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RESEARCH ARTICLE

Cognition improvement in U.S. veterans undergoing treatment for posttraumatic stress disorder: Secondary analyses from a randomized controlled trial

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Abstract

Cognitive dysfunction is a hallmark of posttraumatic stress disorder (PTSD). Although treatments effectively reduce core PTSD symptoms, limited research has examined whether associated cognitive impairments improve following treatment. This study investigated cognitive changes in veterans receiving treatment for PTSD and explored the associations between cognitive improvement and PTSD symptom reduction. U.S. veterans ($N = 85$) with clinically significant PTSD symptoms were randomized to receive either cognitive processing therapy (CPT; $n = 44$) or Sudarshan Kriya yoga (SKY; $n = 41$) in a noninferiority trial. Cognitive function was assessed pre- and posttreatment using the Cambridge Neuropsychological Test Automated Battery. PTSD symptoms were assessed using the Clinician-Administered PTSD Scale (CAPS-5). Following treatment, participants showed significant improvements in episodic visual memory, $d = 0.51$, $p < .001$; motor learning, $d = 0.57$, $p < .001$; and visual sustained attention, $d = 0.37$, $p = .005$. There were no significant differences in cognitive improvement between the CPT and SKY groups. Changes in overall cognitive function were significantly correlated with PTSD symptom reductions across both treatment groups. Regardless of treatment, cognitive function improved alongside PTSD symptom reduction. These findings provide evidence that treating PTSD not only alleviates PTSD symptoms but may also improve associated cognitive function.

Posttraumatic stress disorder (PTSD) is a neuropsychiatric condition that can develop after a traumatic event (American Psychiatric Association [APA], 2013). Symptoms include intrusive thoughts, avoidance behaviors, negative changes in cognitions and mood, and heightened arousal and reactivity. An estimated 5%–15% of U.S. service members develop PTSD in their lifetime (Tanielian & Jaycox, 2008). Cognitive dysfunction in PTSD is a diagnostic criterion (APA, 2013) and observable in empirical studies (Alves de Araujo Junior et al., 2023; Harnett et al., 2020), with individuals frequently exhibiting deficits in attention, working memory, episodic memory, information processing speed, and executive functioning (Scott et al., 2015). Verbal learning and memory abilities show the largest

and most consistent impairments (Johnsen & Asbjørnsen, 2008; Scott et al., 2015), whereas processing speed represents the most robust nonverbal cognitive deficit in PTSD, affecting performance across multiple cognitive domains and impacting daily functioning (Jacob et al., 2019; Scott et al., 2015). Moreover, there is evidence that cognitive dysfunction is a core feature of PTSD pathophysiology rather than a secondary consequence of emotional distress (Quinones et al., 2020). Cognitive deficits in PTSD are closely linked to structural changes in both gray and white matter, particularly in the prefrontal cortex, hippocampus, and amygdala (Harnett et al., 2020). Complementing the structural changes, converging task-based functional magnetic resonance imaging (fMRI) evidence indicates that PTSD is characterized by cognitive impairments in several domains, with selective dysfunctions in brain regions that are part of cortical networks (Dossi et al., 2020).

An important question is whether the cognitive impairments seen in PTSD are reversible following treatment. Cross-sectional designs limit conclusions regarding causality, and few longitudinal studies have measured cognition at baseline and posttreatment. Among these few existing longitudinal studies, treatment-related improvements have been observed most consistently in relation to memory, with medium-to-large effect sizes (Susanty et al., 2022). In contrast, effects on other cognitive domains (e.g., attention, executive function, and information processing) have been largely nonsignificant across studies. Four studies that have employed various therapeutic approaches in veteran samples have demonstrated that many veterans show improvements in PTSD and comorbid symptoms (e.g., anxiety) following treatment, though broader improvements in cognitive function are less reliably observed (Haaland et al., 2016; Jak et al., 2019; Kent et al., 2011; McLay et al., 2014). More longitudinal studies with baseline and posttreatment cognitive assessments are needed to evaluate the effects of treatment on cognition.

We recently demonstrated the noninferiority of a breathing-based yoga treatment (Sudarshan Kriya yoga; SKY; Brown & Gerbarg, 2005) compared to a first-line, evidence-based, trauma-focused psychotherapy (cognitive processing therapy [CPT] without a written account; Resick, Monson, & Chard, 2024; Resick, LoSavio et al., 2024) for treating PTSD in U.S. service members (Bayley et al., 2022). Here, we present secondary analyses of cognitive function, which was measured at baseline and posttreatment, thus providing an ideal opportunity to determine the effects of treatment on cognition in PTSD. We hypothesized that cognitive function would improve following treatment across domains known to be impaired in PTSD, including attention, working memory, episodic memory, information processing speed, and executive functioning. We further hypothesized that cognitive improvements would be proportional to the degree of improvement in PTSD symptoms and would be similar in both treatment groups.

METHOD

Participants

The methods for this randomized controlled noninferiority trial are reported in detail elsewhere (Bayley et al., 2022; Mathersul et al., 2019). In addition to new measures used in this analysis, only key elements are repeated here. Veterans ($N = 85$, $n = 10$ female, $n = 75$ male) with clinically significant PTSD symptoms were recruited and randomized into one of the two treatment groups (CPT or SKY) between September 15, 2015, and September 30, 2019 (Figure 1). Power analyses indicated a minimum of 30 participants per group (Mathersul et al., 2019). As outlined in the inclusion criteria (see Bayley et al., 2022), all participants were veterans with elevated PTSD symptoms, assessed during an initial phone interview and defined as a score of 38 or higher on the PTSD Checklist for *DSM-5* (PCL-5; Weathers, Litz, et al., 2013). A total of 62 participants (72.9%) completed both the baseline and posttreatment cognitive assessments; 23 participants did not complete the Clinician-Administered PTSD Scale for *DSM-5* (CAPS-5; Weathers, Blake, et al., 2013) at posttreatment, resulting in 30 participants in the SKY group ($M_{age} = 60.67$ years, $SD = 10.64$, female: $n = 5$) and 32 participants in the CPT group ($M_{age} = 58.25$ years, $SD = 12.71$, female: $n = 2$). Of these 62 total participants, all except one, who completed 4 of 6 weeks of treatment, met the criteria for CPT completion, defined as attending 75% or more of the treatment sessions. The timeframe for CPT treatment ranged from 5 weeks to 9.6 weeks ($M = 6.51$ weeks, $SD = 1.00$ week).

Procedure

Eligibility, as informed by the inclusion and exclusion criteria (Bayley et al., 2022), was determined through an initial telephone screening. This was followed by an on-site screening visit to evaluate the remaining criteria, obtain informed consent, and collect participants' health history. Measures were administered at baseline and posttreatment by research staff masked to treatment group and included the Beck Depression Inventory–II (BDI-II; Beck et al., 1996) and the CAPS-5 (Weathers, Blake, et al., 2013). Cognitive function was assessed at baseline and posttreatment using alternate versions of the Cambridge Neuropsychological Test Automated Battery (CANTAB; Cambridge Cognition, 2015), counterbalanced across time points. Randomization among recruited participants occurred less than 1 week before treatment began.

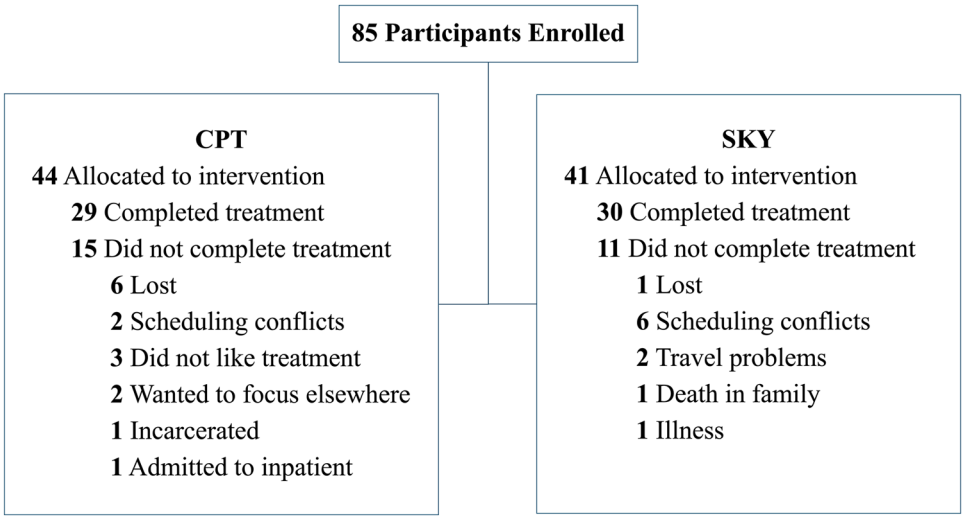

FIGURE 1 *Participant allocation flow diagram*
Note: Adapted from Bayley et al. (2022; Figure 1). CPT = cognitive processing therapy; SKY = Sudarshan Kriya yoga.

CPT without the written account was delivered according to standard clinical treatment protocols (Resick, Monson, & Chard, 2024), individually in two 1-hr sessions per week for 6 weeks, for a total of 12 hr of contact time (i.e., 12 sessions). The SKY intervention was delivered in group format with between three and six participants per group, following the Project Welcome Home Troops (2014) protocol. The SKY program began with an initial 5-day intensive group workshop (3 hr per day, for a total of 15 hr) focused on teaching and practicing the core breathing techniques, meditative practices, and stress-reduction routines foundational to SKY. Following this workshop, participants attended 10 1-hr group sessions twice per week for 6 weeks, for an additional 25 hr of group contact time. In total, the SKY treatment involved approximately 40 hr of structured contact time compared with 12 hr for CPT, though both conditions spanned a 6-week treatment period. As explained in the original protocol (Mathersul et al., 2019), the equivalent duration was deemed to be more crucial than an equivalent number of hours, as the initial intensive was a feature that could not be altered (Mathersul et al., 2019).

Measures

PTSD symptom severity

The CAPS-5 (Weathers, Blake, et al., 2013), a semistructured clinical diagnostic interview for the assessment of PTSD per criteria outlined in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*; APA, 2013), was administered at baseline and posttreatment. CAPS-5 severity scores range from 0 to 80, with higher scores reflecting higher levels of symptom severity. The scale produces high interrater reliability (.78–1.00) and test–retest reliability (.83) and has demonstrated convergent validity with *DSM-IV* version of the measure (r = .83; Weathers et al., 2018). In the current sample, internal reliability for the CAPS-5 was excellent at both baseline, Cronbach's α = .90, 95% confidence interval (CI) [.85, .93], and posttreatment, Cronbach's α = .93, 95% CI [.90, .95] (see Supplementary Table S1 for cluster-specific reliability statistics).

Mood symptoms

The BDI-II (Beck et al., 1996), a 21-item self-report questionnaire designed to assess the severity of depressive symptoms, was administered at baseline and posttreatment. Scores range from 0 to 63, with higher scores indicating higher levels of depressive symptom severity. The BDI-II has demonstrated excellent retest reliability (.73–.96), internal consistency (.90), and validity (Wang & Gorenstein, 2013). In the current sample, internal reliability for the BDI-II was excellent at both baseline, Cronbach's α = .92, 95% CI [.89, .95], and posttreatment, Cronbach's α = .93, 95% CI [.90, .95] (Supplementary Table S1).

Cognitive function

Tests from the CANTAB (Cambridge Cognition, 2015) were used to assess cognitive function. The CABTAB tasks selected for the current study were chosen to evaluate various common cognitive concerns associated with PTSD.

Assessments focused on four domains: episodic visual memory and learning; visual, movement, and comprehension difficulties; visual sustained attention; and working memory and strategy use.

Episodic visual memory and learning

The paired associates learning (PAL) task was administered to assess episodic visual memory and learning. During the task, multiple boxes were displayed on the screen, with each opening sequentially and some containing a specific pattern. After viewing the boxes, the participant was required to identify the boxes that contained the pattern. The main outcome measure was the total number of errors, with higher scores indicating worse episodic memory function (min: 0, max: 111). The PAL task has demonstrated good test–retest reliability (.73; Karlsen et al., 2022).

Visual, movement, and comprehension difficulties

The motor screening task (MOT) was administered to assess visual, movement, and comprehension difficulties. During the task, colored crosses appeared at different locations on the screen, one at a time. Participants were asked to promptly and accurately touch the displayed cross. The outcome measures were the number of errors, calculated as the accuracy (Euclidean distance between finger and target) and latency (average speed of making a response), with higher scores indicating worse function. The MOT was originally designed as a screening measure, meant to be used to invalidate cognitive test performance by identifying individuals with motor or comprehension difficulties, rather than as a primary cognitive outcome (Cambridge Cognition, 2023). Therefore, reliability and validity data for MOT are limited in the peer reviewed literature, as it functions primarily as a quality control task in CANTAB batteries.

Visual sustained attention

The rapid visual information processing (RVP) task was used to assess visual sustained attention. During the task, participants saw a white box in the center of the screen displaying a pseudorandom sequence of digits, varying in length from two to nine digits. Participants were instructed to recognize designated digit sequences that varied from a single target sequence to three different sequences, depending on task difficulty. Once a target sequence was detected, participants were instructed to press a button situated in the center of the touch screen as quickly as possible. The outcome measures were latency (response speed) and the probability of false positives, with higher scores indicating worse function. The RVP task has demonstrated good test–retest reliability (.75; Karlsen et al., 2022).

Working memory and strategy use

The spatial working memory (SWM) task was used to assess working memory and strategy use. During the task, several colored squares were displayed on the screen, and participants were asked to touch the screen to "open" them, using their own strategy to find a yellow token hidden in one of the boxes. Once revealed, the token was added to an empty column on the right side of the screen, and the next trial began (i.e., a new search to find the next token). The difficulty level of the test was adjusted by gradually increasing the number of boxes (up to 12). The box colors and positions were altered with each trial. The outcome measures were the total number of errors (reselecting boxes that had previously been identified as empty or returning to a box that had already been confirmed to contain a token) and strategy (based on the number of search sequences, with lower scores indicative of a more effective strategy use of strategy). Both outcome measures have demonstrated good test–retest reliability (errors: .71, strategy: .79; Karlsen et al., 2022).

Data analysis

Changes in cognitive performance were assessed by calculating the difference between baseline and posttreatment scores. Individual cognitive domain–specific changes (i.e., CANTAB tasks) were standardized by converting them to Z scores ($Z_{\text{PAL\_errors}}$, $Z_{\text{MOT\_latency}}$, $Z_{\text{MOT\_errors}}$, $Z_{\text{RVP\_latency}}$, $Z_{\text{RVP\_false alarms}}$, $Z_{\text{SWM\_errors}}$, $Z_{\text{SWM\_strategy}}$). An overall cognitive composite score was also calculated by averaging the seven Z-score changes.

Analyses were conducted using JASP (JASP Team, 2024) and included t tests (change in cognitive measures following treatment), standard Pearson's correlations, linear regressions (associations between cognitive measures and PTSD symptoms), and repeated measures analyses of variance (ANOVAs; cognitive measures with regard to treatment and time point). Depression often co-occurs with PTSD, with significant depressive symptom levels affecting 30%–50% of individuals diagnosed with PTSD (Campbell et al., 2007). Moreover, cognitive dysfunction is also found in people with major depressive disorder (Douglas & Porter, 2009). Therefore, we also performed an analysis of covariance (ANCOVA) for treatment-related cognitive measure changes and timepoint-corrected for depressive symptom levels, using the BDI-II score as a covariate.

Linear regression analyses were performed to examine the associations between baseline characteristics and treatment outcomes. Results were reported as regression equations to allow for the calculation of predicted values for specific baseline profiles: outcome variable =

intercept + coefficient(s) × predictor(s). Additionally, robust correlation analyses were performed in R using a script that automatically selected the most appropriate correlation method (Pearson's, Spearman skipped or percentage bend) based on outlier detection and normality assessment (Apšvalka, 2022).

We tested all measures (CAP-5, BDI-II, CANTAB) for normality using the Shapiro–Wilk test. If these measures were not normally distributed, a nonparametric test (Wilcoxon signed-rank) was also used. For measures with subtle differences that are, nonetheless, functionally significant, a reliable change index (RCI) can be calculated to facilitate further interpretation. Therefore, reliable change was evaluated using the Jacobson–Truax approach (Jacobson & Truax, 1991), computing an individual RCI for each outcome to distinguish genuine improvement from normal score variability. The equation is given below, with r_{tt} representing the test–retest reliability coefficient.

$$RCI = \frac{post - pre}{SE_{diff}} \text{ where } SE_{diff} = \sqrt{2 \times SEM^2}, SEM = SD_{baseline} \times \sqrt{1 - r_{tt}}$$

The two-tailed 95% criterion classified change as reliable when $|RCI|$ is greater than or equal to 1.96, equivalent to an absolute raw score change of at least 1.96 x SE_{diff}. Improvement direction was defined a priori by measure (e.g., decreases indicate improvement for symptom severity and error or latency metrics; increases indicate improvement for accuracy and sensitivity metrics). Test–retest reliability coefficients (r_{tt}) were drawn from published sources matched by instrument and outcome metric, when available, as follows: BDI-II: .73–.96 (Wang & Gorenstein, 2013); CAPS-5: .78–1.00 (Weathers et al., 2018); and the CANTAB tasks, including PAL: .74–.85, RVP: .55–.78, SWM Errors: .59–.71, and SWM Strategy: .13–.79 (Cacciamani et al., 2018; Gonçalves et al., 2016). Given that a range is provided, the lower-bound and upper-bound values were both used to provide conservative and liberal classifications (Supplementary Table S2).

RESULTS

Effects of treatment on symptoms of PTSD and depression

As reported elsewhere (Bayley et al., 2022), PTSD symptoms, as measured by the CAPS-5 total severity score, improved at posttreatment (Table 1). Depressive symptoms, as measured by the BDI-II, also improved (Table 1). All measures passed the test of normality except for the CAPS-5, $W = 0.96$, $p = .046$. However, the reduction in CAPS-5 total severity score was maintained using nonparametric analysis, $V = 1{,}520$, $p < .001$, $r = .56$. Reliable change, computed via the Jacobson–Truax method, indicated that even with conservative measures, 9.7% of the participants showed reliable improvement for depressive symptoms, with 24.2% showing improvement in PTSD symptoms as measured using the CAPS-5 (Supplementary Table S2).

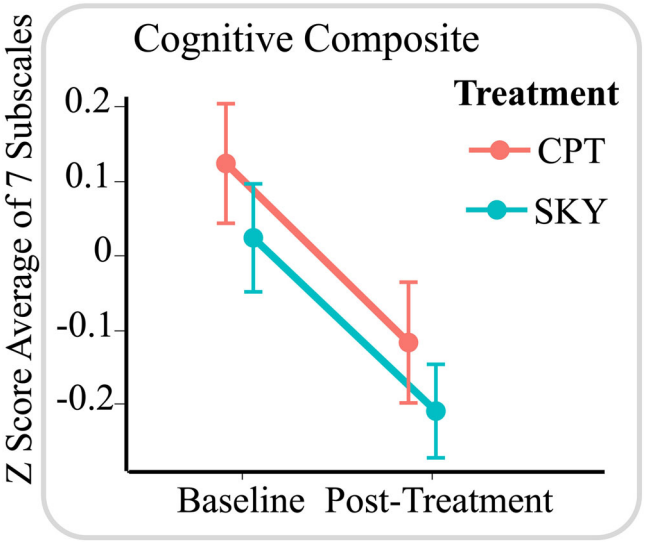

FIGURE 2 *Change in cognitive composite score across time and treatment*

Note: The cognitive composite score is the average of the *Z* scores across all seven Cambridge Neuropsychological Test Automated Battery subscale scores, with lower scores reflecting better function. Time includes baseline and posttreatment, and treatment includes cognitive processing therapy (CPT) and Sudharshan Kriya yoga (SKY).

Changes in cognitive performance

There was no significant Treatment Group x Time interaction for any cognitive domain scores (Figure 2, Supplementary Figure S1, Supplementary Table S3). Participants displayed main effects of treatment-related improvements (i.e., time) in three out of the seven cognitive measures (PAL total error, MOT latency, and RVP mean latency), as well as an improvement in the derived overall cognitive composite score (Table 1; Supplementary Table S4 for treatment-specific changes). Notably, performance in spatial working memory worsened posttreatment (Table 1; Supplementary Figure S1, Panel D; Supplementary Table S2). Across most measures of cognitive function, participants in both the SKY and CPT conditions showed comparable deviations from normality at baseline and posttreatment (Supplementary Table S5). However, all results remained the same following nonparametric testing. Furthermore, after running an ANCOVA with depres-

TABLE 1 Changes in mood and cognitive measures following treatment (baseline to posttreatment)

Output	M_{diff}	95% CI	*d*	*SE*	95% CI	*t*[a]	*df*	*p*
Mood								
CAPS-5	6.90	[3.71, 10.10]	0.55	0.12	[0.28, 0.81]	4.32	61	< .001
BDI-II	10.01	[5.93, 14.09]	0.62	0.12	[0.35, 0.89]	4.91	61	< .001
Cognitive (CANTAB)								
Composite[b]	0.24	[0.13, 0.34]	0.58	0.14	[0.30, 0.84]	4.53	61	< .001
PAL total errors	0.07	[0.03, 0.10]	0.51	0.12	[0.24, 0.77]	4.01	61	< .001
MOT								
Mean latency	1.34	[0.75, 1.94]	0.57	0.14	[0.30, 0.84]	4.50	61	< .001
Mean error	−0.01	[-0.01, 0.00[c]]	−0.25	0.12	[-0.50, 0.00]	−1.98	61	.053
RVP								
Mean latency	0.42	[0.13, 0.70]	0.37	0.14	[0.11, 0.63]	2.92	60	.005
Total false alarms	−0.01	[-0.06, 0.04]	−0.05	0.17	[-0.30, 0.20]	−0.37	61	.713
SWM								
Between errors	−0.06	[-0.09, -0.03]	−0.46	0.13	[-0.72, -0.19]	−3.58	61	< .001
Strategy	−0.05	[-0.07, -0.03]	−0.69	0.17	[-0.97, -0.41]	−5.43	61	< .001

Note. $N = 62$. All reported scores reflect the change in raw scores following treatment (i.e., main effect of time: baseline–posttreatment). PTSD = posttraumatic stress disorder; CI = confidence interval; *df* = degrees of freedom; CAPS-5 = Clinician-Administered PTSD Scale for *DSM-5*; BDI-II = Beck Depression Inventory–II; CANTAB = Cambridge Neuropsychological Test Automated Battery; PAL = CANTAB Paired Associates Learning; MOT = CANTAB Motor Screening Task; RVP = CANTAB Rapid Visual Information Processing; SWM = CANTAB Spatial Working Memory.

[a]Student's *t* test.

[b]Average of the *Z* score changes in all seven outputs from: $\text{sum}(Z_{PAL\_errors} + Z_{MOT\_latency} + Z_{MOT\_errors} + Z_{RVP\_latency} + Z_{RVP\_false\ alarms} + Z_{SWM\_errors} + Z_{SWM\_strategy})/7$

[c]The actual upper bound for the confidence interval was 6.91×10^{-5}

sive symptoms as a covariate, the results did not change (Supplementary Table S6).

Associations between cognition and PTSD symptoms

To explore whether PTSD symptom reductions were associated with cognitive improvement, a linear regression was conducted using CAPS-5 total severity score change as a predictor of change in the cognitive composite score. Overall, larger symptom reductions were significantly associated with higher degrees of cognitive improvement, $B = -0.01$ ($SE = 0.00$), $\beta = -.25$, $p = .047$ (Figure 3; Supplementary Table S7). Given the exploratory nature of this analysis, we also included treatment condition (SKY vs. CPT) and CAPS-5 change in an expanded model. In this model (Supplementary Table S7), the main effect of CAPS-5 change was no longer significant, $B = 0.001$ ($SE = 0.01$), $\beta = .03$, $p = .875$, nor was the main effect of treatment significant, $B = -0.11$ ($SE = 0.12$), $p = .359$. Though the CAPS-5 Change x Treatment interaction term was nonsignificant, $B = -0.01$ ($SE = 0.01$), $p = .059$, the results indicate a possible difference in the strength of the association between the two groups. Consistent with this finding, exploratory robust correlations indicated that the association between PTSD symptom change and cognitive change was significant among CPT participants, $r_{ss} = -.43$, $p = .015$, but not among SKY participants, $r_{ss} = .05$, $p = .776$.

DISCUSSION

In this study, we investigated the effects of PTSD treatment on cognition. Two treatments were used: a first-line, evidence-based trauma-focused psychotherapy (CPT) and an experimental breathing-based yoga meditation practice (SKY). We have previously demonstrated similar efficacy for both treatments in treating PTSD (Bayley et al., 2022). As hypothesized, cognition, as assessed using a cognitive composite score derived from selected CANTAB tasks, showed overall improvement following treatment. This improvement was driven by improvements in episodic visual memory (PAL) and faster response times on tasks of visual sustained attention (RVP) and motor learning (MOT). To contextualize potential practice effects, an individual-level reliable change analysis was conducted (Supplementary Table S2). The resulting classifications align with the main outcomes for symptoms of depression and PTSD, indicating that a meaningful subset of participants experienced improvements exceeding measurement error, whereas a smaller subset worsened or showed no reliable change. For cognitive measures, although the results indicated mostly no change across time, it is important to note

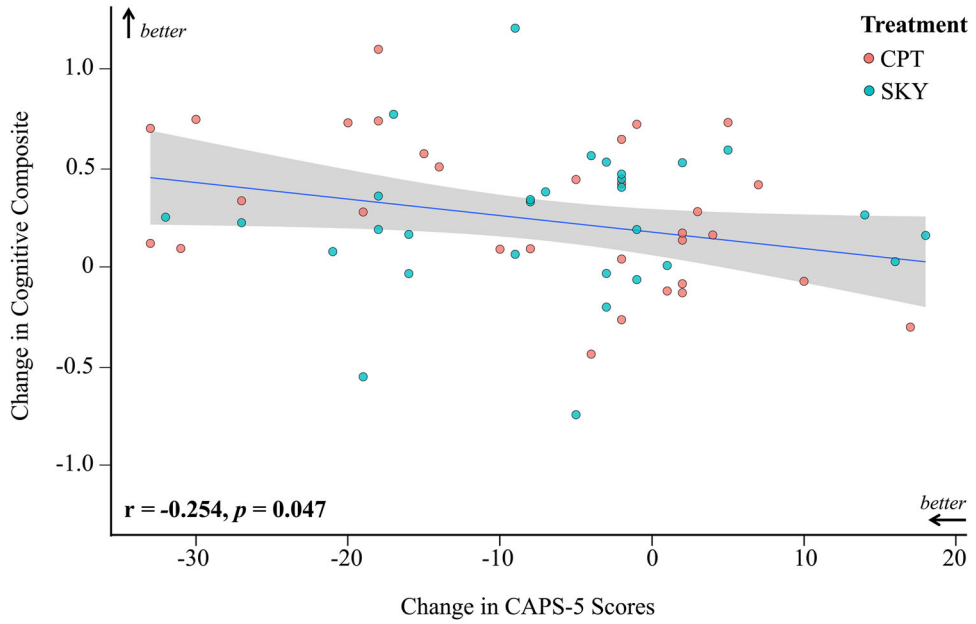

FIGURE 3 *Associations between changes in cognitive composite scores and posttraumatic stress disorder (PTSD) symptom measure scores*
Note: PTSD score change was calculated as the posttreatment-baseline difference in Clinician-Administered PTSD Scale for *DSM-5* (CAPS-5) scores, such that lower scores reflect improvement in symptoms. Cognitive composite score change was calculated as the baseline–posttreatment difference in Cambridge Neuropsychological Test Automated Battery (CANTAB) cognitive composite scores, such that higher scores reflect improvement in cognitive functioning.

that we could not locate the test–retest reliabilities for some of the selected CANTAB tasks in the literature, and those that could be found were not well-matched to our study design and sample. Indeed, further validation of test–retest reliability metrics is needed for the CANTAB (Karlsen et al., 2022). Changes in cognition did not differ between the two treatment groups. Also, as hypothesized, overall, the degree of treatment-related improvement in cognitive function was related to treatment-related improvements in PTSD symptom severity. However, this only reached statistical significance in the CPT condition.

Participants in the CPT group demonstrated domain-specific improvements in episodic memory, motor learning, and sustained attention, consistent with prior research showing CPT's efficacy in modifying trauma-related cognition to enhance cognitive functioning (Watkins, 2018). Similarly, cognition among participants in the SKY group generally improved in the same domains. This aligns with recent research indicating the positive role of yoga treatments for cognitive function, though this has not previously been investigated in a sample with a clinical PTSD diagnosis. For instance, yoga seems to mostly enhance cognitive function in elderly samples (Eilat-Adar et al., 2023; Giridharan et al., 2024), whereas no impact on cognition has been observed in younger samples (Aleksandra et al., 2021), suggesting possible floor effects whereby yoga may only exert positive effects on cognition among individuals who already have deficits.

The decline in SWM performance in both groups was surprising and likely reflects residual deficits in visuospatial processing within the context of broader cognitive improvement. Few prior studies have examined SWM function changes following PTSD treatment. The available studies have focused on general cognitive improvements (Boyd et al., 2019), suggested nonspecific cognitive effects (Susanty et al., 2024), or targeted related but distinct cognitive processes (Bomyea et al., 2015). Therefore, it is difficult to account for our observation of worse performance in spatial memory without further targeted research. Most published PTSD treatment studies have focused on symptom changes alone without consideration of cognitive changes. Given that individuals seeking treatment for PTSD are often those most likely to exhibit cognitive deficits (Scott et al., 2015), it is also important to not just characterize cognitive deficits associated with PTSD but also investigate shifts and potential reversals in one's cognitive capacity as a result of treatment. Previous research has shown similar efficacy for a trauma-focused psychotherapy and a yoga-based treatment in improving symptoms of PTSD, depression, and negative affect (Bayley et al., 2022), as well as sleep (Mathersul et al., 2023) and emotion regulation (Mathersul et al., 2022). Here, we

build upon these findings by demonstrating that these treatments are also similar in improving cognition.

This study has a number of strengths, including the novelty of its longitudinal design, which stands apart from the usual cross-sectional design used to evaluate the cognitive impact of PTSD. Moreover, this pre–post assessment structure allowed for a robust evaluation of cognitive changes over time. There are a number of limitations. Given the dropout rate of approximately 27%, our sample was limited in size. This could be due to the length of committed participation required, which should be considered and evaluated in future studies. On a different note, cognitive function is more typically measured in the clinic using standardized clinical batteries, such as the Wechsler Adult Intelligence Scale (Wechsler, 2024), to index general cognitive ability and include knowledge-based and fluid domains of cognition. For the present short retest interval and domain-level hypotheses, we prioritized the CANTAB's computerized tasks (e.g., learning/memory, attention, processing speed), which provide fine-grained indices and alternate versions of the same task to prevent practice effects which are well-suited to detecting modest change over brief intervals. On the other hand, the CANTAB originated in primate research, relying on touchscreen-based, nonverbal tasks, which may not comprehensively evaluate cognitive domains involving verbal memory or language-based processing, thereby engaging different neural networks than traditional verbal tasks. Furthermore, individuals with significant PTSD symptom levels were included regardless of whether they met the full criteria for PTSD or whether their symptoms were their primary concern, making this a broadly representative sample. However, in justification of our choice of participants, many individuals have clinically significant symptoms warranting treatment yet fall short of meeting the diagnostic criteria (Dalgleish et al., 2020).

Future studies could address the stability of cognitive improvement following PTSD treatments and their correlation with sustained PTSD symptom reduction. A further research direction could usefully explore biomarkers (e.g., hippocampal–prefrontal connectivity) to predict individual responses to cognitive-focused versus somatic-focused therapies. The efficacy of combining complementary treatments to target both physiology and psychology may open an avenue to multimodal therapeutic treatments for PTSD. These future directions would significantly contribute to the field's understanding of the complex interplay between PTSD treatment modalities, symptom reduction, and cognitive functioning.

AUTHOR CONTRIBUTIONS

Zulkayda Mamat: Writing - original draft; Writing - review & editing; Formal analysis; Visualization. **Danielle C. Mathersul**: Investigation; Writing - review & editing. **Peter J Bayley**: Conceptualization; Writing - review & editing; Funding acquisition; Investigation; Supervision.

AUTHOR NOTE

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OPEN PRACTICES STATEMENT

The preregistration for this study can be accessed at https://clinicaltrials.gov/study/NCT02366403. Neither the data nor the materials have been made available on a permanent third-party archive; requests for the data or materials can be sent via email to Peter J. Bayley at peter.bayley@va.gov.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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