Research paper

The power of appraisals in predicting PTSD symptom improvement following cognitive rehabilitation: A randomized clinical trial

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ABSTRACT

Keywords:
Cognitive training
Cognitions
Trauma
Neuropsychological functioning
TBI

Background: Patients with PTSD often voice concern over their perceived change in cognitive functioning. However, these negative appraisals do not always align with objective neuropsychological performance, yet are strongly predictive of PTSD symptom severity and self-reported functional impairment.

Methods: The present study involves a secondary analysis examining the role of appraisals of a subsample of 81 adults with full or subthreshold PTSD on treatment outcomes in a randomized controlled trial investigating the effectiveness of a cognitive rehabilitation treatment, Strategic Memory and Reasoning Training (n = 38), compared to a psychoeducation control arm, the Brain Health Workshop (n = 43). Neither condition addressed PTSD symptoms, focusing instead on cognitive skills training and psychoeducation about the brain.

Results: Intent-to-treat models showed statistically significant improvements for both groups on composite scores of executive functioning and memory. Additionally, both groups experienced clinically significant reductions in PTSD symptoms (assessed via the Clinician-Administered PTSD Interview) and the SMART group showed fewer negative appraisals about cognitive functioning following training. Change in appraisals of cognitive functioning was associated with change in PTSD as well as change in quality of life, with no differential associations based on group status. In contrast, neurocognitive test score changes were not associated with change in symptoms or functional outcomes.

Limitations: We did not collect data on other appraisals (e.g., self-efficacy), which could have further elucidated pathways of change.

Conclusions: Our findings suggest that interventions that do not directly target PTSD symptoms can lead to PTSD symptom change via change in appraisals of functioning.

The role of appraisals in posttraumatic stress disorder (PTSD) is substantial. The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013) emphasizes the significance of negative appraisals through the addition of two new diagnostic criteria: criterion D2 (“persistent negative beliefs and expectations about oneself or the world”) and criterion D3 (“persistent distorted blame of self or others for causing the traumatic event or for resulting consequences”). These additions align with the long-standing cognitive theory of PTSD (Ehlers and Clark, 2000), which described commonly observed appraisals of self-blame and beliefs that the world is a dangerous place in patients with PTSD. The mechanistic role of negative appraisals in both development and recovery from PTSD has been documented in countless studies (see Brown et al., 2019 for a review). Although this literature is largely cross-sectional, some PTSD treatment studies have shown that change in cognitions predicted improvement in PTSD symptom severity (Brown et al., 2019). PTSD-related appraisals typically involve negative beliefs about the world (“No one can be trusted,” “Nowhere is safe”) and the self (“I am to blame”). Critical to this theory and the present study is the trauma survivor’s perception that the trauma has “ruined” them (Benight et al., 2017). Social cognitive theorists (Benight and Bandura, 2004; Benight et al., 2015) have highlighted the importance of negative appraisals related to trauma survivors’ performance or competence post-trauma, most notably self-efficacy about the ability to cope with traumatic events.
stress symptoms. Individuals with PTSD also hold appraisals about their cognitive functioning. Patients are often aware of a change in cognitive function that accompanies trauma or the development of PTSD and voice concern about memory and attention difficulties. Indeed, PTSD has been consistently associated with mild and subtle impairments in attention, working memory, verbal memory, processing speed, and executive functions (e.g., Polak et al., 2012; Samuelson, 2011; Scott et al., 2015). Notably, and perhaps surprisingly to many patients, performance is often found to be in the average to slightly below average range (e.g., Gilbertson et al., 2001; Twamley et al., 2009; Samuelson et al., 2006; Scott et al., 2015; Vasterling et al., 1998). Yet, self-perception of cognitive functioning has been found to be unrelated to objective neurocognitive performance (O’Neil et al., 2019; Spencer et al., 2010) or mediated by PTSD symptom severity (Mattson et al., 2019), suggesting that the accuracy of these appraisals may be compromised in PTSD. A recent study of veterans initiating PTSD treatment showed that approximately one-half to one-third of patients exhibited clinically significant cognitive deficits, whereas almost all self-reported cognitive problems. Additionally, veterans reported many subjective complaints that were not validated by objective cognitive tests (O’Neil et al., 2019).

Perceptions of cognitive impairment are also highly related to psychological distress in patients with PTSD and other mental health problems (Binder et al., 1999; Hart et al., 2000; Samuelson et al., 2017a; Samuelson et al., 2017b; Spencer et al., 2010). In a study of Iraq and Afghanistan veterans with PTSD, perception of cognitive problems, rather than objective neuropsychological performance, was associated with poorer functional outcomes (Samuelson et al., 2017a). Notably, in mild traumatic brain injury, a condition highly comorbid with PTSD (Hoge et al., 2008; Tanielian et al., 2008), associations between subjective report of cognitive functioning and objective performance are largely non-significant or weakly correlated (Belanger et al., 2016; Drag et al., 2012; French et al., 2014; Jak et al., 2015; Spencer et al., 2010), and both cognitive deficits and complaints observed in mild TBI are largely explained by the presence of PTSD (O’Neil et al., 2014; Storzbach et al., 2015; Vasterling et al., 2012). Importantly, negative appraisals of cognitive functioning are not necessarily specific to PTSD and are seen in depression, TBI, pain, and dementia (e.g., Drag et al., 2012; Hart et al., 2000; Mendona et al., 2016; Serra-Blasco et al., 2019). In individuals with PTSD, these appraisals appear to be related to other negative posttraumatic cognitions about the self and world and one’s efficacy in managing posttraumatic symptoms, suggesting that negative perceptions of cognitive functioning may represent a specific type of posttraumatic self-appraisal (Samuelson et al., 2017b). This nascent body of research demonstrating the negative impact of potentially inaccurate appraisals on functional outcomes and symptom severity raises interesting questions for PTSD treatment. It remains to be seen if targeting this type of negative appraisal in cognitive therapies, in addition to the classic posttraumatic cognitions regarding the self and the world, would improve PTSD treatment response. Or, if targeting cognitive functioning through cognitive rehabilitation could improve the accuracy of appraisals.

Cognitive rehabilitation (CR) interventions have been successfully used with a variety of psychiatric disorders involving cognitive impairments including schizophrenia, depressive disorders, and attention deficit disorder (e.g., Cortese et al., 2015; Kaneko and Keshavan, 2012; Motter et al., 2016). Little research has applied CR to PTSD, although some studies have little to improve cognitive functioning in comorbid PTSD and TBI given their frequent comorbidity and overlaying cognitive deficits. These studies typically involve compensatory approaches, in which the emphasis is on managing cognitive deficits through the learning and application of new skills. One notable example of a compensatory intervention is Cognitive Symptom Management and Rehabilitation Therapy (CogSMART; Twamley et al., 2014), a manualized 12-week intervention developed for veterans with TBI. Recently, researchers have added components of CogSMART to Cognitive Processing Therapy (CPT; Jak et al., 2019), an evidence-based trauma processing therapy, for patients with comorbid PTSD and TBI. Compared to the group that received CPT alone, the group that received both CPT and CogSMART showed significantly greater improvements on tests of attention and learning, verbal learning and memory, and executive functioning with small to medium effect sizes, in addition to reductions in PTSD symptoms (Jak et al., 2019). However, this study did not determine if improvements in cognitive functioning were associated with changes in appraisals about cognitive functioning, or if cognitive appraisal changes or cognitive test performance improvements were associated with PTSD symptom improvement.

In contrast to compensatory treatments, “top-down” remediation interventions aim to re-train the deficient skills rather than replace them with other strategies. Top-down approaches focus on activation of higher-order systems, targeting control processes mediated by the prefrontal cortex to guide goal-directed behavior. These higher-order systems involve executive functioning, strongly linked to PTSD-related impairments (Aupperle et al., 2012; Polak et al., 2012). An example of this type of approach is Strategic Memory and Advanced Reasoning Training (SMART; Chapman and Gamino, 2008), which targets remediation of executive functions by teaching metacognitive strategies.

One challenge of the use of CR interventions among patients with PTSD is that they can be difficult to attract patient buy-in because they do not directly target PTSD symptoms and often involve repetitive practice. As a result, participants may doubt the relevance of the interventions and show lower motivation to engage in such treatment (Fine et al., 2018; Samuelson et al., 2015). Alternatively, they can be appealing to patients with PTSD, as trauma processing interventions for PTSD are often experienced as daunting and associated with high levels of drop-out (Najavits, 2015). There may be the potential for both primary gains (improving neurocognitive functioning) and secondary gains (reducing PTSD symptoms) through CR. The notion of targeting neurocognitive functions that are theoretically linked to PTSD, rather than PTSD symptoms directly, follows the Research Domain Criteria framework set forth by National Institute of Mental Health (NIMH), which recommends that interventions engage the mechanisms by which symptom change occurs (Insel et al., 2010).

Recently, researchers have tested the effectiveness of a computerized CR program, a hybrid of the remediation trainings Lumosity and MyBrainSolutions, in improving neurocognitive functioning and reducing PTSD symptoms in a sample of 97 trauma-exposed adults admitted to emergency rooms (Ben-Zion et al., 2018). Compared to control group participants engaging in web-based gaming or reading tasks, the cognitive training group showed superior gains in cognitive flexibility performance and reductions in PTSD symptoms six months post-trauma. In addition, improvement in cognitive flexibility was correlated with PTSD symptom improvement. Importantly, given the need for focus on mechanisms of symptom change, these authors also established cognitive flexibility to be a key biomarker associated with PTSD severity extracted from over 200 markers (Ben-Zion et al., 2020). These findings highlight the success of a remediation-focused CR early intervention in promoting recovery from PTSD symptoms in addition to cognitive gains, as well as establishing a neurocognitive marker as a mechanism by which change occurs.

We have recently reported on the effectiveness of the remediation training SMART on cognitive outcomes in individuals with mTBI and/or PTSD (Samuelson et al., 2020). SMART aims to teach metacognitive strategies to enhance cognitive rehabilitation management through goal setting and inhibiting distracting or irrelevant stimuli. It prioritizes deeper level synthesis of information to obtain the “gist” while encouraging fluid and flexible thinking (Chapman et al., 2015, 2016). Delivered in a group format, the traditional 15-hour training has demonstrated improvement in cognitive functioning relative to control groups in samples of participants with TBI (Cook et al., 2014; Cook et al., 2015; Han et al., 2018; Vas et al., 2011; Vas et al., 2016) as well as cognitively healthy individuals (Anand et al., 2010; Chapman et al., 2015).
Given the important role of appraisals in PTSD, it is possible that change in appraisals of cognitive functioning, beyond actual change in neurocognitive performance, may predict symptom reduction and functional outcomes changes. Several studies have shown that change in posttraumatic cognitions (e.g., appraisals of self-blame and the world being a dangerous place) predicted reduction in PTSD symptoms in cognitive behavioral therapy (Ehlers et al., 2005; Ehlers et al., 2014; Foa et al., 1999; Kleim et al., 2013). These studies highlight that reduction of negative appraisals serves as the active mechanism of change in PTSD treatment. However, to our knowledge, no studies have examined appraisals regarding cognitive functioning in PTSD-targeted treatments or CR treatments for PTSD.

1. Methods

The study was a double-blinded, parallel arm randomized clinical trial (RCT) conducted in the United States. The study was registered after data collection but prior to data analysis (NCT04554537). A sample of 108 adults with full or subthreshold PTSD were recruited and evaluated for the study, with 81 participants randomized to either SMART or the comparison group, BHW. This subset of PTSD+ participants was taken from the larger study (N = 144 recruited, 128 eligible and randomized) examining the effectiveness of SMART on individuals with mild TBI, PTSD, or both. Inclusion criteria were English-speaking adults between the ages of 18 to 65 years. The larger study allowed for diagnosis of either mild or moderate TBI (as defined by Ohio State University TBI Identification Method; Corrigan and Bogner, 2007) and/or diagnosis of subthreshold or full PTSD (as determined by Clinician-Administered PTSD Scale for DSM-5; CAPS-5; Weathers et al., 2018); the present analysis required PTSD or subthreshold PTSD diagnosis. Exclusion criteria included self-report on a phone screen interview of pre-existing cerebral palsy, stroke, pervasive developmental disorder, intellectual disability, epilepsy, psychotic disorder, bipolar disorder, or a current alcohol or drug use disorder. Further exclusion criteria included poor effort on the Test of Memory Malingering (TOMM; Tombaugh, 1996), a test of symptom validity administered at the first visit, and involvement in neuropsychological testing or cognitive training in the past three months as this could introduce practice effects. Participants were asked to refrain from using alcohol or non-prescription drugs, including marijuana unless medically prescribed, on days of testing. We did not require clinical impairment on neurocognitive tests or self-reported cognitive problems.

We grouped together participants with full and subthreshold PTSD given prior research showing comparable levels of impairment in occupational and social functioning and distress (e.g., Schützwohl and Maercker, 1999; Stein et al., 1997) as well as response to treatment in prior RCTs (e.g., Maercker et al., 2006). Eighteen participants were receiving therapy at the time that they were screened for the study, and 14 of these participants were receiving treatment specifically for PTSD; all participants had been engaged in these therapies for longer than three months. We allowed for concurrent psychotropic use and included it as a covariate in all models given its influence on neurocognitive functioning. Forty percent of the SMART group and 35% of the BHW group reported current psychotropic use, $\chi^2 = 0.20$, $p = .66$.

Participants were primarily recruited through community flyers and newspaper or Craigslist ads (58%), postings at a mid-sized Western university (20%), or a registry of individuals who expressed interest in participating in trauma-focused research studies (22%). The mean age of participants was 41.7 ($SD = 12.8$), and the majority were female (61%) and had received at least a Bachelor’s Degree (51%). More demographic information is provided in Table 1.

Fig. 1 provides a Consolidated Standards of Reporting Trials (CONSORT) flow diagram. The diagram includes numbers for both the overarching study as well as the current study sample. Of the 108 participants in the current study sample who were consented and completed the baseline interview, 16 participants did not meet eligibility criteria and 11 participants dropped out prior to testing and randomization. Reasons for ineligibility included failure on the TOMM ($n = 6$), report of severe TBI or bipolar disorder ($n = 2$), and failure to meet diagnosis of PTSD or subthreshold PTSD on the CAPS-5 ($n = 8$). Of the final group who completed testing and randomization ($N = 81$), 38 participants (47%) were randomized to the SMART group and 43 randomized to the BHW group (53%). Eight participants dropped out prior to starting the training, six dropped out during the training, and one person dropped out prior to Time Point 2 (TP2) assessment.
The mean time since participants witnessed violence or death (7%, CAPS-5 gold standard) was incurred during motor vehicle accidents; 26% were sports-related accidents or injuries; 12% resulted from the person being physically assaulted; 12% were related to military activities (e.g., blast injuries, IED explosions); and 29% involved other types of accidents (e.g., falls, hitting head on stationary objects).

2.4. Ohio state university TBI identification method (OSU-TBI-IM; Corrigan and Bogner, 2007)

At the eligibility interview, the trained clinical evaluator assessed lifetime TBI history with the OSU-TBI-IM, a structured interview. The interview requires the participant to recall all injuries involving a blow to the head or neck, fall, blast exposure, or vehicular accident that may have caused an injury to the brain. For each injury, the nature of altered consciousness is assessed. The OSU-TBI-IM demonstrates strong inter-rater reliability and predictive validity (Corrigan and Bogner, 2007). Participants are classified as sustaining an mTBI if loss of consciousness (LOC) or alteration in consciousness for all injuries was less than 30 minutes, and moderate TBI if any injury involved LOC between 30 minutes and 24 hours. The majority of the current sample had a lifetime history of mild TBI (89%), which was not entirely surprising given an inclusion criterion of the study was either mTBI history or PTSD, and recruitment directly targeted these two groups. In the overall sample, 79% reported TBI events incurred from a single event (e.g., car accident, recreational accident) while 31% reported repeated events (e.g., sports collisions, blast exposure, etc.). Twenty-one percent of events reported were incurred during motor vehicle accidents; 26% were sports-related accidents or injuries; 12% resulted from the person being physically assaulted; 12% were related to military activities (e.g., blast injuries, IED explosions); and 29% involved other types of accidents (e.g., falls, hitting head on stationary objects).

2.5. Cognitive symptom self-report questionnaire (CSRQ; Spina et al., 2006)

The CSRQ - Cognitive (CSRQ-Cog) subscale was used to measure self-perception of cognitive problems. It consists of 10 items, self-reported over the prior two weeks, pertaining to memory and attention concerns and includes items such as “I have felt I have a good memory” and “I have been able to remember numbers.” Concurrent validity has been established (Spina et al., 2006) through significant correlations with the Cognitive Failures Questionnaire (Broadbent et al., 1982). Cronbach’s alpha was acceptable (α = .74) for this sample.

2.6. World health organization quality of life scale (WHOQOL-BREF; WHOQOL Group, 1998)

The WHOQOL-BREF consists of 26 items measuring quality of life across four domains: physical health, psychological health, social relationships, and environment. The WHOQOL-BREF has shown good discriminant, content, and construct validity as well as internal consistency and test-retest reliability (Guay et al. 2015; WHOQOL Group, 1998). We used the total score as a global measure of quality of life and functioning across multiple domains. Cronbach’s alpha (α = .86) was good for this sample.

2.7. Neuropsychological assessments

The detailed neuropsychological battery is described elsewhere (Samuelson et al., 2020) but included measures of verbal memory and learning (California Verbal Learning Test - Second Edition, CVLT-II; Delis et al., 2000; Logical Memory Test of the Wechsler Memory Scale – Fourth Edition, WMS-IV; Wechsler, 2008), visual memory (Brief Visuospatial Memory Test-Revised, BVMTR; Benedict, 1997), attention (Continuous Performance Test, CPT-3; Connors, 2016), working

**Table 1**

Baseline characteristics and comparisons between groups.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Overall</th>
<th>SMART (n = 38)</th>
<th>BHW (n = 43)</th>
<th>t or x²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (M, SD)</td>
<td>41.67</td>
<td>41.79</td>
<td>41.56</td>
<td>0.08</td>
<td>.94</td>
</tr>
<tr>
<td>Age (M, SD)</td>
<td>41.79</td>
<td>41.79</td>
<td>41.56</td>
<td>0.08</td>
<td>.94</td>
</tr>
<tr>
<td>Male gender (n, %)</td>
<td>30</td>
<td>16 (42.10)</td>
<td>14</td>
<td>1.45</td>
<td>.49</td>
</tr>
<tr>
<td>Male gender (n, %)</td>
<td>30</td>
<td>16 (42.10)</td>
<td>14</td>
<td>1.45</td>
<td>.49</td>
</tr>
<tr>
<td>Status: Number of civilian vs veteran or military (n, %)</td>
<td>44</td>
<td>20 (55.60)</td>
<td>24</td>
<td>0.28</td>
<td>.60</td>
</tr>
<tr>
<td>Status: Number of civilian vs veteran or military (n, %)</td>
<td>44</td>
<td>20 (55.60)</td>
<td>24</td>
<td>0.28</td>
<td>.60</td>
</tr>
<tr>
<td>Bachelor’s Degree or more education (n, %)</td>
<td>49</td>
<td>17 (47.30)</td>
<td>23</td>
<td>0.44</td>
<td>.51</td>
</tr>
<tr>
<td>Bachelor’s Degree or more education (n, %)</td>
<td>49</td>
<td>17 (47.30)</td>
<td>23</td>
<td>0.44</td>
<td>.51</td>
</tr>
<tr>
<td>TBI history (n, %)</td>
<td>72</td>
<td>30 (85.71)</td>
<td>42</td>
<td>6.42</td>
<td>.01</td>
</tr>
<tr>
<td>TBI history (n, %)</td>
<td>72</td>
<td>30 (85.71)</td>
<td>42</td>
<td>6.42</td>
<td>.01</td>
</tr>
<tr>
<td>Time since trauma in years (M, SD)</td>
<td>14.51</td>
<td>18.31</td>
<td>10.92</td>
<td>2.31</td>
<td>.03</td>
</tr>
<tr>
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<td>14.51</td>
<td>18.31</td>
<td>10.92</td>
<td>2.31</td>
<td>.03</td>
</tr>
<tr>
<td>Psychotropic use (n, %)</td>
<td>28</td>
<td>14 (40.00)</td>
<td>14</td>
<td>0.20</td>
<td>.66</td>
</tr>
<tr>
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<td>28</td>
<td>14 (40.00)</td>
<td>14</td>
<td>0.20</td>
<td>.66</td>
</tr>
<tr>
<td>Executive Functioning</td>
<td>0.1</td>
<td>0.04</td>
<td>-0.02</td>
<td>0.30</td>
<td>.77</td>
</tr>
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<td>0.1</td>
<td>0.04</td>
<td>-0.02</td>
<td>0.30</td>
<td>.77</td>
</tr>
<tr>
<td>Composite Score</td>
<td>(1.0)</td>
<td>(0.96)</td>
<td>(1.03)</td>
<td></td>
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</tr>
<tr>
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<td>(1.0)</td>
<td>(0.96)</td>
<td>(1.03)</td>
<td></td>
<td></td>
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<tr>
<td>Memory Composite Score</td>
<td>0.10</td>
<td>0.08</td>
<td>0.11</td>
<td>-0.10</td>
<td>.92</td>
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<tr>
<td>Memory Composite Score</td>
<td>0.10</td>
<td>0.08</td>
<td>0.11</td>
<td>-0.10</td>
<td>.92</td>
</tr>
<tr>
<td>CAPS-5 Total Score</td>
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<td>32.94</td>
<td>34.56</td>
<td>-0.36</td>
<td>.72</td>
</tr>
<tr>
<td>CAPS-5 Total Score</td>
<td>34.09</td>
<td>32.94</td>
<td>34.56</td>
<td>-0.36</td>
<td>.72</td>
</tr>
<tr>
<td>CSRQ-Cognitive subscale score</td>
<td>(6.39)</td>
<td>(6.26)</td>
<td>(6.58)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CSRQ-Cognitive subscale score</td>
<td>(6.39)</td>
<td>(6.26)</td>
<td>(6.58)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>WHOQOL Total Score</td>
<td>74.29</td>
<td>77.58</td>
<td>71.79</td>
<td>1.52</td>
<td>.13</td>
</tr>
<tr>
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<td>1.52</td>
<td>.13</td>
</tr>
</tbody>
</table>


2. Measures

2.1. Life events checklist (LEC-5; Gray et al., 2004)

The LEC-5 is a traumas history measure that was administered prior to the CAPS-5. Participants were then asked to identify their worst event, and the CAPS-5 was administered in relation to that event. Criterion A re-experiencing symptom, one avoidance symptom, two symptoms arousing symptoms. Subthreshold PTSD (n = 21, 26%) is assigned if the participant meets diagnostic criteria for the re-experiencing symptom cluster and at least two other symptom clusters (Blanchard et al., 1994). Cronbach’s alpha for this sample was good (α = .80).

2.3. PTSD checklist for DSM-5 (PCL-5; Weathers et al., 2013)

This 20-item self-report questionnaire assessing self-reported PTSD symptom severity was administered. Scores range from 0-80 with higher scores representing higher symptom severity. The PCL-5 has shown strong test-retest reliability, internal consistency, and convergent validity in trauma-exposed samples (Blevins et al., 2015; Bovin et al., 2016). Participants completed this questionnaire using the same Criterion A event endorsed on the CAPS. Cronbach’s alpha was high (α = .93) for this sample.
memory (Paced Auditory Serial Addition Test, PASAT; Gronwall and Sampson, 1974; Digit Span test of the Wechsler Adult Intelligence Scale - Fourth Edition, WAIS-IV; Wechsler, 2008), processing speed (Coding and Symbol Search from WAIS-IV; Wechsler, 2008), strategic learning (Verbal Selective Learning Task, VSLT; Castel et al., 2002; Hanten et al., 2002), and executive functioning (Trail Making Test, Color-Word Interference Test, and Verbal Fluency Test from the Delis-Kaplan Executive Functioning System, D-KEFS; Delis et al., 2001). Some measures included alternate forms for TP2 testing to reduce practice effects: CVLT-II, BVMT-R, VSLT, and D-KEFS Verbal Fluency Test. Participants also completed the TOMM (Tombaugh, 1996) to assess cognitive effort and were excluded from the study if they scored below 45 on Trial 2, a cutoff shown to detect insufficient effort (Haber and Fichtenberg, 2006). The TOMM is a widely used, psychometrically sound measure of effort (Teichner et al., 2000).

3. Procedures

The study protocol was approved by the University of Colorado Colorado Springs Institutional Review Board and data were collected between August 2016 and May 2019. No changes to methodology were implemented after trial commencement. Informed consent was obtained from all participants. Interested participants first completed a phone screen interview to determine potential eligibility. If eligible at that stage, they were invited for an initial study visit interview and testing, conducted at a university research office. For the initial visit, participants were administered the OSU-TBI-IM, the CAPS-5, and the TOMM by trained graduate students in clinical psychology to determine eligibility. At a second visit, eligible participants were administered neuropsychological assessments that established the baseline measures for the study.

Training was initiated within one month of the baseline assessment, and post-training assessments were conducted within one month of completion of the training. At six months, participants completed self-report measures and neuropsychological tests only. Participants were compensated for each assessment visit.

Following the baseline testing sessions, participants were randomly assigned to either SMART or BHW by a project coordinator not involved in assessment or treatment, using a computer-generated randomizer and a 1:1 allocation ratio and simple randomization. Participants, outcome assessors, and data analysts were kept blinded to randomization allocation. The project coordinator enrolled participants and assigned interventions. The interventions were administered either at a university research office or at a university-affiliated clinic. SMART was delivered in small groups (n = 2 to 8) consisting of two 3-hour sessions over two days, followed by one 3-hour session a month later. Sessions focused on strategic attention, integrative reasoning, and cognitive control functions (Chapman and Mudar, 2014). Newspaper articles, stories, pictures, and audio or video clips were used to illustrate each strategy, and the application of strategies in daily life was emphasized and homework was assigned to practice skills between sessions. The Brain Health Workshop (BHW) has been used in multiple studies as a comparison training
4. Data analysis

This paper describes analysis of secondary outcomes: PTSD symptoms, self-report of cognitive functioning, and quality of life. Findings regarding the RCT’s primary outcomes—performance on neuropsychological tests—are described elsewhere (Samuelson et al., 2020). Descriptive statistics (t-tests and chi-square tests) were first conducted to examine potential differences between the treatment groups on all baseline characteristics, between study completers and drop-outs, and between participants with full versus subthreshold PTSD. Preliminary Pearson correlations provide bivariate relationships between all variables. A principal component analysis (PCA) of all neuropsychological test variables was conducted on the entire sample for the larger study (including those who did not meet criteria for PTSD), in order to reduce the number of statistical comparisons. Details are described elsewhere (Samuelson et al., 2020), and the two extracted components (memory and executive functioning) were used in the present study.

Linear mixed-effects models (LMM) were used to examine within- and between-groups differences in neuropsychological functioning, PTSD symptomatology, and quality of life over time (from baseline to end of training). All analyses were conducted using the intent-to-treat (ITT) principle, meaning that all randomized subjects were included in the analyses. These models utilized restricted maximum likelihood estimation, which tolerates observations that are missing at random (MAR). The Satterthwaite correction was used to adjust degrees of freedom for smaller samples. For models testing group differences over time, fixed effects included time, group, and a group x time interaction. As psychotropic use could potentially be related to neuropsychological outcomes, we included this as a covariate in analyses. Given the mean length of time since trauma (M = 14.5) and large variability (SD = 13.8, range 10 months to 47 years) in this sample, we also included this variable as a covariate. A random intercept of participant and a random slope of time were included. All models used an unstructured covariance matrix. Statistical significance was evaluated at a two-sided alpha of p < .05. To control for Type I Error, a False Discovery Rate (FDR) was applied. LMM were assessed over two time points (baseline to post-training) on all measures except for the PCL-5. Because the PCL-5 was also administered at Time Point 3 (TP3; 6-month follow-up), we present LMM for all three time points for that variable only. Prior to analysis, all variables were standardized to the baseline score. Thus, parameter estimates reflect standardized differences and can be used as estimates of effect size.

Following primary ITT analyses, we examined group differences in reliable change (RCI; Jacobson and Truax, 1992) to determine if changes seen in both groups were clinically significant. For these analyses, trajectories of change through unconditional (i.e., random intercept and slope) LMM served as the change score. Pearson chi-square was used to test whether groups differed in reliable change.

In order to examine mechanisms of change in CAPS-5 symptoms and quality of life, two ordinary least squares (OLS) multiple regressions were used. Trajectories of change were extracted for each individual participant from predicted values of unconditional LMM (with a fixed effect of time, and random intercept for participant and random slope of time). These change scores were then used as either independent or dependent variables in the models. Change in CAPS-5 symptoms and change in quality of life served as dependent variables, while change in CSRQ-Cog, memory composite performance, and executive function composite performance served as the independent variables. We also examined the interaction between change in CSRQ-Cog and group on change in CAPS-5 symptoms to determine if change in perceived cognitive problems differentially predicted change in PTSD symptoms based on group status.

A power analysis indicated that there was sufficient power to detect a small to moderate effect (Cohen’s d = .31) for the group x time interactions for PTSD symptom improvement on the CAPS-5 in the LMM and a small effect in the OLS regressions (approximate Cohen’s d = .28) involving variables predicting change in PTSD symptoms. A previous study using a cognitive training intervention found a moderate to large effect size of Cohen’s d = .63 in improvement of PTSD symptoms on the CAPS-5 (Ben-Zion et al., 2018). Of note, the larger study from which this secondary analysis was drawn (Samuelson et al., 2020) comprised of a sample of 144 participants (128 randomized), the basis of which was determined through a power analysis for linear mixed models with three timepoints to demonstrate cognitive improvements in participants with PTSD involved in cognitive rehabilitation (Ben-Zion et al., 2018). The study was closed when 144 participants were reached.

5. Results

Table 1 depicts demographic data for the total group, SMART group, and BHW group. There were no significant between-group differences in study variables at baseline. However, there was a significant difference between groups on mTBI status, with 87% of the SMART group and 100% of the BHW group reporting an mTBI history on the OSU-TBI-SF. As noted, for the overall study, mTBI and/or PTSD status were inclusion criteria so high rates were expected; however, given the random assignment, group differences were unexpected. Following evaluation of the models using the full sample size, we conducted sensitivity analyses removing the five participants without mTBI (presented below).

We also conducted t-tests or chi-square tests to assess for potential differences between the 65 participants who completed the training through post-assessment and the 16 participants who dropped out either before or during the training. No significant differences were observed (all p > .06). Finally, to ensure that participants with subthreshold PTSD were similar to those with full PTSD as they were forming one group, we conducted t-tests comparing groups on all variables of interest. The only significant difference between the groups was, unsurprisingly, CAPS-5 total score (p < .001); no other differences reached statistical significance (all p ≥ .187).

Results of the PCA are detailed elsewhere (Samuelson et al., 2020). The PCA extracted two primary components: one which consisted of primarily memory and learning and attention measures (CVLT-II Trials 1-5, CVLT-II Long Delay Free Recall, BVMT-R Trials 1-3 Total, BVMT-R Long Delay Free Recall, WAIS-IV Digit Span, WMS-IV Logical Memory Immediate and Delayed, CPT-3 Omissions, CPT-3 Commissions, VSLT Total score) and one consisting of executive functioning measures (PASAT Total score, WAIS-IV Processing Speed Index, D-KEFS Number-Letter Switching, D-KEFS Letter Fluency, D-KEFS Category Fluency, and D-KEFS Color-Word Inhibition/ Switching).

Means and standard deviations for the neuropsychological assessments and outcome measures at both time points can be found in Table 2. Correlations between primary variables are included in Table 3. CAPS-5 scores and self-reported PTSD symptoms on the PCL-5 were strongly correlated at Time Point 1 (TP1; r = .72, p < .001) and moderately correlated at TP2 (r = .60, p < .001). Self-reported cognitive functioning (CSRQ-Cog) and objective neuropsychological composite scores were not significantly correlated at TP1 (r = -.22 to -.07, p = .061 to .545), but were significantly negatively correlated at TP2 (r = -.40 to 0.42).
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5.1. Group differences over time: neurocognitive functioning

Linear mixed models first tested group x time changes in the SMART and BHW groups. Time since trauma and psychotropic medication use were included as covariates. Both groups improved on memory (\(|d| = 0.51, |t| = 4.99, p < .001\)) and executive function (\(|d| = 0.23, |t| = 2.64, p = .011\)), but there were no group x time effects (memory: \(|d| = 0.03, |t| = 0.17, p = .862\); executive function: \(|d| = 0.08, |t| = 0.68, p = .500\)).

Tests of reliable change showed that 32% (n = 12) of the SMART group and 28% (n = 12) of the BHW group demonstrated clinically significant change on executive functioning; there was not a significant difference between groups (\(\chi^2 = 0.13, p = .718\)). Clinically significant change was observed in 42% (n = 16) of the SMART group and 37% (n = 16) of the BHW group on memory tasks, with no significant difference between groups (\(\chi^2 = 0.20, p = .653\)).

5.2. Group differences over time: PTSD symptom severity

Both groups showed significant improvements on CAPS-5 (\(|d| = 0.95, |t| = 4.33, p < .001\); Fig. 2), although there was not a significant group x time effect (\(|d| = 0.28, |t| = 0.92, p = .360\)). Forty-four percent of the sample (equivalent in both groups) no longer met criteria for a PTSD diagnosis after the intervention. Of those with full PTSD at baseline, 39% no longer met diagnosis, 20% decreased to subthreshold PTSD, and 41% continued to meet criteria for full PTSD. Of those with subthreshold PTSD at baseline, 56% no longer met criteria for PTSD, 31% continued to meet criteria for subthreshold PTSD, and 13% increased to full PTSD. Reliable change calculations further demonstrated that 65% (n = 24) of the SMART group and 53% (n = 23) of the BHW group showed clinically significant improvements; there was not a significant difference between groups (\(\chi^2 = 1.06, p = .303\)).

Next, to determine if PTSD symptom reduction was maintained after six months, LMM examined PCL-5 scores at all three time points. Both groups showed significant improvements over time on PCL-5 (\(|d| = 0.50, |t| = 4.24, p < .001\)). The group x time effect (\(|d| = 0.28, |t| = 1.71, p = .092\)) was approaching significance, suggestive of a small effect size, for participants in the SMART group reporting greater reductions in PTSD symptoms. Mean self-reported PTSD symptoms decreased from 42.4 (SD = 16.4) to 34.3 (SD = 19.1) at TP2 and to 29.5 (SD = 19.7) at TP3. The SMART group showed a 15.7-point mean reduction (SD = 19.0) in PCL-5 score and the BHW group showed a 8.2-point mean reduction (SD = 22.0) between TP1 and TP3.

5.3. Group differences over time: subjective report of cognitive functioning

Both groups showed significant improvements over time on CSRQ-Cog (\(|d| = 0.67, |t| = 3.70, p = .001\)). Mean self-reported cognitive

![Fig. 2. Change in Clinician Administered PTSD Scale for DSM-5 (CAPS) mean scores for the SMART and BHW groups across time points.](image-url)
problems decreased from 34.1 (SD = 12.6) to 24.5 (SD = 16.9) from TP1 to TP2. The group x time effect (|d| = 0.58, |t| = 2.24, p = .029) was significant, with participants in the SMART group reporting more cognitive improvements (Fig. 3). There was a significant time (|d| = 0.39, |t| = 2.69, p = .010) but not group x time effect (|d| = 0.30, |t| = 1.43, p = .158) in quality of life. Mean quality of life increased from 74.3 (SD = 16.1) to 78.4 (SD = 16.8).

5.4. Group differences over time: sensitivity analyses

Because there were significant differences between the treatment groups on mTBI history, we conducted a series of sensitivity analyses where the five participants without mTBI were removed. The same pattern of findings were seen as with the full sample; there were significant changes over time across all study outcomes (all p ≤ .047), but no significant group x time interaction across outcomes (all p ≥ .119).

6. Mechanisms of change over time

Ordinary least squares (OLS) multiple regressions were conducted to assess relationships between change in objective and subjective report of cognitive functioning and change in PTSD symptoms and quality of life. Change in perception of cognitive functioning, but not change in objective memory or executive functioning performance, was associated with change in PTSD symptoms (β = .32, |t(74)| = 2.78, p = .007; Fig. 4). Similarly, change in perception of cognitive functioning, but not change in memory or executive functioning performance, was associated with change in quality of life (β = -.47, |t(74)| = 4.43, p < .001). There were no significant group effects (p = .735 for PTSD symptoms change, p = .918 for quality of life change); both SMART and BHW groups showed similar prediction patterns, where change in perception of cognitive functioning predicted change in clinical outcomes.

6.1. Mediation analyses: subjective and objective cognitive functioning as mediators of the relationship between PTSD symptom severity and quality of life

Given prior research showing that subjective report of cognitive functioning, but not objective performance, mediated the relationship between PTSD symptom severity and quality of life (Samuelson et al., 2017a) we conducted additional multiple mediation analyses to attempt to replicate these findings and to determine if these relationships are different following intervention. PTSD symptom severity (total CAPS-S score) served as the independent variable, subjective cognitive functioning (CSRQ-Cog) and the two composite measures of objective cognitive functioning (Executive Composite and Memory Composite) were tested as multiple mediators, and quality of life (WHOQOL) served as the dependent variable for TP1 and TP2 separately. SEM models mimicked the mediation model run in Samuelson et al. (2017a). In the TP1 model, a significant indirect effect of PTSD symptoms on quality of life was observed through subjective cognitive functioning (ab = .11, 95% CI: -0.23 to 0.01). No indirect effects were observed through either of the objective cognitive functioning variables (Executive Composite 95% CI: -0.02 to 0.11; Memory Composite 95% CI: -0.18 to 0.01). The same pattern of results was seen in the TP2 model, suggesting that participating in SMART or BHW did not affect the role of subjective cognitive complaints on quality of life.

6.2. Secondary and exploratory analyses

6.2.1. Completer case analyses

As ITT analyses are relatively conservative, we conducted completer case analyses as well (i.e., participants that completed all follow-up assessments). The pattern of results remained the same as the original linear mixed models (no significant changes in any paths or size of effects), providing additional evidence that findings were not spuriously due to the ITT approach.

6.2.2. Examination of differences in relationship between subjective report and objective cognitive performance pre- and post-training

Given the finding of a non-significant correlation between subjective report and objective cognitive performance at TP1 and a significant correlation at TP2, we ran a series of r-to-z transformation analyses to determine if the strength of correlation was different for TP1 versus TP2 and if so, it varied by group assignment. The correlation between CSRQ-Cog and the Executive composite was r = -.07 at TP1 and r = -.40 at TP2 which was a statistically significant difference (z = 2.01, p = .022), meaning that subjective report and objective executive functioning performance were significantly more closely aligned following training. Examining the treatment groups separately, the BHW group showed a significant change in strength of correlation, in which poorer perceptions were related to poorer functioning (r = -.11 to -.58, z = 2.25, p = .012) but the SMART group did not (r = -.02 to -.17, z = 0.57, p = .285). There was not a statistically significant difference in strength of correlation between CSRQ-Cog and the Memory composite (z = 0.81, p = .209) and neither group showed a significant change.

6.2.3. Examination of symptom clusters

Finally, we conducted a series of linear mixed models examining each of the four PTSD symptom clusters (CAPS-S summed score for reexperiencing, avoidance, negative alterations in cognitions and mood,
and hyperarousal). All four models exhibited the same pattern of results (all time effects, \( p < .001 \) to \( p = .017 \); all group x time effects, \( p > .05 \)), indicating that the treatments exhibited similar effects across symptoms.

7. Discussion

In a sample of adults with subthreshold or full PTSD, participation in a 9-hour intervention of cognitive training or a time-matched control group of psychoeducation showed statistically and clinically significant improvements in PTSD symptoms and quality of life. Improvements on memory and executive functioning performance were also observed, with no significant difference between treatment groups. The only significant difference between groups was in self-report of cognitive functioning, in which participants in the SMART group reported greater improvements. Change in perceptions of cognitive functioning were associated with changes in PTSD symptoms and quality of life. Our findings suggest that PTSD symptom improvement can be seen in interventions not directly targeting symptoms, and that change is partially attributable to appraisal processes. Whereas several PTSD evidence-based treatment studies have similarly shown that change in cognitions and appraisals predicted PTSD symptom change (Benight et al., 2019; Eilers et al., 2005; Eilers et al., 2014; Fox et al., 1995; Klein et al., 2015), this is the first study to assess appraisals of cognitive functioning.

We also replicated earlier work showing that perceptions of cognitive problems, but not objective neurocognitive performance, mediated the relationship between PTSD symptoms and quality of life (Samuelson et al., 2017a). Interestingly, while there was no significant relationship between subjective report of cognitive functioning and objective performance at baseline, consistent with some earlier research (O’Neill et al., 2019; Spencer et al., 2010), negative appraisals of cognitive functioning were significantly associated with poorer neurocognitive performance following treatment. Thus, following an intervention aimed at improving cognitive functioning, subjective report and objective performance became better aligned. The BHW group demonstrated a stronger degree of change in the correlation between self-report and objective executive functioning performance at TP1 and TP2 compared to the SMART group. This suggests that psychoeducation about cognitive functioning following head injury or psychiatric disorder onset and neuroplasticity may contribute to a more accurate evaluation of abilities that aligns with actual performance.

In addition, our findings support the potential of interventions that do not directly target PTSD symptoms to contribute to improved clinical and functional outcomes in trauma survivors with PTSD. Although we expected some degree of PTSD symptom improvement in the SMART group compared to the BHW group, we observed significant improvements in both groups, with no group x time effect. Notably, the PTSD symptom improvement observed in this study (approximately 10-point CAPS-5 change) is clinically meaningful, decreasing from mean overall levels of mild to moderate PTSD (\( M = 34.0 \)) to below cutoff scores suggestive of PTSD (\( M = 24.0 \)). A meta-analysis of psychotherapy for PTSD applied a reduction of 10-12 points as indicative of meaningful improvement on the CAPS-5 (Steenkamp et al., 2015). Following intervention, 44% of participants (equivalent in both treatment groups; 39% of those with full PTSD and 56% of subthreshold) no longer met diagnosis for PTSD. Importantly, PTSD symptom reduction was maintained after six months, with PCL-5 scores seeing a total reduction of 15.7 points for the SMART group and 8.2 points for the BHW group.

Interestingly, this degree of change in the SMART group approaches the 15.7 point decrease on CAPS-5 in a prior study of a computerized neurocognitive training intervention, in which cognitive flexibility changes predicted PTSD symptom changes. In contrast, our results showed larger PTSD symptom improvement, and appraisals of cognitive functioning, rather than objective neurocognitive performance, predicted PTSD improvement. A primary difference between the two studies involves time since trauma. The earlier study involved early intervention in recent trauma survivors recruited from emergency rooms, examining change in neuropsychological functioning and PTSD symptoms during a dynamic and changing period (Ben-Zion et al., 2018). The current sample consisted of adults with more chronic PTSD from events occurring between 10 months and 47 years prior. Our findings are particularly promising, as chronic PTSD has often been seen as less malleable in previous research (Hammer et al., 2004).

As noted, the parent study from which these secondary data were drawn examined the effectiveness of SMART in adults with a history of primarily mild TBI and/or a history of PTSD. As such, the majority (89%) of participants in this secondary analysis had an mTBI history which may have influenced results. Importantly, though, patients with mTBI, like those with PTSD, are often inaccurate in appraising their cognitive functioning, and perceptions do not always align with objective neurocognitive performance (Drag et al., 2012; French et al., 2014; Spencer et al., 2010). Patients with both PTSD and mTBI may be aware of the potential impact of their diagnoses on the brain and cognitive functioning and can be especially attuned to perceived changes or deficits.

Similar to findings in the parent study (Samuelson et al., 2020) with a larger sample including participants with only mild or moderate TBI, we observed statistically significant improvements in memory and executive functions in both groups for the current study, with 65% of the SMART group and 53% of the BHW group showing clinically significant improvements. Memory scores improved approximately one-half of a standard deviation (35-40% of sample exhibiting reliable clinical change), and executive functioning scores improved between one-fourth and one-half of a standard deviation (approximately 30% of sample exhibiting reliable clinical change). Alternate forms were utilized to minimize practice effects on some, but not all, measures. These improvements are similar to those observed in two prior treatment studies with participants with PTSD that utilized alternate forms with some (Nijdam et al., 2018) or all (Ben-Zion et al., 2018) measures. Although we anticipated superior changes in the SMART group compared to the BHW group, both groups showed comparable improvements, with the exception of self-reported cognitive problems, in which the SMART group reported greater improvements. Both SMART and BHW provide psychoeducation, which has been shown to be an efficacious intervention in the acute phase in reducing self-reported cognitive problems and postconcussive symptoms in patients with mTBI (Miller and Mittenberg, 1998; Ponsford et al., 2002; Snell et al., 2009), and in improving performance on a measure of attention and information processing speed performance (Cooper et al., 2017). As expectancies of poor recovery from cognitive changes due to mTBI is predictive of longer-term self-report of cognitive problems (Snell et al., 2015), psychoeducation aimed at modifying these expectancies and cognitive training to strengthen cognitive functioning may be similarly beneficial for patients with PTSD. In addition, our results extend this nascent TBI literature to indicate that psychoeducation alone can produce modest gains in neurocognitive performance for individuals with PTSD. Our results suggest that relatively benign interventions that emphasize psychoeducation about neuroplasticity and expectancies for cognitive functioning may modify appraisals and expectations, which in turn could contribute to objective cognitive improvements.

Notwithstanding the benefits of psychoeducation, it was surprising that both training groups showed similar improvements in PTSD symptoms and quality of life, which might suggest that expectancy effects were contributing factors. All participants were aware that they

Ben-Zion et al. (2018) also observed PTSD symptom improvements (5-point decrease on CAPS-5) in a prior study of a computerized neurocognitive training intervention, in which cognitive flexibility changes predicted PTSD symptom changes. In contrast, our results showed larger PTSD symptom improvement, and appraisals of cognitive functioning, rather than objective neurocognitive performance, predicted PTSD improvement. A primary difference between the two studies involves time since trauma. The earlier study involved early intervention in recent trauma survivors recruited from emergency rooms, examining change in neuropsychological functioning and PTSD symptoms during a dynamic and changing period (Ben-Zion et al., 2018). The current sample consisted of adults with more chronic PTSD from events occurring between 10 months and 47 years prior. Our findings are particularly promising, as chronic PTSD has often been seen as less malleable in previous research (Hammer et al., 2004).
were enrolled in an interventional study that aimed to improve their cognitive functioning, and we did not describe the BHW arm as a control or comparison group. As psychoeducation may have been perceived as an active intervention - as it can be - participants in both arms may have had expectancy effects that affected their self-reported ratings of symptom change. The use of a structured interview for PTSD attenuates that effect to some degree. Importantly, the study was not described to participants as being an intervention for PTSD or PTSD-related cognitive deficits. Trauma-exposed participants with and without a history of TBI were recruited and PTSD status was not described as an inclusion criterion. Thus, it is unlikely that the participants would have strong expectancy effects around the cognitive training improving PTSD symptoms, although they likely exhibited expectancy effects around their perceptions of improved cognitive functioning. This is not necessarily a weakness, though, as the mechanistic action of both training arms is in part to improve expectations, emphasizing that cognitive functioning is not static and that individuals can “train their brain” to improve cognition.

A number of limitations should be noted. First, to reduce participant burden, we unfortunately did not collect CAPS-5 data at TP3, which would have allowed for observation of maintenance of effects using a structured interview. However, self-report data from the PCL-5 indicated that PTSD symptom reductions were maintained. Second, we required participants to be stable in other psychiatric treatments (>three months) prior to enrolling in the study, but we did not require them to refrain from entering treatment after enrolling in the study. As most participants in the study had chronic PTSD, and the study window between baseline and end-of-treatment was only one month, it is unlikely that participants began another treatment during that period. It is possible, though, that entry into other treatments in the six months following may have contributed to maintained PTSD symptom reductions seen on the PCL-5. Third, the high rates of mTBI histories in this particular sample makes generalization to other PTSD samples without TBI more challenging. Finally, as PTSD symptom improvement was substantial for this short intervention that did not directly target PTSD, it would have been helpful to collect more in-depth diagnostic information (e.g., the Structured Clinical Interview of DSM Disorders) to better characterize this sample. It may be that this particular sample had lower rates of comorbid depression and anxiety which could impact treatment effects.

Future research should continue to examine mechanistic variables that underlie PTSD symptom change, particularly in treatments that do not target PTSD symptoms directly. Of particular relevance is self-efficacy, which has been defined as an appraisal of one’s abilities as it pertains to different situations (Bandura, 1997). Appraisals of cognitive functioning likely involve some degree of cognitive self-efficacy, or beliefs that one can manage cognitive demands and even improve one’s cognitive functioning. We did not collect cognitive self-efficacy data in this study, but we anticipate that this construct may be particularly relevant for PTSD patients as it involves beliefs about one’s ability to manage cognitive concerns as opposed to simply appraisals of cognitive functioning, as was measured here. We are not aware of a measure that specifically targets beliefs that one can improve one’s cognitive functioning, which may be a particularly useful construct in the context of CR as well as psychoeducation about neuroplasticity.

Results suggest some promising clinical implications. First and foremost, interventions that do not directly target PTSD but provide participants with memory skills, psychoeducation about neuroplasticity, and indirectly or directly target appraisals and self-efficacy, hold clinical promise for patients with PTSD. Unexpectedly, both the SMART and BHW training arms resulted in improvements in neurocognitive performance and clinical outcomes. Findings need to be replicated in further PTSD samples, but these results suggest that psychoeducation alone may be modestly helpful for patients with PTSD. Psychoeducation about neuroplasticity and the potential to positively change the brain can be powerful for patients with negative appraisals about their cognitive functioning, as well as those with objective deficits. Results highlight the importance of a unique posttraumatic cognition in PTSD – appraisals that the brain is damaged and that cognitive functioning has declined. Thus, in patients with disorders that affect cognitive functioning, like PTSD and TBI, cognitive-behavioral treatments targeting appraisals about cognitive functioning and brain changes may be particularly effective. Neurocognition-related appraisal work could be especially meaningful for patients preparing to undergo evidence-based trauma processing treatments for PTSD that rely to some degree on memory recall. To our knowledge, no previous research has directly addressed negative appraisals of cognitive functioning in PTSD interventions. Promising research with older adults suggests that this approach could be helpful in improving self-efficacy. Older adults who received cognitive restructuring aimed at promoting adaptive beliefs about memory coupled with memory skills training showed greater increases in their sense of control and perceived ability to improve memory, but not memory performance, compared to participants who received memory training only (Lachman et al., 1992; Rapp et al., 2002). Brief interventions with trauma survivors and experimental paradigms targeting self-efficacy appraisals more broadly have shown success in reducing distress and improving coping (Brown et al., 2016; Krans et al., 2018; Morina et al., 2018; Rahman et al., 2019).

Our results suggest that addressing maladaptive appraisals and expectations about one’s cognitive abilities, and one’s self-efficacy to manage or improve them, could be beneficial for patients with PTSD and mTBI. In addition to classic CBT techniques for challenging appraisals, brief computer-based “reappraisal training,” which has been successful in reducing intrusive thoughts (Woud et al., 2012), may be most effective when appraisals have low “truth value” (Gross, 2002). In patients with PTSD or mTBI, an example of a low “truth value” appraisal is the perception of cognitive problems when objective performance is unimpaired. In neuropsychological assessment, when a client’s appraisals do not align with objective performance, neuropsychologists should present the evidence of non-impaired performance to clients and provide psychoeducation about this common appraisal process seen in PTSD and mTBI.

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CRediT authorship contribution statement

Kristin W. Samuelson: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Supervision, Writing - original draft, Writing - review & editing. Krista Engle: Data curation, Formal analysis, Project administration, Writing - review & editing. Alisa Bartel: Data curation, Project administration. Joshua T. Jordan: Formal analysis, Writing - original draft, Writing - review & editing. Tyler Powers: Data curation, Project administration, Formal analysis, Writing - review & editing. Linda Abadjian: Project administration. Charles C. Benight: Conceptualization, Funding acquisition, Writing - review & editing.

Declaration of Competing Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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