estimated differences in the rate of cognitive change compared to the reference group or per one unit increase in symptom severity. These models were fit to each of the 10 imputed datasets, with results combined using Rubin's rule (Rubin, 1987). Estimated associations with a p value less than 0.05 were considered statistically significant.

In both cross-sectional and longitudinal analyses, we started with a crude model with no covariates. Next, we included covariates in increasingly adjusted models. In the first adjusted model, we accounted for possible sociodemographic confounders (e.g. parental education). In the second adjusted model, we examined the impact of biobehavioral factors (e.g. BMI, smoking) on a possible association of PTSD status with cognition. Finally, to assess the impact of co-morbidities on the association, the third model was additionally adjusted for health conditions (e.g. diabetes, stroke).

We conducted several secondary analyses. First, since PTSD and depression are highly co-morbid (Rytwinski, Scur, Feeny, & Youngstrom, 2013), we further adjusted for depressive symptoms in 2008 and history of depression during 2003–2007 to examine if a PTSD-cognition association was accounted for by depression. Next, to evaluate the potential impact of practice effects in the cognitive tasks in the longitudinal analysis, we further adjusted for prior practice (Vivot et al., 2016). Moreover, to evaluate whether results were affected by the imputation of missing values, particularly participant's education, we explored several alternative imputation methods (e.g. single imputation, hot-deck imputation) and compared results. Finally, to examine potential bias due to selection into the Cogstate sample and loss to follow-up, we conducted inverse probability weighted (IPW) analyses. The 5-level categorical PTSD variable that incorporated both PTSD status and symptom severity was assessed in secondary analyses. See online Supplementary Materials for more details.

Results

Participant characteristics and PTSD status

A total of 12 270 trauma-exposed women were included in this study. Most were non-Hispanic White (95.9%), with age at baseline cognitive assessment ranging from 50 to 71 years (mean = 61). The prevalence of any PTSD symptoms was 67.3% (N = 8253); 2997 (24.4%) and 784 (6.4%) women had unresolved mild and moderate-severe symptoms at the time of PTSD assessment in 2008, respectively; 3347 (27.3%) and 1125 (9.2%) women reported remitted mild and moderate-severe symptoms, respectively. Participant characteristics by PTSD status are shown in Table 1. Compared to women whose PTSD symptoms had remitted as of 2008, those with unresolved PTSD symptoms had lower levels of parental education, had higher BMI, were more likely to be current smokers, and had higher levels of depressive symptoms. The distribution of trauma types and lifetime PTSD symptoms by PTSD status is shown in online Supplementary Table S1.

Cross-sectional analysis

Compared to trauma/no PTSD symptoms, unresolved PTSD symptoms at the time of 2008 assessment were associated with significantly lower scores in both cognitive composites assessed six years later. Specifically, in analyses without adjustment, women with any unresolved PTSD symptoms showed mean differences of −0.10 standard deviation (S.D.) (95% CI −0.14 to −0.06) in psychomotor speed/attention and −0.10 S.D. (95% CI −0.13 to −0.06) in learning/working memory, compared to women with no PTSD symptoms ever (online Supplementary Table S2). Among women with unresolved symptoms, one unit increase in past-month PTSD symptom severity was associated with −0.02 (95% CI −0.04 to 0.001) and −0.04 S.D. (95% CI −0.05 to −0.02) mean differences in psychomotor speed/attention and learning/working memory, respectively (Table 2). Using a cutoff of four, we found mean differences between women with unresolved mild (1–3) PTSD symptoms and women with trauma/no PTSD symptoms of −0.09 S.D. (95% CI −0.13 to −0.05) in psychomotor speed/attention and −0.07 S.D. (95% CI −0.11 to −0.04) in learning/working memory; while for women with unresolved moderate-severe (4–7) PTSD symptoms, these mean differences were −0.14 S.D. (95% CI −0.21 to −0.07) in psychomotor speed/attention and −0.18 S.D. (95% CI −0.23 to −0.12) in learning/working memory (Table 3, online Supplementary Fig. S1). To help interpret these differences, we found that each additional year of age was associated with a −0.04 S.D. decrease in psychomotor speed/attention and a −0.03 S.D. decrease in learning/working memory; thus, unresolved mild and moderate-severe PTSD symptoms were associated with the equivalent of approximately two and five years of cognitive aging, respectively.

We observed no significant difference in baseline cognition between women with remitted PTSD symptoms as of 2008 compared to the women with no PTSD symptoms ever (online Supplementary Table S2, Table 3). Among women with remitted PTSD symptoms, increase in lifetime PTSD symptom severity was not associated with baseline cognition (Table 2). Above results on unresolved and remitted PTSD were unchanged after adjusting for sociodemographic and biobehavioral factors (Tables 2, 3, online Supplementary Table S2) and health conditions (data not shown).

Longitudinal analysis

Compared to women with no PTSD symptoms, worse rates of changes in learning/working memory were observed in both women with unresolved PTSD (mean difference = −0.03 S.D./year, 95% CI −0.06 to −0.01) and remitted PTSD (mean difference = −0.02 S.D./year, 95% CI −0.05 to 0.0005) (online Supplementary Table S3). Specifically, unresolved mild (1–3) symptoms at the time of PTSD assessment had a marginally significant association with worse rates of changes in learning/working memory (mean difference = −0.03 S.D./year, 95% CI −0.05 to 0.0004) that further reached statistical significance when adjusted for sociodemographic and biobehavioral factors (Table 4). In addition, unresolved moderate-severe (4–7) PTSD symptoms were significantly associated with worse rates of changes in learning/working memory (mean difference = −0.05 S.D./year, 95% CI −0.09 to −0.01), and the results remained unchanged with the adjustment of covariates (Table 4, online Supplementary Fig. S1). Among women with unresolved PTSD symptoms, increase in past-month symptom severity was not linearly associated with the rate of change in learning/working memory (online Supplementary Table S4).

By contrast, we observed a significant linear association between increase in lifetime symptom severity and changes in learning/working memory among women with remitted PTSD

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Table 1. Participant characteristics by PTSD status ($N = 12\,270$)

	No PTSD sx ($n = 4017$)	Remitted 1–3 PTSD sx ($n = 3347$)	Remitted 4–7 PTSD sx ($n = 1125$)	Unresolved 1–3 PTSD sx ($n = 2997$)	Unresolved 4–7 PTSD sx ($n = 784$)
Age at baseline (years)	61.2 (4.6)	60.9 (4.6)	60.7 (4.6)	61.1 (4.5)	61.1 (4.4)
Age at worst trauma (years)	28.8 (12.4)	29.2 (13.9)	29.1 (13.5)	30.9 (16.1)	32.4 (17.6)
Years since worst trauma	25.2 (12.8)	24.5 (14.1)	24.5 (13.9)	23.1 (16.1)	21.5 (17.7)
Follow-up length (months)	10.3 (9.1)	10.7 (9.4)	10.8 (9.7)	10.8 (9.6)	10.4 (9.5)
Racial identity, Non-Hispanic White % (*n*)	95.8 (3803)	96.4 (3194)	96.1 (1067)	95.6 (2839)	94.6 (737)
Parental education, % (*n*)					
High school	51.0 (2005)	47.2 (1542)	45.7 (500)	50.8 (1485)	51.5 (393)
Some college	25.4 (997)	26.4 (864)	23.7 (260)	24.9 (728)	24.4 (186)
College plus	23.6 (927)	26.4 (864)	30.6 (335)	24.4 (713)	24.1 (184)
Participant education, % (*n*)					
Associate's	24.7 (752)	20.5 (527)	19.3 (163)	23.5 (530)	25.1 (143)
Bachelor's	40.1 (1218)	41.7 (1071)	39.7 (336)	40.0 (901)	37.8 (215)
Master's	29.9 (909)	33.2 (853)	33.8 (286)	30.9 (697)	31.3 (178)
Doctorate	5.3 (160)	4.7 (120)	7.2 (61)	5.6 (126)	5.8 (33)
Body mass index (kg/m^2)	27.2 (6.1)	27 (6)	27.3 (6.4)	27.5 (6.5)	28.5 (6.8)
Smoking status, % (*n*)					
Never	66.9 (2684)	67.6 (2261)	62.8 (706)	63.5 (1901)	57.9 (454)
Past	28.5 (1144)	28.5 (952)	32.4 (364)	31.0 (930)	34.3 (269)
Current	4.6 (186)	3.9 (131)	4.8 (54)	5.5 (165)	7.8 (61)
Alcohol intake, % (*n*)					
None	31.0 (1190)	30.9 (995)	33.6 (359)	30.6 (868)	39.0 (286)
0–20 g/day	62.0 (2377)	61.4 (1974)	59.1 (632)	61.7 (1746)	54.9 (403)
20+ g/day	7.0 (267)	7.7 (246)	7.3 (78)	7.7 (218)	6.1 (45)
Physical activity (MET h/week)	23.6 (27.6)	24.6 (29.5)	25.1 (30.6)	23.3 (27.3)	24.5 (28.9)
Diet quality on the Alternate Healthy Eating Index[a]	57.6 (11.8)	58.7 (12)	59.2 (11.9)	58.4 (11.9)	58.4 (13)
Depressive symptoms (CES-D)[b]	4.4 (3.9)	4.7 (3.8)	5.4 (4.5)	8.1 (4.8)	13.7 (5.9)
Diagnosed depression[c], % (*n*)	13.4 (540)	17.1 (573)	30.6 (344)	33.3 (999)	53.6 (420)
Hypertension[d], % (*n*)	27.3 (1095)	26.7 (892)	26.8 (301)	32.5 (975)	33.9 (266)
Diabetes[d], % (*n*)	4.2 (168)	4.4 (147)	4.2 (47)	4.5 (136)	6.0 (47)
Myocardial infarction[d], % (*n*)	0.6 (24)	0.8 (27)	0.8 (9)	1.7 (52)	2.7 (21)
Stroke[d], % (*n*)	0.9 (35)	0.8 (28)	1.2 (14)	1.3 (38)	1.8 (14)

MET, metabolic equivalent of tasks; sx, symptoms.
Values are means (s.d.) for continuous variables or percentages (N) for categorical variables. Values of categorical variables may not sum to 100% due to rounding or missing data.
[a]Alternate Healthy Eating Index without alcohol consumption was used in the analysis; higher scores reflect better diet quality (possible range = 0–100).
[b]Center for Epidemiologic Studies Depression (CES-D) scale, short form (possible range = 0–30).
[c]History of clinician-diagnosed depression reported at the 2003–2013 questionnaires.
[d]History of clinician-diagnosed health conditions reported at the 1989–2013 questionnaires.

symptoms (online Supplementary Table S4). Specifically, one unit increase in lifetime symptom severity was associated with a mean difference of −0.01 s.d./year (95% CI −0.02 to −0.004) in the rate of change in learning/working memory. Using a cutoff of four, we observed significantly worse cognitive changes in learning/working memory for women with remitted moderate-severe (4–7) PTSD symptoms (−0.06 s.d./year, 95% CI −0.10 to −0.03) compared to women with no PTSD symptoms, but not with remitted mild (1–3) PTSD symptoms (−0.01 s.d./year, 95% CI −0.03 to 0.02) (Table 4, online Supplementary Fig. S1). No significant association was observed between PTSD status or symptom severity and psychomotor speed/attention (Table 4, online Supplementary Tables S3, S4). Results were unchanged after controlling for sociodemographic and biobehavioral factors (Table 4,

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Table 2. Association between PTSD symptom severity and Cogstate composite scores at baseline

	Model 0		Model 1		Model 2	
	b (95% CI)	*p*	*b* (95% CI)	*p*	*b* (95% CI)	*p*
Remitted PTSD symptoms ($n = 4472$)[a]						
Psychomotor speed/attention	0.002 (−0.01 to 0.02)	0.83	0.000 (−0.02 to 0.02)	>0.99	−0.002 (−0.02 to 0.02)	0.98
Learning/working memory	−0.002 (−0.02 to 0.01)	0.75	−0.01 (−0.02 to 0.01)	0.37	−0.005 (−0.02 to 0.01)	0.48
Unresolved PTSD symptoms ($n = 3781$)[b]						
Psychomotor speed/attention	−0.02 (−0.04 to 0.001)	0.06	−0.02 (−0.04 to 0.004)	0.06	−0.02 (−0.04 to 0.003)	0.10
Learning/working memory	−0.04 (−0.05 to −0.02)	<0.01	−0.04 (−0.05 to −0.02)	<0.01	−0.03 (−0.05 to −0.02)	<0.01

Beta coefficients of PTSD groups from linear regression models are shown, representing the difference in baseline Cogstate composites for every unit increase in symptom severity.
Model 0: no covariates.
Model 1: age at baseline cognitive assessment, race/ethnicity, parental education, participant education.
Model 2: Model 1 + body mass index, smoking status, alcohol use, physical activity, diet quality.
[a]Analysis was restricted to women with remitted PTSD symptoms as of 2008. Lifetime PTSD symptom severity was modeled as the predictor.
[b]Analysis was restricted to women with unresolved PTSD symptoms as of 2008. Past-month PTSD symptom severity was modeled as the predictor.

online Supplementary Tables S3, S4) and health conditions (data not shown).

Secondary analyses

Adjustment for depression

In the cross-sectional analysis, the estimated decrease in cognitive composite scores for women with unresolved PTSD symptoms, compared to those with no PTSD, remained statistically significant after adjusting for depression, but estimates were attenuated by 25.0 to 53.8%, depending on the model (online Supplementary Table S5). In the longitudinal analysis, including the main effects of depression did not change the results (online Supplementary Table S6). However, when further adjusted for time × depression interactions, the associations of unresolved PTSD symptoms with changes in learning/working memory attenuated by 22.5 to 29.5% and were no longer statistically significant. The association of remitted moderate-severe PTSD symptoms with the rate of change in cognition was similar to that in the unadjusted analyses (online Supplementary Table S6).

Adjustment for practice effects

Longitudinal results were consistent after adjusting for practice effects and were robust to the different parameterization of practice effects, including the categorical and the continuous number of prior tests, the square root of the number of prior tests, and the indicator for baseline *v.* post-baseline assessments (online Supplementary Table S7). Cognitive composite scores decreased over time in all PTSD groups after adjusting for the number of prior tests (categorical) (Fig. 1); the estimated rate of decline in

Table 3. Association between PTSD status (5 levels) and Cogstate composite scores at baseline

	Model 0		Model 1		Model 2	
	b (95% CI)	*p*	*b* (95% CI)	*p*	*b* (95% CI)	*p*
Psychomotor speed/attention ($n = 12\,248$)						
No PTSD sx	Ref	Ref	Ref	Ref	Ref	Ref
Remitted 1–3 PTSD sx	−0.02 (−0.07 to 0.02)	0.27	−0.04 (−0.08 to 0.01)	0.09	−0.04 (−0.08 to 0.01)	0.09
Remitted 4–7 PTSD sx	−0.02 (−0.08 to 0.04)	0.52	−0.04 (−0.10 to 0.02)	0.24	−0.04 (−0.09 to 0.02)	0.25
Unresolved 1–3 PTSD sx	−0.09 (−0.13 to −0.05)	<0.01	−0.10 (−0.14 to −0.05)	<0.01	−0.09 (−0.14 to −0.05)	<0.01
Unresolved 4–7 PTSD sx	−0.14 (−0.21 to −0.07)	<0.01	−0.14 (−0.21 to −0.07)	<0.01	−0.14 (−0.21 to −0.07)	<0.01
Learning/working memory ($n = 12\,263$)						
No PTSD sx	Ref	Ref	Ref	Ref	Ref	Ref
Remitted 1–3 PTSD sx	0.02 (−0.01 to 0.05)	0.27	0.01 (−0.03 to 0.04)	0.69	0.01 (−0.03 to 0.04)	0.68
Remitted 4–7 PTSD sx	0.0002 (−0.05 to 0.05)	0.99	−0.02 (−0.07 to 0.03)	0.40	−0.02 (−0.06 to 0.03)	0.53
Unresolved 1–3 PTSD sx	−0.07 (−0.11 to −0.04)	<0.01	−0.08 (−0.11 to −0.04)	<0.01	−0.07 (−0.11 to −0.04)	<0.01
Unresolved 4–7 PTSD sx	−0.18 (−0.23 to −0.12)	<0.01	−0.18 (−0.24 to −0.13)	<0.01	−0.17 (−0.23 to −0.12)	<0.01

Beta coefficients of PTSD groups from linear regression models are shown, representing the difference in baseline Cogstate composites compared to trauma-exposed women with no PTSD.
sx, symptoms.
Model 0: no covariates.
Model 1: age at baseline cognitive assessment, race/ethnicity, parental education, participant education.
Model 2: Model 1 + body mass index, smoking status, alcohol use, physical activity, diet quality.

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Table 4. Association between PTSD status (5 levels) and rate of change in Cogstate composite scores

	Model 0		Model 1		Model 2	
	b (95% CI)	*p*	*b* (95% CI)	*p*	*b* (95% CI)	*p*
Psychomotor speed/attention (*n* = 12 248)						
No PTSD sx	Ref	Ref	Ref	Ref	Ref	Ref
Remitted 1–3 PTSD sx	−0.02 (−0.05 to 0.01)	0.23	−0.02 (−0.05 to 0.01)	0.21	−0.02 (−0.05 to 0.01)	0.21
Remitted 4–7 PTSD sx	0.01 (−0.03 to 0.05)	0.70	0.01 (−0.03 to 0.05)	0.71	0.01 (−0.03 to 0.05)	0.71
Unresolved 1–3 PTSD sx	−0.01 (−0.04 to 0.02)	0.41	−0.01 (−0.04 to 0.02)	0.36	−0.01 (−0.04 to 0.02)	0.36
Unresolved 4–7 PTSD sx	−0.01 (−0.06 to 0.03)	0.55	−0.02 (−0.06 to 0.03)	0.50	−0.02 (−0.06 to 0.03)	0.49
Learning/working memory (*n* = 12 263)						
No PTSD sx	Ref	Ref	Ref	Ref	Ref	Ref
Remitted 1–3 PTSD sx	−0.01 (−0.03 to 0.02)	0.47	−0.01 (−0.04 to 0.01)	0.40	−0.01 (−0.04 to 0.01)	0.40
Remitted 4–7 PTSD sx	−0.06 (−0.10 to −0.03)	<0.01	−0.06 (−0.10 to −0.03)	<0.01	−0.06 (−0.10 to −0.03)	<0.01
Unresolved 1–3 PTSD sx	−0.03 (−0.05 to 0.0004)	0.05	−0.03 (−0.05 to −0.00)	0.04	−0.03 (−0.05 to −0.00)	0.04
Unresolved 4–7 PTSD sx	−0.05 (−0.09 to −0.01)	0.02	−0.05 (−0.09 to −0.01)	0.01	−0.05 (−0.09 to −0.01)	0.01

Beta coefficients of the time × PTSD interaction terms from the linear mixed-effects models are shown, representing the difference in 1-year change in Cogstate composites compared to trauma-exposed women with no PTSD. sx, symptoms.
Model 0: no covariates.
Model 1: age at baseline cognitive assessment, race/ethnicity, parental education, participant education.
Model 2: Model 1 + body mass index, smoking status, alcohol use, physical activity, diet quality.

learning/working memory was 0.07 S.D./year among women with trauma/no PTSD, and nearly twice as fast among women with remitted or unresolved moderate-severe PTSD symptoms (0.13 S.D./year and 0.12 S.D./year respectively).

Alternative imputation of participant education

Results were consistent across different imputation methods, including group-specific mode, hot-deck imputation, missing indicator, and single imputation (online Supplementary Tables S8, S9). As single imputation results were similar to MI, we conducted the following IPW analyses in one of the imputed datasets.

Selection into Cogstate and IPW

Women in this study were similar to those in the original PTSD cohort ($N = 44\,573$) from which the cognitive samples were drawn in many characteristics, although the former had slightly better health behaviors and fewer diseases (online Supplementary Table S10). Accounting for the probability of selection from the original PTSD cohort into the Cogstate sample using IPW, overall results remained the same except for a mild attenuation in the association between unresolved mild PTSD symptoms and the rate of change in learning/working memory in the longitudinal analysis (−0.02 S.D./year, 95% CI −0.05 to 0.00, online Supplementary Table S11).

Loss to follow-up during Cogstate assessments and IPW

During the longitudinal assessment period, women who continued to the next assessment had higher cognition on average than those who did not respond to the next assessment, particularly between the first and the second assessments and in learning/working memory (online Supplementary Fig. S2). PTSD

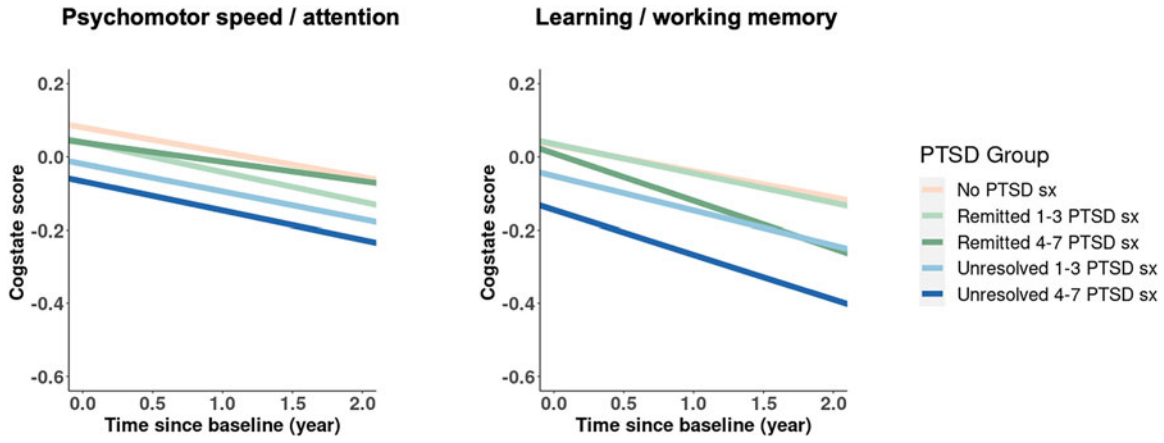

Figure 1. Fitted linear cognitive trajectories by PTSD status (5 levels) in 2008. Plots were based on results from models adjusted for age at baseline cognitive assessment, race/ethnicity, parental education, participant education, and categorical indicator of the number of prior cognitive tests. Fitted linear trajectories were calculated for Non-Hispanic White women ages 61 at baseline, with the highest education of Bachelor's degree and parental education of high school. sx, symptoms.

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status was not associated with the probability of dropout (online Supplementary Fig. S3). Sample characteristics were similar across women who completed different numbers of cognitive assessments (online Supplementary Table S12). After accounting for loss to follow-up using IPW, the difference in the change of learning/working memory composite compared to women with trauma/no PTSD remained significant for women with remitted or unresolved moderate-severe PTSD symptoms; however, it was attenuated and no longer significant for women with unresolved mild PTSD symptoms ($p = 0.14$, online Supplementary Table S13).

Discussion

In this cohort study, unresolved PTSD symptoms as of 2008 were associated with significantly poorer cognitive function at baseline in 2014 in psychomotor speed/attention and learning/working memory and faster cognitive decline during follow-up in learning/working memory, compared to the reference group of women with trauma/no PTSD. In women whose PTSD remitted by 2008, while we found no significant difference in baseline cognition compared to women with trauma/no PTSD, increase in lifetime PTSD symptom severity was significantly associated with a faster decline in learning/working memory. The observed associations were independent of sociodemographic, biobehavioral, and health factors, and were robust to adjustment for practice effects, imputation of missing values, sample selection, and loss to follow-up. Co-morbid depression partially accounted for the associations between PTSD and cognition, potentially due to the overlapping symptoms between the two conditions. Our findings add to the literature on the cognitive sequelae of PTSD and provide a more granular evaluation of the dynamic association between PTSD and cognition.

Similar to findings from previous research in mixed-gender samples (Clouston et al., 2016; Eren-Koçak et al., 2009), our cross-sectional analysis found that cognitive function did not differ in women with remitted PTSD compared to those who never had PTSD. These findings may be interpreted in two ways. First, PTSD is associated with adverse cognitive outcomes that can become alleviated as PTSD symptoms improve. The DSM-5 criteria for PTSD include cognitive symptoms such as attention and memory problems (American Psychiatric Association, 2013), and previous research has shown that these cognitive symptoms improve as individuals recover from PTSD (Ben-Zion et al., 2018; Nijdam et al., 2018). In addition, PTSD remission may be accompanied by behavioral and biological changes that contribute to improved cognition, such as improved quality of life (Schnurr & Lunney, 2016), sleep quality (Rousseau et al., 2021), blood pressure (Schubert et al., 2019), and hormone levels (Olff, de Vries, Güzelcan, Assies, & Gersons, 2007a), as well as reduced DNA strand breakage (Morath et al., 2014). Some neuroimaging studies of PTSD also found changes in functional activation and neurocircuitry mechanisms in response to psychotherapies (Cisler et al., 2016a, 2016b; Helpman et al., 2016; Simmons, Norman, Spadoni, & Strigo, 2013), and specific changes in brain structural network among individuals with remitted PTSD that had been linked to improved cognitive processing (Sun et al., 2018).

Second, we cannot rule out the possibility that lower cognition preceded and predicted PTSD remission. Pre-trauma or pre-PTSD cognitive ability has been shown to be associated with PTSD onset and severity (Aupperle, Melrose, Stein, & Paulus, 2012; Scott et al., 2015), as well as PTSD trajectories (Samuelson et al., 2020) and treatment outcomes (Haaland et al., 2016; Nijdam, de Vries, Gersons, & Olff, 2015; van Rooij, Kennis, Vink, & Geuze, 2016). However, in this study, cognitive function was only measured after PTSD assessment; thus, we could not determine whether cognitive function predicted PTSD remission. Future research with pre- and post-PTSD assessments of cognition will be necessary for a comprehensive characterization of the cognitive trajectory over the course of PTSD.

While women with remitted PTSD showed no difference in cognitive function assessed six years later from the reference group of women who never had PTSD, our longitudinal analysis indicated that women who had previously experienced high levels of PTSD symptoms were still prone to a more rapid decline in learning/working memory after symptom remission, at a rate comparable to that observed in women with unresolved symptoms of similar levels. These findings indicate that there may be lingering cognitive effects even after the remission of PTSD symptoms and suggest the potential need for continued support among women who have experienced high levels of PTSD symptoms in the past. PTSD is associated with dysregulated physiological and behavioral responses, including sleep problems (U.S. Department of Veterans Affairs, 2021), substance abuse (Brady, Back, & Coffey, 2004), and many other chronic illnesses. These adverse health outcomes are more severe among individuals with higher levels of PTSD or persistent PTSD (D'Andrea, Sharma, Zelechoski, & Spinazzola, 2011); thus, such individuals may experience more potent, longer-lasting effects. Indeed, one study found that remitted PTSD patients still experienced poorer physical, psychological, and social functioning than those who never developed PTSD (Bryant et al., 2016). Furthermore, there is also evidence from previous research suggesting that PTSD may leave biological traces that can persist following symptom remission. For example, a neuroimaging study in U.S. military veterans showed that not all brain network architecture changes associated with PTSD returned to the level of trauma-exposed controls after PTSD remission (Sun et al., 2018); another study found that HPA axis feedback sensitivity remained unchanged after PTSD symptoms abated (Schubert et al., 2019). Together with previous evidence, our findings imply that the adverse health consequences of PTSD may not fully resolve following PTSD remission, which may leave individuals with a greater likelihood of an impaired aging process. However, no other longitudinal studies have examined cognitive decline during aging in relation to remitted and unresolved PTSD. Therefore, additional studies are warranted to replicate our findings.

This study builds upon previous research indicating that lifetime PTSD is associated with poorer cognition and faster cognitive decline by providing a more detailed characterization of this relationship regarding the remission of PTSD symptoms. Furthermore, in this study, PTSD was associated with greater impairment in learning/working memory compared to psychomotor speed/attention and accelerated decline in learning/working memory only. As age, mild cognitive impairment, and Alzheimer's disease (AD) also associate with greater impairments in learning/working memory compared to psychomotor speed/attention (Lim et al., 2012; Maruff et al., 2013), it is possible that cognitive domains that represent earlier or more severe deficits in the aging process are particularly susceptible to the effects of PTSD. Further research is needed to understand the biological basis of these PTSD-related cognitive differences and how they may predict the risk of AD.

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Our study has several limitations. First, our definitions of remitted and unresolved PTSD were limited by the data available. PTSD can take various courses across individuals, including stable remitting, fluctuating, or chronic (Galatzer-Levy et al., 2013; Marmar et al., 2015; Steinert et al., 2015). However, information about PTSD symptom onset and fluctuations over time was unavailable. Thus, PTSD status measured at a single time point in 2008 may not accurately reflect the duration or chronicity of PTSD symptoms or PTSD status at the time of cognitive assessment. In addition, not all the symptoms that make up the diagnostic criteria of PTSD were assessed, and limited information on treatment-seeking made it challenging to distinguish between natural remission *v.* remission from treatment. Future research that integrates more frequent and comprehensive assessment of PTSD symptoms will allow for a thorough investigation of the dynamic changes in symptom severity over time. Second, this sample consisted of individuals in the early years of aging, and the follow-up was relatively short, which may have limited our ability to detect modest associations between remitted PTSD and cognition. Thus, further research with longer follow-ups to older ages will be needed. Third, the presence of emotional symptoms, such as loss of interest and feeling jumpy, among women with unresolved symptoms at the time of the PTSD assessment could be a sign of early developing underlying neurodegenerative diseases; however, we could not directly assess this possibility due to the lack of data on neurodegenerative disease status. Fourth, observational studies are subject to confounding and selection bias. While our analysis was robust to a series of covariate adjustment and secondary analyses, unmeasured confounders or competing risks may partially account for the observed results. Fifth, while the traumatic experiences included in this study (such as sexual harassment and the death of a close family member or friend) are not specific to nurses, the NHS II cohort is predominantly white professionals; further research is needed to determine whether the findings generalize to more diverse populations.

Our study has many strengths. First, by leveraging data from a large, prospective cohort, we were able to analyze the largest sample to date, to the best of our knowledge, assessing the relationship of PTSD symptom remission with cognition. Second, we conducted both cross-sectional and longitudinal analysis, providing a comprehensive evaluation of the dynamic changes in cognition in relation to PTSD status. Third, the study was comprised of women. While women have a higher risk of PTSD than men (Christiansen & Berke, 2020), recover slower from PTSD (Breslau, 2009), and experience higher rates of dementia (Snyder et al., 2016), they have been underrepresented in PTSD-cognition research.

To summarize, we found that unresolved but not remitted PTSD was associated with worse cognitive function assessed six years later and that accelerated cognitive decline was observed during follow-up among women with either unresolved or remitted high levels of PTSD symptoms. These findings suggest the value of integrating cognitive assessments and interventions into routine mental health care for PTSD patients and providing resources and support for managing cognitive decline in this population. The observed accelerated cognitive decline may imply poorer cognitive aging and highlights the importance of screening and prevention of dementia not only for women experiencing persistent PTSD symptoms but also for those who have previously experienced high levels of PTSD symptoms. Co-occurring depression partially explained the link between unresolved PTSD symptoms and cognition, emphasizing the importance of monitoring and managing depression in PTSD treatment. Future research is needed to illuminate the pathways underlying the association of PTSD with worse cognitive trajectories and to determine whether effective PTSD treatment can prevent cognitive decline and other long-term adverse health outcomes associated with PTSD.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0033291723001915

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Competing interest. Dr Sumner received grants from the National Heart, Lung, and Blood Institute of the National Institutes of Health (NIH) and the US Department of Defense outside the submitted work. Dr Kubzansky received grants from the NIH during the conduct of the study and grants from the NIH and the US Department of Defense and royalties from Oxford University Press outside the submitted work. Dr Koenen received grants from Cohen Veterans Bioscience and the NIH during the conduct of the study; personal fees from BakerHostetler, Capita, Converys, Discovery Vitality, the European Central Bank, and the US Department of Justice; and royalties from Guilford Press and Oxford University Press outside the submitted work. Dr Roberts received grants from the NIH during the conduct of the study outside the submitted work. Dr Kang received grants from the NIH and Pfizer, Inc during the conduct of the study outside the submitted work. The authors do not have any other conflicts of interest to declare.

References

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Association. https://dsm.psychiatryonline.org/doi/book/10.1176/appi.books.9780890425596

Andresen, E. M., Malmgren, J. A., Carter, W. B., & Patrick, D. L. (1994). Screening for depression in well older adults: Evaluation of a short form of the CES-D. *American Journal of Preventive Medicine*, *10*(2), 77–84. doi: 10.1016/s0749-3797(18)30622-6

Aupperle, R. L., Melrose, A. J., Stein, M. B., & Paulus, M. P. (2012). Executive function and PTSD: Disengaging from trauma. *Neuropharmacology*, *62*(2), 686–694. doi: 10.1016/j.neuropharm.2011.02.008

Ben-Zion, Z., Fine, N. B., Keynan, N. J., Admon, R., Green, N., Halevi, M., … Shalev, A. Y. (2018). Cognitive flexibility predicts PTSD symptoms: Observational and interventional studies. *Frontiers in Psychiatry*, *9*, 477.

Boscarino, J. A. (2006). Posttraumatic stress disorder and mortality among U.S. Army veterans 30 years after military service. *Annals of Epidemiology*, *16*(4), 248–256. doi: 10.1016/j.annepidem.2005.03.009

Brady, K. T., Back, S. E., & Coffey, S. F. (2004). Substance abuse and posttraumatic stress disorder. *Current Directions in Psychological Science*, *13*(5), 206–209. doi: 10.1111/j.0963-7214.2004.00309.x

Brand, J. (1999). *Development, implementation and evaluation of multiple imputation strategies for the statistical analysis of incomplete data sets*. s.n.], S.l.

Breslau, N. (2009). The epidemiology of trauma, PTSD, and other posttrauma disorders. *Trauma, Violence & Abuse*, *10*(3), 198–210. doi: 10.1177/1524838009334448

Breslau, N., Peterson, E. L., Kessler, R. C., & Schultz, L. R. (1999). Short screening scale for DSM-IV posttraumatic stress disorder. *The American Journal of Psychiatry*, *156*(6), 908–911. doi: 10.1176/ajp.156.6.908

Bryant, R. A., McFarlane, A. C., Silove, D., O'Donnell, M. L., Forbes, D., & Creamer, M. (2016). The lingering impact of resolved PTSD on subsequent functioning. *Clinical Psychological Science*, *4*(3), 493–498. doi: 10.1177/2167702615598756

Chiuve, S. E., Fung, T. T., Rimm, E. B., Hu, F. B., McCullough, M. L., Wang, M., … Willett, W. C. (2012). Alternative dietary indices both strongly predict risk of chronic disease. *The Journal of Nutrition*, *142*(6), 1009–1018. doi: 10.3945/jn.111.157222

Christiansen, D. M., & Berke, E. T. (2020). Gender- and sex-based contributors to Sex differences in PTSD. *Current Psychiatry Reports*, *22*(4), 19. doi: 10.1007/s11920-020-1140-y

Cisler, J. M., Sigel, B. A., Kramer, T. L., Smitherman, S., Vanderzee, K., Pemberton, J., & Kilts, C. D. (2016a). Modes of large-scale brain network organization during threat processing and posttraumatic stress disorder symptom reduction during TF-CBT among adolescent girls. *PLoS ONE*, *11*(8), e0159620. doi: 10.1371/journal.pone.0159620

Cisler, J. M., Sigel, B. A., Steele, J. S., Smitherman, S., Vanderzee, K., Pemberton, J., … Kilts, C. D. (2016b). Changes in functional connectivity of the amygdala during cognitive reappraisal predict symptom reduction during trauma-focused cognitive-behavioral therapy among adolescent girls with post-traumatic stress disorder. *Psychological Medicine*, *46*(14), 3013–3023. doi: 10.1017/S0033291716001847

Clouston, S. A. P., Diminich, E. D., Kotov, R., Pietrzak, R. H., Richards, M., Spiro, A., … Luft, B. J. (2019). Incidence of mild cognitive impairment in World Trade Center responders: Long-term consequences of re-experiencing the events on 9/11/2001. *Alzheimer's & Dementia (Amsterdam, Netherlands)*, *11*, 628–636. doi: 10.1016/j.dadm.2019.07.006

Clouston, S. A. P., Kotov, R., Pietrzak, R. H., Luft, B. J., Gonzalez, A., Richards, M., … Bromet, E. J. (2016). Cognitive impairment among World Trade Center responders: Long-term implications of re-experiencing the 9/11 terrorist attacks. *Alzheimer's & Dementia: Diagnosis, Assessment & Disease Monitoring*, *4*, 67–75. doi: 10.1016/j.dadm.2016.08.001

D'Andrea, W., Sharma, R., Zelechoski, A. D., & Spinazzola, J. (2011). Physical health problems after single trauma exposure: When stress takes root in the body. *Journal of the American Psychiatric Nurses Association*, *17*(6), 378–392. doi: 10.1177/1078390311425187

Desmarais, P., Weidman, D., Wassef, A., Bruneau, M.-A., Friedland, J., Bajsarowicz, P., … Nguyen, Q. D. (2020). The interplay between posttraumatic stress disorder and dementia: A systematic review. *The American Journal of Geriatric Psychiatry*, *28*(1), 48–60. doi: 10.1016/j.jagp.2019.08.006

Dyball, D., Evans, S., Boos, C. J., Stevelink, S. A. M., & Fear, N. T. (2019). The association between PTSD and cardiovascular disease and its risk factors in male veterans of the Iraq/Afghanistan conflicts: A systematic review. *International Review of Psychiatry*, *31*(1), 34–48. doi: 10.1080/09540261.2019.1580686

Eren-Koçak, E., Kılıç, C., Aydın, I., & Hızlı, F. G. (2009). Memory and prefrontal functions in earthquake survivors: Differences between current and past post-traumatic stress disorder patients. *Acta Psychiatrica Scandinavica*, *119*(1), 35–44. doi: 10.1111/j.1600-0447.2008.01281.x

Fani, N., Kitayama, N., Ashraf, A., Reed, L., Afzal, N., Jawed, F., & Bremner, J. D. (2009). Neuropsychological functioning in patients with posttraumatic stress disorder following short-term paroxetine treatment. *Psychopharmacology Bulletin*, *42*(1), 53–68.

Fredrickson, J., Maruff, P., Woodward, M., Moore, L., Fredrickson, A., Sach, J., & Darby, D. (2010). Evaluation of the usability of a brief computerized cognitive screening test in older people for epidemiological studies. *Neuroepidemiology*, *34*(2), 65–75. doi: 10.1159/000264823

Galatzer-Levy, I. R., Ankri, Y., Freedman, S., Israeli-Shalev, Y., Roitman, P., Gilad, M., & Shalev, A. Y. (2013). Early PTSD symptom trajectories: Persistence, recovery, and response to treatment: Results from the Jerusalem Trauma Outreach and Prevention Study (J-TOPS). *PloS One*, *8*(8), e70084. doi: 10.1371/journal.pone.0070084

Gilsanz, P., Winning, A., Koenen, K. C., Roberts, A. L., Sumner, J. A., Chen, Q., … Kubzansky, L. D. (2017). Post-traumatic stress disorder symptom duration and remission in relation to cardiovascular disease risk among a large cohort of women. *Psychological Medicine*, *47*(8), 1370–1378. doi: 10.1017/S0033291716003378

Greenberg, M. S., Tanev, K., Marin, M.-F., & Pitman, R. K. (2014). Stress, PTSD, and dementia. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, *10*(3 Suppl), S155–S165. doi: 10.1016/j.jalz.2014.04.008

Haaland, K. Y., Sadek, J. R., Keller, J. E., & Castillo, D. T. (2016). Neurocognitive correlates of successful treatment of PTSD in female veterans. *Journal of the International Neuropsychological Society*, *22*(6), 643–651. doi: 10.1017/S1355617716000424

Hammers, D., Spurgeon, E., Ryan, K., Persad, C., Barbas, N., Heidebrink, J., … Giordani, B. (2012). Validity of a brief computerized cognitive screening test in dementia. *Journal of Geriatric Psychiatry and Neurology*, *25*(2), 89–99. doi: 10.1177/0891988712447894

Helpman, L., Marin, M.-F., Papini, S., Zhu, X., Sullivan, G. M., Schneier, F., … Neria, Y. (2016). Neural changes in extinction recall following prolonged exposure treatment for PTSD: A longitudinal fMRI study. *NeuroImage: Clinical*, *12*, 715–723. doi: 10.1016/j.nicl.2016.10.007

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593–602. doi: 10.1001/archpsyc.62.6.593

Koenen, K. C., Ratanatharathorn, A., Ng, L., McLaughlin, K. A., Bromet, E. J., Stein, D. J., … Kessler, R. C. (2017). Posttraumatic stress disorder in the World Mental Health Surveys. *Psychological Medicine*, *47*(13), 2260–2274. doi: 10.1017/S0033291717000708

Koyama, A. K., Hagan, K. A., Okereke, O. I., Weisskopf, M. G., Rosner, B., & Grodstein, F. (2015). Evaluation of a self-administered computerized cognitive battery in an older population. *Neuroepidemiology*, *45*(4), 264–272. doi: 10.1159/000439592

Kubzansky, L. D., Koenen, K. C., Spiro, A., Vokonas, P. S., & Sparrow, D. (2007). Prospective study of posttraumatic stress disorder symptoms and coronary heart disease in the normative aging study. *Archives of General Psychiatry*, *64*(1), 109–116. doi: 10.1001/archpsyc.64.1.109

Lawn, R. B., Jha, S. C., Liu, J., Sampson, L., Murchland, A. R., Sumner, J. A., … Koenen, K. C. (2022). The association of posttraumatic stress disorder, depression, and head injury with mid-life cognitive function in civilian women. *Depression and Anxiety*, *39*(3), 220–232. doi: 10.1002/da.23233

Lim, Y. Y., Ellis, K. A., Harrington, K., Ames, D., Martins, R. N., Masters, C. L., … the AIBL Research Group. (2012). Use of the CogState brief battery in the assessment of Alzheimer's disease related cognitive impairment in the Australian Imaging, Biomarkers and Lifestyle (AIBL) study. *Journal of Clinical and Experimental Neuropsychology*, *34*(4), 345–358. doi: 10.1080/13803395.2011.643227

Lim, Y. Y., Jaeger, J., Harrington, K., Ashwood, T., Ellis, K. A., Stöffler, A., … Maruff, P. (2013a). Three-month stability of the CogState brief battery in healthy older adults, mild cognitive impairment, and Alzheimer's Disease: Results from the Australian Imaging, Biomarkers, and Lifestyle-Rate of Change Substudy (AIBL-ROCS). *Archives of Clinical Neuropsychology*, *28*(4), 320–330. doi: 10.1093/arclin/act021

Lim, Y. Y., Pietrzak, R. H., Ellis, K. A., Jaeger, J., Harrington, K., Ashwood, T., … Maruff, P. (2013b). Rapid decline in episodic memory in healthy older adults with high amyloid-β. *Journal of Alzheimer's Disease*, *33*(3), 675–679. doi: 10.3233/JAD-2012-121516

Marmar, C. R., Schlenger, W., Henn-Haase, C., Qian, M., Purchia, E., Li, M., … Kulka, R. A. (2015). Course of posttraumatic stress disorder 40 years after the Vietnam war: Findings from the national Vietnam veterans longitudinal study. *JAMA Psychiatry*, *72*(9), 875–881. doi: 10.1001/jamapsychiatry.2015.0803

Maruff, P., Lim, Y. Y., Darby, D., Ellis, K. A., Pietrzak, R. H., Snyder, P. J., … AIBL Research Group. (2013). Clinical utility of the cogstate brief battery in identifying cognitive impairment in mild cognitive impairment and Alzheimer's disease. *BMC Psychology*, *1*(1), 30. doi: 10.1186/2050-7283-1-30

Maruff, P., Thomas, E., Cysique, L., Brew, B., Collie, A., Snyder, P., & Pietrzak, R. H. (2009). Validity of the CogState brief battery: Relationship to standardized tests and sensitivity to cognitive impairment in mild traumatic brain

injury, schizophrenia, and AIDS dementia complex. *Archives of Clinical Neuropsychology: The Official Journal of the National Academy of Neuropsychologists*, *24*(2), 165–178. doi: 10.1093/arclin/acp010

Morath, J., Moreno-Villanueva, M., Hamuni, G., Kolassa, S., Ruf-Leuschner, M., Schauer, M., … Kolassa, I. T. (2014). Effects of psychotherapy on DNA strand break accumulation originating from traumatic stress. *Psychotherapy and Psychosomatics*, *83*(5), 289–297.

Morgan, C. A., Hazlett, G., Wang, S., Richardson, E. G., Schnurr, P., & Southwick, S. M. (2001). Symptoms of dissociation in humans experiencing acute, uncontrollable stress: A prospective investigation. *The American Journal of Psychiatry*, *158*(8), 1239–1247. doi: 10.1176/appi.ajp.158.8.1239

Morina, N., Wicherts, J. M., Lobbrecht, J., & Priebe, S. (2014). Remission from post-traumatic stress disorder in adults: A systematic review and meta-analysis of long term outcome studies. *Clinical Psychology Review*, *34*(3), 249–255. doi: 10.1016/j.cpr.2014.03.002

Nijdam, M. J., de Vries, G.-J., Gersons, B. P. R., & Olff, M. (2015). Response to psychotherapy for posttraumatic stress disorder: The role of pretreatment verbal memory performance. *The Journal of Clinical Psychiatry*, *76*(08), e1023–e1028. doi: 10.4088/JCP.14m09438

Nijdam, M. J., Martens, I. J., Reitsma, J. B., Gersons, B. P., & Olff, M. (2018). Neurocognitive functioning over the course of trauma-focused psychotherapy for PTSD: Changes in verbal memory and executive functioning. *British Journal of Clinical Psychology*, *57*(4), 436–452.

Olff, M. (2017). Sex and gender differences in post-traumatic stress disorder: An update. *European Journal of Psychotraumatology*, *8*(sup4), 1351204. doi: 10.1080/20008198.2017.1351204

Olff, M., de Vries, G.-J., Güzelcan, Y., Assies, J., & Gersons, B. P. R. (2007a). Changes in cortisol and DHEA plasma levels after psychotherapy for PTSD. *Psychoneuroendocrinology*, *32*(6), 619–626. doi: 10.1016/j.psyneuen.2007.04.001

Olff, M., Langeland, W., Draijer, N., & Gersons, B. P. R. (2007b). Gender differences in posttraumatic stress disorder. *Psychological Bulletin*, *133*(2), 183–204. doi: 10.1037/0033-2909.133.2.183

Rimm, E. B., Stampfer, M. J., Colditz, G. A., Chute, C. G., Litin, L. B., & Willett, W. C. (1990). Validity of self-reported waist and hip circumferences in men and women. *Epidemiology* (*Cambridge, Mass.*), *1*(6), 466–473. doi: 10.1097/00001648-199011000-00009

Roberts, A. L., Agnew-Blais, J. C., Spiegelman, D., Kubzansky, L. D., Mason, S. M., Galea, S., … Koenen, K. C. (2015). Posttraumatic stress disorder and incidence of type 2 diabetes mellitus in a sample of women: A 22-year longitudinal study. *JAMA Psychiatry*, *72*(3), 203–210. doi: 10.1001/jamapsychiatry.2014.2632

Roberts, A. L., Kubzansky, L. D., Chibnik, L. B., Rimm, E. B., & Koenen, K. C. (2020a). Association of posttraumatic stress and depressive symptoms with mortality in women. *JAMA Network Open*, *3*(12), e2027935. doi: 10.1001/jamanetworkopen.2020.27935

Roberts, A. L., Liu, J., Lawn, R. B., Jha, S. C., Sumner, J. A., Kang, J. H., … Koenen, K. C. (2022). Association of posttraumatic stress disorder with accelerated cognitive decline in middle-aged women. *JAMA Network Open*, *5*(6), e2217698. doi: 10.1001/jamanetworkopen.2022.17698

Roberts, A. L., Sumner, J. A., Koenen, K. C., Kubzansky, L. D., Grodstein, F., Rich-Edwards, J., … Weisskopf, M. G. (2020b). Childhood abuse and cognitive function in a large cohort of middle-aged women. *Child Maltreatment*, *27*(1), 100–113. doi: 10.1177/1077559520970647

Rosen, R., Shao, Y., Zhang, Q., Bao, J., Zhang, Y., Masurkar, A., … Reibman, J. (2022). Cognitive function among World Trade Center-exposed community members with mental health symptoms. *International Journal of Environmental Research and Public Health*, *19*(6), 3440. doi: 10.3390/ijerph19063440

Rousseau, P. F., Vallat, R., Coste, O., Cadis, H., Nicolas, F., Trousselard, M., … Khalfa, S. (2021). Sleep parameters improvement in PTSD soldiers after symptoms remission. *Scientific Reports*, *11*(1), 8873. doi: 10.1038/s41598-021-88337-x

Rubin, D. B. (Ed.). (1987). *Multiple imputation for nonresponse in surveys*. Hoboken, NJ, USA: John Wiley & Sons, Inc. doi: 10.1002/9780470316696

Rytwinski, N. K., Scur, M. D., Feeny, N. C., & Youngstrom, E. A. (2013). The co-occurrence of major depressive disorder among individuals with posttraumatic stress disorder: A meta-analysis. *Journal of Traumatic Stress*, *26*(3), 299–309. doi: 10.1002/jts.21814

Samuelson, K. W., Newman, J., Abu Amara, D., Qian, M., Li, M., Schultebraucks, K., … Marmar, C. R. (2020). Predeployment neurocognitive functioning predicts postdeployment posttraumatic stress in army personnel. *Neuropsychology*, *34*(3), 276–287. (2019-72839-001). doi: 10.1037/neu0000603

Scherrer, J. F., Salas, J., Friedman, M. J., Cohen, B. E., Schneider, F. D., Lustman, P. J., … Schnurr, P. P. (2020a). Clinically meaningful posttraumatic stress disorder (PTSD) improvement and incident hypertension, hyperlipidemia, and weight loss. *Health Psychology*, *39*(5), 403–412. (2020-22412-001). doi: 10.1037/hea0000855

Scherrer, J. F., Salas, J., Norman, S. B., Schnurr, P. P., Chard, K. M., Tuerk, P., … Lustman, P. J. (2019). Association between clinically meaningful posttraumatic stress disorder improvement and risk of type 2 diabetes. *JAMA Psychiatry*, *76*(11), 1159–1166. doi: 10.1001/jamapsychiatry.2019.2096

Scherrer, J. F., Salas, J., Schneider, F. D., Friedman, M. J., van den Berk-Clark, C., Chard, K. M., … Cohen, B. E. (2020b). PTSD Improvement and incident cardiovascular disease in more than 1000 veterans. *Journal of Psychosomatic Research*, *134*, 110128. doi: 10.1016/j.jpsychores.2020.110128

Schnurr, P. P., & Lunney, C. A. (2016). Symptom benchmarks of improved quality of life in PTSD. *Depression and Anxiety*, *33*(3), 247–255. doi: 10.1002/da.22477

Schnurr, P. P., Vielhauer, M., Weathers, F., & Findler, M. (1999). *Brief Trauma Questionnaire*.

Schubert, C. F., Schreckenbach, M., Kirmeier, T., Gall-Kleebach, D. J., Wollweber, B., Buell, D. R., … Schmidt, U. (2019). PTSD psychotherapy improves blood pressure but leaves HPA axis feedback sensitivity stable and unaffected: First evidence from a pre-post treatment study. *Psychoneuroendocrinology*, *100*, 254–263. doi: 10.1016/j.psyneuen.2018.10.013

Schuitevoerder, S., Rosen, J. W., Twamley, E. W., Ayers, C. R., Sones, H., Lohr, J. B., … Thorp, S. R. (2013). A meta-analysis of cognitive functioning in older adults with PTSD. *Journal of Anxiety Disorders*, *27*(6), 550–558. doi: 10.1016/j.janxdis.2013.01.001

Scott, J. C., Matt, G. E., Wrocklage, K. M., Crnich, C., Jordan, J., Southwick, S. M., … Schweinsburg, B. C. (2015). A quantitative meta-analysis of neurocognitive functioning in posttraumatic stress disorder. *Psychological Bulletin*, *141*(1), 105–140. doi: 10.1037/a0038039

Simmons, A. N., Norman, S. B., Spadoni, A. D., & Strigo, I. A. (2013). Neurosubstrates of remission following prolonged exposure therapy in veterans with posttraumatic stress disorder. *Psychotherapy and Psychosomatics*, *82*(6), 382–389. doi: 10.1159/000348867

Snyder, H. M., Asthana, S., Bain, L., Brinton, R., Craft, S., Dubal, D. B., … Carrillo, M. C. (2016). Sex biology contributions to vulnerability to Alzheimer's disease: A think tank convened by the women's Alzheimer's research initiative. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, *12*(11), 1186–1196. doi: 10.1016/j.jalz.2016.08.004

Steinert, C., Hofmann, M., Leichsenring, F., & Kruse, J. (2015). The course of PTSD in naturalistic long-term studies: High variability of outcomes. A systematic review. *Nordic Journal of Psychiatry*, *69*(7), 483–496. doi: 10.3109/08039488.2015.1005023

Sumner, J. A., Hagan, K., Grodstein, F., Roberts, A. L., Harel, B., & Koenen, K. C. (2017). Posttraumatic stress disorder symptoms and cognitive function in a large cohort of middle-aged women. *Depression and Anxiety*, *34*(4), 356–366. doi: 10.1002/da.22600

Sumner, J. A., Kubzansky, L. D., Elkind, M. S. V., Roberts, A. L., Agnew-Blais, J., Chen, Q., … Koenen, K. C. (2015). Trauma exposure and posttraumatic stress disorder symptoms predict onset of cardiovascular events in women. *Circulation*, *132*(4), 251–259. doi: 10.1161/CIRCULATIONAHA.114.014492

Sun, D., Davis, S. L., Haswell, C. C., Swanson, C. A., LaBar, K. S., Fairbank, J. A., & Morey, R. A. (2018). Brain structural covariance network topology in remitted posttraumatic stress disorder. *Frontiers in Psychiatry*, *9*, 90. doi: 10.3389/fpsyt.2018.00090

U.S. Department of Veterans Affairs. (2021). Sleep Problems in Veterans with PTSD – PTSD: National Center for PTSD [General Information]. Retrieved December 2, 2021, from https://www.ptsd.va.gov/professional/treat/cooccurring/sleep_problems_vets.asp.

https://doi.org/10.1017/S0033291723001915 Published online by Cambridge University Press

van Buuren, S. (2007). Multiple imputation of discrete and continuous data by fully conditional specification. *Statistical Methods in Medical Research*, *16* (3), 219–242. doi: 10.1177/0962280206074463

Vancampfort, D., Rosenbaum, S., Ward, P. B., Steel, Z., Lederman, O., Lamwaka, A. V., … Stubbs, B. (2016). Type 2 diabetes among people with posttraumatic stress disorder: Systematic review and meta-analysis. *Psychosomatic Medicine*, *78*(4), 465–473. doi: 10.1097/PSY.0000000000000297

van Rooij, S. J. H., Kennis, M., Vink, M., & Geuze, E. (2016). Predicting treatment outcome in PTSD: A longitudinal functional MRI study on trauma-unrelated emotional processing. *Neuropsychopharmacology: Official Publication of the American College of Neuropsychopharmacology*, *41*(4), 1156–1165. doi: 10.1038/npp.2015.257

Vermetten, E., Vythilingam, M., Southwick, S. M., Charney, D. S., & Bremner, J. D. (2003). Long-term treatment with paroxetine increases verbal declarative memory and hippocampal volume in posttraumatic stress disorder. *Biological Psychiatry*, *54*(7), 693–702. doi: 10.1016/s0006-3223(03)00634-6

Vivot, A., Power, M. C., Glymour, M. M., Mayeda, E. R., Benitez, A., Spiro, A., … Gross, A. L. (2016). Jump, Hop, or skip: Modeling practice effects in studies of determinants of cognitive change in older adults. *American Journal of Epidemiology*, *183*(4), 302–314. doi: 10.1093/aje/kwv212

Walter, K. H., Palmieri, P. A., & Gunstad, J. (2010). More than symptom reduction: Changes in executive function over the course of PTSD treatment. *Journal of Traumatic Stress*, *23*(2), 292–295. doi: 10.1002/jts.20506