Somatic burden and perceived cognitive problems in trauma-exposed adults with posttraumatic stress symptoms or pain

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Abstract

Objective: Posttraumatic stress disorder (PTSD) is associated with somatic and cognitive changes, which may be magnified when accompanied by persistent pain. The mechanisms of somatic sensation processing may extend to cognitive symptoms, revealing a potential generalization of impairment across cognitive and somatic domains in PTSD. We hypothesized that somatic burden would mediate relationships between PTSD, pain, and perceived cognitive impairment.

Methods: Two samples—360 trauma-exposed college students and 268 mechanical Turk users—completed self-report measures.

Results: Both samples revealed similar findings. There was a significant indirect effect of PTSD and pain on perceived cognitive problems through somatic burden. There remained a direct effect of PTSD symptoms. These findings indicate that in trauma-exposed samples with pain, somatic burden rather than pain severity accounts for perceived cognitive problems.

Conclusion: High somatic burden may reflect an underlying appraisal about somatic cues, which extend in part to interpretation of cognitive cues.

KEYWORDS
appraisals, cognitive functioning, pain, PTSD, somatic
INTRODUCTION

The body is comprised of organs that are susceptible to injury or decay over time. One such organ is the brain. It is generally accepted that the body and brain will deteriorate with age such that pain will escalate while cognition declines. However, at any age, a traumatic event may generate similar—yet unexpected—somatic and cognitive changes. As perceived physical changes in heart rate, breathing, headache, and stomach pain provoke concern about the body, perceived cognitive changes in memory, concentration, comprehension, and attention provoke concern about the brain.

These concerns may be especially relevant in Posttraumatic Stress Disorder (PTSD) and pain, conditions characterized by changes in somatic and cognitive symptoms. The mechanisms underlying these perceptions of somatic and cognitive impairment, and whether they are linked, remain unclear. In somatic conditions, bodily symptom appraisals may subsume cognitive symptoms and contribute to overall symptom burden. This study investigated the extent to which somatic burden mediated the relationships between PTSD, pain, and perceived cognitive impairment.

POSTTRAUMATIC STRESS DISORDER

When a stressor is encountered, the sympathetic nervous system is activated, which triggers somatic changes in muscle tension, heart rate, breathing, and sweat gland activity (Hoehn-Saric & McLeod, 2000). In response to an acute stressor, the stress response is expected to occur but eventually subside (Clinchy, Sheriff, & Zanette, 2013). The chronic activation and sensitization of this system, particularly in response to memories of acute threats or benign stimuli, may lead to pathological hyperarousal and contribute to the development of PTSD (Horowitz, 2011; Yehuda, McFarlane, & Shalev, 1998). The diagnostic criteria for PTSD include one to two symptoms from each of the four symptom clusters: re-experiencing (e.g., intrusive memories), avoidance (e.g., of reminders), negative alterations of cognitions or mood (e.g., negative beliefs, detachment), and hyperarousal (e.g., heightened startle response).

Emerging evidence links PTSD symptoms to high "disease burden" due to the association with lasting changes in autonomic functioning, neurocircuitry, major organ systems, sleep, and early mortality (McLeay et al., 2017; O’Donovan et al., 2015; Yehuda et al., 2015). Disruptions in these systems may cause patients to experience general and unfamiliar somatic complaints (Gupta, 2013; McFarlane, 2017; Yehuda et al., 2015). Compared to other psychiatric conditions, individuals with PTSD were at least twice as likely to report, and be at risk for developing, concurrent somatic symptoms (Andreski, Chilcoat, & Breslau, 1998).

Somatic cues, however, are not the only persistent symptom that trauma survivors may experience. Cognitive symptoms also accompany PTSD. Multiple meta-analyses have documented mild cognitive deficits in PTSD, most notably in domains of memory, processing speed, set shifting, visuospatial functioning, inhibition, psychomotor speed, attention, and information encoding (Brewin, Kleiner, Vasterling, & Field, 2007; Johnsen & Asbjørnsen, 2008; Polak, Witteveen, Reitsma, & Olff, 2012; Scott et al., 2015). Thus, trauma survivors likely experience comorbid and ongoing somatic and cognitive changes, but it is unclear whether they are linked.

2.1 | Pain

Frequently accompanying PTSD is a particularly noxious somatic symptom—pain. Pain is experienced and defined by subjective sensory perceptions and interpretations (Moriarty, McGuire, & Finn, 2011). Acute pain represents nociceptive input related to real or potential tissue damage, such as one may experience when placing their hand on a hot stove or after surgery (Dubin & Patapoutian, 2010). Acute pain may persist beyond the expected healing time
of 3 to 6 months becoming chronic pain. Chronic pain has been shown in some cases to emerge without any organic cause, suggesting biological as well as psychological influences (Elman, Zubieta, & Borsook, 2011; Merskey, 1986; Moriarty et al., 2011). Pain sensations are often perceived as more threatening because of its association with damage to the body (Merskey, 1986). The fear and anxiety that accompany the experience of pain often worsen its severity (Crombez, Eccleston, Baeyens, & Eelen, 1998; Ploghaus et al., 2001; Unruh & Ritchie, 1998), suggesting appraisals play a strong role.

Concurrent pain that occurs alongside PTSD may originate from an acute injury sustained during a trauma (Geisser, Roth, Bachman, & Eckert, 1996; Otis, Keane, & Kerns, 2003; Sharp & Harvey, 2001). Individuals with PTSD are over two times more likely to report physical complaints of general comorbid pain (McFarlane, Atchison, & Papay, 1994). A large epidemiological study found individuals with chronic pain conditions were 3.69 times more likely to suffer comorbid PTSD compared to those without chronic pain (Kessler et al., 2005; McWilliams, Cox, & Enns, 2003). PTSD and pain processing share similar neural networks (Pitman, Shin, & Rauch, 2001) and are associated with similar neurobiological abnormalities over time (Rauch, Shin, & Phelps, 2006; Scioli-Salter et al., 2015). Dysregulations in stress responses (McLean, Clauw, Abelson, & Liberzon, 2005), hyperarousal (Baliki, Geha, Apkarian, & Chialvo, 2008; Daniels et al., 2010), and pain modulation (Kosek, Ekholm, & Hansson, 1996) have also been observed in both PTSD and chronic pain.

Similar to PTSD, individuals reporting pain, whether acute or chronic, may also experience mild cognitive impairments. Chronic pain is associated with changes in memory, processing speed, psychomotor speed, and attention (Hart, Martelli, & Zasler, 2000). Acute pain, such as laboratory-induced pain, has also been shown to negatively impact neuropsychological test performance (Pitman et al., 2001). However, contrary to PTSD, the effects are often related to the accompanying psychological distress (e.g., depression), somatic vigilance, current mood, fatigue, and sleep status rather than symptom severity itself (Hart et al., 2000). These findings indicate that for individuals with persistent pain, general somatic symptoms and appraisals may drive outcomes more than pain severity.

2.2 Appraisals of cognitive performance

As both pain and PTSD are linked to cognitive changes, it is not surprising that they are accompanied by cognitive complaints. Although the cognitive differences tend to be subtle with minimal impact on cognitive performance, those experiencing the changes likely notice the differences, especially following a discrete traumatic event. This awareness of cognitive changes, which may or may not be causally attributed to the traumatic event and/or injury, may prompt clients to report worse functioning than is observed on testing. This discrepancy in self-report and objective functioning has been observed in samples with pain (Maor, Olmer, & Mozes, 2001; Middleton, Denney, Lynch, & Parmenter, 2006; Schmand et al., 1998; Schnurr & MacDonald, 1995) and PTSD (Binder, Storzbach, Anger, Campbell, & Rohlman, 1999; Samuelson, Abadjian et al., 2017; Mattson, Nelson, Sponheim, & Disner, 2019). Moreover, subjective impairment, but not objective performance, predicted functional outcomes (Hart et al., 2000; Samuelson, Bartel, Valadez, & Jordan, 2017; Samuelson, Abadjian et al., 2017; Spencer, Drag, Walker, & Bieliauskas, 2010). A recent study showed that the link between objective and subjective cognition was mediated by PTSD severity, further indicating that underlying this discrepancy is distress and appraisals (Mattson et al., 2019). This pattern extends to other populations with mild cognitive impairment, in which patients with depression (Farrin, Hull, Unwin, Wykes, & David, 2003; Williams, Little, Scates, & Blockman, 1987), negative mood (Larrabee & Levin, 1986), and mild head injuries (Gass & Apple, 1997; Spencer et al., 2010) report worse cognitive impairment than testing shows.

There are several potential explanations underlying this incongruity. It may be partially due to the poor sensitivity of neuropsychological tests to catch subtle impairments in higher functioning patients, or it may reflect these individuals’ ability to perform well in optimal, nontrigged conditions. An additional explanation involves applying somatic symptom appraisal theories to cognitive symptoms.
2.3 | Somatic symptom perception

Both biological and psychological processes underlie the perceptions and experience of somatic signals, including pain. The gate control theory of pain proposes that sensory pain signals travel through "gates" to the brain that can open and close depending on situational and psychological factors (Melzack & Wall, 1965). Eriksen and Ursin (2004) further describe that many sensory experiences do not make it to our conscious awareness, including physiological arousal during a stressful event as attention to these signals is mitigated by distraction (e.g., survival). Once the event concludes, persisting symptoms come to command full attentional resources.

Both PTSD and pain are characterized by the awareness of such somatic symptoms enduring beyond the acute stage. The ongoing experience of these symptoms, particularly outside the context of an actual life- or injury-threatening event, may induce misgivings or even fear of one's somatic symptoms (Tsao et al., 2009; Tsur, Defrin, Lahav, & Solomon, 2018; Van der Kolk, 2014; Vervoort, Goubert, Eccleston, Bijttebier, & Crombez, 2005; Vlaeyen & Linton, 2000). The model of somatosensory amplification, developed initially for hypochondriasis (Barsky, 1992; Barsky & Wyshak, 1990; Barsky, Goodson, Lane, & Cleary, 1988), posits that, particularly under stress, individuals may experience ambiguous somatic symptoms more intensely, which commands their attention and ultimately reinforces misperceptions. Three factors aside from potential secondary gain perpetuate symptom amplification: persistent belief that the symptoms represent disease, will not improve, and are disabling (Barsky & Borus, 1999).

This somatic hypochondriasis has been extended to "neurocognitive hypochondriasis" (Boone, 2009). This construct is distinct from malingering or conversion disorder because patients truly believe they are experiencing cognitive decline, despite normal test findings, and their symptoms are not fabricated (Boone, 2009). Boone further described potentially underlying tendencies common in patients: overestimating prior abilities while underestimating current ones, and establishing casual links between a discrete event and any noticeable changes. Following a stressful event, both cognitive and somatic changes may be linked to the trauma and mistakenly interpreted or overamplified (Boone, 2009). In fact, similar to somatic symptoms, normal and benign cognitive lapses have been previously misattributed to injury (Ferguson, Mittenberg, Barone, & Schneider, 1999; Mittenberg, DiGiulio, Perrin, & Bass, 1992). It may be that, similar to somatic sensations, benign cognitive lapses do not make it to our conscious awareness or cause alarm until they become associated with fearful stimuli.

2.4 | Somatic burden

All of these somatic sensations and their accompanying appraisals may contribute to an overall sense of burden. Somatic burden is the cumulative experience of and distress related to somatic symptoms. Burden assesses the degree to which patients report being "bothered by" these somatic symptoms, including pain, in the last 2 weeks (Gierk et al., 2014). Higher somatic burden is significantly related to depression, anxiety, and health status and uniquely predicts healthcare use, hospitalization, and death (Barsky, Orav, & Bates, 2005; Gierk et al., 2014). Although the presence of somatic symptoms does not imply pathological somatization, in severe cases, higher somatic burden may precede the development of Somatic Symptom Disorder (SSD). The diagnosis of SSD requires "multiple, current, somatic symptoms that are distressing or result in significant disruption of daily life" and "very high levels of worry." The distinction between SSD and somatic burden lies in the pathological interpretation and presentation of symptoms compared to the presence of and disturbance by symptoms. General somatic symptoms, therefore, are "expressions of personal suffering" (American Psychiatric Association, 2013) that do not necessarily convey pathology but provide important clinical data. Not directly included, but a possible extension of somatic burden, are cognitive symptoms.
2.5 | The present study

We theorize that, following a trauma, appraisals about bodily dysfunction extend to the brain. In turn, somatic burden may serve as the underlying mechanism by which PTSD and pain following trauma relate to perceptions of cognitive problems. We hypothesized that somatic burden would exert an indirect effect on the relationships between pain severity, PTSD symptom severity, and perception of cognitive problems in a sample of trauma-exposed adults.

Specifically, as PTSD has been extensively connected to mild cognitive differences, we hypothesized that the relationship between PTSD severity and perceived cognitive problems would only be partially mediated by somatic burden. Conversely, we hypothesized that the relationship between pain severity and perceived cognitive problems would be fully mediated by somatic burden, anticipating that more generalized somatic symptoms fully explain any relationship between pain and appraisals of poorer cognitive functioning. Pain is often studied in clinical populations and in isolation of nonpain somatic symptoms. This seclusion distinguishes pain from other somatic symptoms, which limits our understanding of how both pain and non-pain somatic sensations are coexperienced (Sullivan & Katon, 1993). Thus, in the current study we do not specifically examine a homogeneous or clinical population of individuals with pain, but rather a sample of trauma survivors who are at higher risk for experiencing pain.

3 | METHODS

3.1 | Participants

Two samples of trauma-exposed adults were collected. A sample of 360 undergraduate students enrolled in psychology courses were recruited from a mid-sized public university in the Western United States and were offered extra credit for completing the study. Participants were at least 18 years of age, living in the United States, fluent in English, and currently enrolled at least part-time. The mean age of participants was 23.05 (standard deviation [SD] = 6.93; range = 18–54; median = 20.50). The majority of participants identified as White/Caucasian (80.95%), not Hispanic/Latino (73.13%), were female (77.16%), and had no military affiliation (83.61%).

The second sample of 268 participants was recruited online from Amazon’s Mechanical Turk (mTurk). Participants were at least 18 years of age and restricted to those living in the United States. Research has demonstrated that mTurk users match the general U.S. population more closely than undergraduate samples and Internet board samples (Buhrmester, Kwang, & Gosling, 2011; Paolacci, Chandler, & Ipeirotis, 2010). The mean age of participants was 34.40 (SD = 9.55; range = 20–64; median = 32.0). The majority of participants identified as White/Caucasian (79.85%), not Hispanic/Latino (86.89%), were male (53.56%), and had no military affiliation (94.36%). The median years of education for the mTurk sample was <16 years.

We utilized both an undergraduate and community sample to achieve more representation in a cost- and time-efficient manner. Both samples responded to an advertisement seeking participants who had experienced a traumatic event. To participate, the participants had to endorse this screening question to meet the Diagnostic and Statistical Manual of Mental Disorders-5th edition (DSM-5; American Psychiatric Association, 2013) criteria for a traumatic event: “Have you ever experienced a stressful or traumatic life event where you believed your life was in danger, or the life of a loved one was in danger, or you were in danger of being seriously injured? For example, a serious accident, disaster, physical or sexual assault, combat, domestic violence, or childhood physical or sexual abuse.”

Before completion of the PTSD-Checklist for DSM-5 (PCL-5; Weathers et al., 2013), participants were asked to describe their worst traumatic event in their lifetime and respond to the PCL-5 with that event in mind. These event descriptions were reviewed by the authors to verify that they met the Criterion A definition of the DSM-5 diagnostic criteria and were subsequently included in analyses. We classified trauma type in to five categories:
accidents and/or disasters (38.31% for undergraduate sample, 57.68% for mTurk), adult interpersonal violence or assault (22.25% and 17.23%), child maltreatment (19.44% and 14.61%), combat trauma (3.94% and 1.87%), and other, which included sudden death of a loved one and life-threatening illness (16.06% and 8.61%).

Participants were also asked if they “experience chronic pain, as defined by continuous or recurrent pain lasting at least 3 months or longer,” which was endorsed by 60.60% of participants from the undergraduate sample and 39.40% from the mTurk participants. Those endorsing current pain were asked to indicate the areas in which they felt the most pain based on a modified version of item two from the Brief Pain Inventory. This checklist included the following areas: face, head, neck, shoulders, arms and/or hands, chest, stomach, legs and/or knees, groin, and ankles and/or feet. The most common complaints involved pain in the head (24.00% for undergraduate sample and 17.90% for mTurk), legs and/or knees (9.60% and 14.50%), shoulders (6.40% and 9.70%), and neck (8.00% and 6.80%). In both samples, the remaining participants reported pain in all other areas (e.g., chest). Of those with current pain, 63.60% of the undergraduate participants reported pain in multiple areas, compared to 39.40% in the mTurk sample.

3.2 | Measures

Participants completed a 10-item demographic questionnaire inquiring about age, race, ethnicity, military service, and education.

The PTSD Checklist for DSM-5 (PCL-5; Weathers et al., 2013) is a 20-item self-report questionnaire that measures severity of PTSD symptoms based on DSM-5 criteria. Each item corresponds to a symptom and is ranked 0–4 with the following descriptors: Not at all (0), A little bit (1), Moderately (2), Quite a bit (3), and Extremely (4). Symptom scores are summed and the total ranges from 0 to 80, with higher scores indicating more severe symptom expression. The PCL-5 has shown good test-retest reliability, internal consistency, and convergent validity in trauma-exposed samples (Blevins, Weathers, Davis, Witte, & Domino, 2015; Bovine et al., 2016; Weathers, Litz, Herman, Huska, & Keane, 1993). Cronbach’s α was high for both samples, at 0.96 for the undergraduate sample and 0.95 for the mTurk sample.

The Brief Pain Inventory-Short Form (BPI-SF; Cleeland & Ryan, 1991) is an abbreviated version of the Brief Pain Inventory (BPI; Cleeland & Ryan, 1991; Cleeland & Ryan, 1994; Cleeland, 1989, 1990), which allows for more clinically useful descriptors of pain, and is the recommended version for outcome studies due to its brevity and ease of use. The BPI-SF measures both severity of pain (intensity) and its impact on daily functioning (interference) in a 24-hr recall period. Questions relating to the severity of pain measure the intensity of pain experienced, for example, “Please rate your pain by marking the box beside the number that tells how much pain you have right now.” Each severity of pain item is ranked from No Pain (0) to Pain As Bad As You Can Imagine (10). Items regarding the impact of pain on daily functioning measure the interference pain has on aspects daily life, such as general activity, mood, or sleep. Each item is ranked from Does Not Interfere (0) to Completely Interferes (10). The BPI has shown good psychometrics assessing various pain types (Cleeland & Ryan, 1994). The questions regarding ratings of pain severity at various time points (worst, least, average, and right now) were combined into a composite index of pain severity, as recommended by the creators (Cleeland & Ryan, 1994). Internal consistency was α = 0.90 for the undergraduate sample and α = 0.95 for the mTurk sample.

The Somatic Symptom Scale (SSS-8) is a condensed 8-item questionnaire derived from the highly validated 15-item Patient Health Questionnaire (PHQ-15; Kroenke, Spitzer, & Williams, 2002). Initially developed for the DSM-5 field trials (Narrow et al., 2013) assessing the new SSD, the SSS-8 measures somatic symptom burden. Scores range from 0 to 32 with the following severity categories: no to minimal (0–3 points), low (4–7 points), medium (8–11 points), high (12–15 points), and very high (16–32 points). Each category escalation signified a 53% (95% CI, 44–63%) increase in healthcare-related appointments (Gierk et al., 2014). The SSS-8 factor structure reveals symptom clusters for gastrointestinal, pain, fatigue, and cardiopulmonary somatic expression and has shown good reliability and construct validity (Gierk et al., 2014). It is highly correlated with a modified version of the PHQ-15 and demonstrates at least comparable psychometric properties (Toussaint, Kroenke, Baye, & Lourens, 2017).
Moreover, its sensitivity to change was recently established in relation to depression, anxiety, and disability (Gierk, Kohlmann, Hagemann-Goebel, Löwe, & Nestoriuc, 2017). In this study, somatic symptom burden was measured as a continuous variable. Cronbach’s α for the undergraduate sample was 0.80, and α for the mTurk sample was 0.85.

The Cognitive Self-Report Questionnaire (CSRQ; Spina, Ruff, & Mahncke, 2006), cognitive subscale is a 10-item self-report measure that assesses perception of cognitive problems, such as memory and attention difficulties, within the last 2 weeks. The CSRQ has demonstrated concurrent validity (Spina et al., 2006) through significant correlations with the Cognitive Failures Questionnaire (Broadbent, Cooper, FitzGerald, & Parkes, 1982). Cronbach’s α for the undergraduate sample was 0.82, and α for the mTurk sample was 0.77.

3.3 | Data analysis

The two samples were first compared on independent, dependent, and proposed mediating variables through Mann-Whitney U tests, with the Success Rate Difference (SRD) as an estimate of effect size. The SRD is a transformation of the Area Under the Curve (defined as 2*AUC-1) and can be interpreted as a correlation coefficient (range is 0–1). The analyses were conducted and analyzed separately by sample.

To test the hypothesis that the relationship between pain and PTSD (independent variables) and perception of cognitive problems (dependent variable) is mediated by somatic burden, the significance of the indirect effect was assessed through the product of coefficients method. Bias-corrected bootstrapped confidence intervals were used with 1,000 replications to evaluate significance of the indirect effect. Bootstrapping (Efron & Tibshirani, 1994) is a resampling procedure in which a parameter of interest (in this case, the indirect effect) is sampled with replacement from the original data set, and recalculated a specified number of times (in this instance, 1,000) to provide a more robust estimate of the parameter’s distribution. Bootstrapped indirect effects were considered significant if the 95% confidence intervals (CIs) did not contain zero. This approach is considered superior to other tests of indirect effects, as the product of coefficients typically does not follow a normal distribution (MacKinnon, Lockwood, & Williams, 2004). All analyses were conducted using Stata Version 15.1 (StataCorp, 2017). Kappa squared ($\kappa^2$) was used as a measure of effect size for indirect effects, with values of 0.01, 0.09, and 0.25 indicating small, moderate, and large effect sizes, respectively (Preacher & Kelley, 2011).

4 | RESULTS

Means and standard deviations are reported in Table 1. The undergraduate sample reported greater PTSD symptom severity on the PCL ($|z| = 18.21; p < .001; \text{SRD} = 0.86$), somatic burden on the SSS-8 ($|z| = 4.46; p < .001; \text{SRD} = 0.21$), and perceived cognitive problems ($|z| = 5.22; p < .001; \text{SRD} = 0.25$). There were no differences on the BPI ($|z| = 0.38; p = .707$).

The undergraduate sample had greater odds of missing data than the mTurk sample (20.5% vs. 8.6%, odds ratio [OR] = 2.74; $x^2[1] = 16.75; p < .001$). Data were determined to be missing at random for each of the two samples as no patterns of missingness were evident, nor were there any correlates of missingness that could be identified. Because of this, full information maximum likelihood was implemented (FIML) to retain all cases. FIML is considered a gold standard approach to handling data that is missing at random (Enders, 2010).

Table 1 shows Spearman correlations and descriptive statistics for study variables. There were large associations between each of the predictors; however, tolerance statistics ($>0.44$) and variance inflation factors ($<1.96$ for both samples) suggested that multicollinearity was not a problem.

Mediation models were run separately for each sample. The overall proportion of variance explained by the model for the undergraduate sample was 49.6%. PTSD symptoms and pain severity accounted for 46.9% of the variance in somatic burden and 22.2% of the variance in perceived cognitive problems. There were significant total effects of PTSD symptoms and pain severity on perceived cognitive problems ($b = 0.12; z = 6.25; p < .001$, and
The mediation model revealed significant moderate indirect effects of PTSD ($\alpha\beta = 0.04; 95\% \text{ BCI} = [0.02, 0.06]; \chi^2 = 0.105$) and pain ($\alpha\beta = 0.56; 95\% \text{ BCI} = [0.31, 0.89]; \chi^2 = 0.116$) on perception of cognitive problems through somatic burden (see Figure 1). There remained a direct effect of PTSD symptoms ($c' = 0.09; |z| = 4.14; p < .001$) but there was no direct effect for pain severity ($c' = 0.03; |z| = 0.13; p = .894$).

A similar pattern of results was seen for the mTurk sample. The overall proportion of variance explained by the model for the mTurk sample was 54.3%. PTSD symptoms and pain accounted for 51.5% of the variance in somatic burden and 25.5% of the variance in perceived cognitive problems. There was a significant total effect of PTSD symptoms ($b = 0.24; z = 6.46; p < .001$) and a near significant total effect of pain severity ($b = 0.46; z = 1.77; p = .077$). The mediation model revealed significant moderate indirect effects of PTSD ($\alpha\beta = 0.08; 95\% \text{ BCI} = [0.04, 0.13]; \chi^2 = 0.115$) and pain ($\alpha\beta = 0.69; 95\% \text{ BCI} = [0.40, 1.09]; \chi^2 = 0.131$) on perception of cognitive problems through somatic burden. A direct effect remained for PTSD symptoms ($c' = 0.16; |z| = 4.02; p < .001$) but there was no direct effect for pain severity ($c' = -0.23; |z| = 0.76; p = .446$). Total results can be found in Table 2.

### Table 1: Descriptive statistics and intercorrelations of primary study variables in both samples

<table>
<thead>
<tr>
<th>Variable</th>
<th>Undergraduate sample (N = 360)</th>
<th>mTurk sample (N = 268)</th>
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</thead>
<tbody>
<tr>
<td>1. PCL-5 total score</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>2. BPI severity</td>
<td>0.43***</td>
<td>0.41***</td>
</tr>
<tr>
<td>3. SSS-8</td>
<td>0.55*** 0.64*** 1</td>
<td>0.60*** 0.63*** 1</td>
</tr>
<tr>
<td>4. CSRQ-cognitive</td>
<td>0.42*** 0.30*** 0.43*** 1</td>
<td>0.51*** 0.31*** 0.52*** 1</td>
</tr>
</tbody>
</table>

Abbreviations: BPI, brief pain inventory; PCL-5, posttraumatic stress disorder checklist for DSM-5; SD, standard deviation; SSS, somatic symptoms scale.

***p < .001.

$B = 0.60; z = 2.71; p = .007$, respectively). The mediation model revealed significant moderate indirect effects of PTSD ($\alpha\beta = 0.04; 95\% \text{ BCI} = [0.02, 0.06]; \chi^2 = 0.105$) and pain ($\alpha\beta = 0.56; 95\% \text{ BCI} = [0.31, 0.89]; \chi^2 = 0.116$) on perception of cognitive problems through somatic burden (see Figure 1). There remained a direct effect of PTSD symptoms ($c' = 0.09; |z| = 4.14; p < .001$) but there was no direct effect for pain severity ($c' = 0.03; |z| = 0.13; p = .894$).
In two samples (undergraduate college students and community mTurk users) of trauma survivors, we found that somatic burden mediated the relationships between PTSD symptom severity, pain severity, and perception of cognitive problems. Somatic burden partially accounted for the effect of PTSD symptoms on perception of cognitive problems, as there remained a direct effect of PTSD symptom severity. In contrast, there was not a direct effect of pain severity on perception of cognitive problems when accounting for the indirect effect of somatic burden. This finding indicates that somatic burden, rather than pain severity itself, influences perceptions of cognitive problems in trauma-exposed pain sufferers.

The pattern of results was similar in two separate samples, demonstrating that these findings are generalizable across two frequently used samples in trauma research: undergraduates and mTurk users. Notably, however, the undergraduate sample reported surprisingly high rates of current pain and significantly higher levels of symptoms related to PTSD, somatic burden, and perceived cognitive impairment. This pattern lends evidence to the (albeit controversial) claims that student samples are not representative of the general population (Hanel & Vione, 2016; Peterson, 2001). These findings might also suggest that trauma-exposed psychology majors have an over-reporting style. By examining these constructs in two samples with differing levels of symptom severity, we established that somatic burden mediates relationships between PTSD and pain symptom severity and perceptions of cognitive problems, reflecting the strength of appraisals over symptom intensity.

Complaints of cognitive problems have been frequently described in both pain sufferers and trauma-exposed individuals, yet the mechanisms driving these negative and discrepant self-report responses were unclear. Previous studies have found that objective cognitive performance is not always related to subjective cognitive performance (Binder et al., 1999; Maor et al., 2001; Middleton et al., 2006; Samuelson, Abadjian et al., 2017; Schmand et al., 1998; Schnurr & MacDonald, 1995; Mattson et al., 2019), and that subjective, rather than objective, cognitive performance predicts psychosocial functioning in patients with PTSD (Samuelson, Abadjian et al., 2017). In our samples, there remained a large direct effect of PTSD on perceived cognitive functioning, suggesting that appraisals may be partially driven by an accurate, likely objective change in cognitive functioning, as well as by accompanying somatic burden. Future studies could examine the role of objective neurocognitive performance in these relationships.

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>Results of mediation models</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parameter</td>
<td>Undergraduate sample (n = 360)</td>
</tr>
<tr>
<td></td>
<td>β</td>
</tr>
<tr>
<td>Somatic burden</td>
<td></td>
</tr>
<tr>
<td>PTSD symptoms</td>
<td>0.35</td>
</tr>
<tr>
<td>Pain severity</td>
<td>0.47</td>
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<tr>
<td>Perceived cognitive problems</td>
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<tr>
<td>Somatic burden</td>
<td>0.29</td>
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<tr>
<td>PTSD symptoms</td>
<td>0.24</td>
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<tr>
<td>Pain severity</td>
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<tr>
<td>Indirect effects</td>
<td></td>
</tr>
<tr>
<td>Parameter</td>
<td>αβ</td>
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<tr>
<td>PTSD symptoms</td>
<td>0.04</td>
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<tr>
<td>Pain severity</td>
<td>0.56</td>
</tr>
</tbody>
</table>

Abbreviations: BCI, bias-corrected bootstrap confidence intervals; PTSD, posttraumatic stress disorder.
*p < .05.

5 | DISCUSSION

In two samples (undergraduate college students and community mTurk users) of trauma survivors, we found that somatic burden mediated the relationships between PTSD symptom severity, pain severity, and perception of cognitive problems. Somatic burden partially accounted for the effect of PTSD symptoms on perception of cognitive problems, as there remained a direct effect of PTSD symptom severity. In contrast, there was not a direct effect of pain severity on perception of cognitive problems when accounting for the indirect effect of somatic burden. This finding indicates that somatic burden, rather than pain severity itself, influences perceptions of cognitive problems in trauma-exposed pain sufferers.

The pattern of results was similar in two separate samples, demonstrating that these findings are generalizable across two frequently used samples in trauma research: undergraduates and mTurk users. Notably, however, the undergraduate sample reported surprisingly high rates of current pain and significantly higher levels of symptoms related to PTSD, somatic burden, and perceived cognitive impairment. This pattern lends evidence to the (albeit controversial) claims that student samples are not representative of the general population (Hanel & Vione, 2016; Peterson, 2001). These findings might also suggest that trauma-exposed psychology majors have an over-reporting style. By examining these constructs in two samples with differing levels of symptom severity, we established that somatic burden mediates relationships between PTSD and pain symptom severity and perceptions of cognitive problems, reflecting the strength of appraisals over symptom intensity.

Complaints of cognitive problems have been frequently described in both pain sufferers and trauma-exposed individuals, yet the mechanisms driving these negative and discrepant self-report responses were unclear. Previous studies have found that objective cognitive performance is not always related to subjective cognitive performance (Binder et al., 1999; Maor et al., 2001; Middleton et al., 2006; Samuelson, Abadjian et al., 2017; Schmand et al., 1998; Schnurr & MacDonald, 1995; Mattson et al., 2019), and that subjective, rather than objective, cognitive performance predicts psychosocial functioning in patients with PTSD (Samuelson, Abadjian et al., 2017). In our samples, there remained a large direct effect of PTSD on perceived cognitive functioning, suggesting that appraisals may be partially driven by an accurate, likely objective change in cognitive functioning, as well as by accompanying somatic burden. Future studies could examine the role of objective neurocognitive performance in these relationships.
Pain severity, however, was not directly related to the perception of cognitive impairments when accounting for the effect of somatic burden. Pain is recognized as having social, psychological, and biological influences (Turk & Okifuji, 1999) and is highly correlated with somatoform disorders (Birket-Smith, 2001; Dersh, Polatin, & Gatchel, 2002). In their extensive review, Hart et al. (2000) found that high somatic vigilance, but not high pain intensity, exerted an effect on patients’ cognitive performance. Thus, individuals exposed to arousing events or stimuli may be partially preoccupied with somatic/arousal symptoms that command limited cognitive resources. Pain severity measures the physical experience of pain but does not necessarily account for pain-related appraisals, such as catastrophizing or anxiety sensitivity. Somatic symptom burden, however, accounts for multiple symptoms that do not necessarily stem from a single syndrome, illness, injury, or disorder. Thus, it is possible that the assessment of somatic burden may capture underlying appraisals toward bodily cues.

Since even mild bodily cues may be interpreted as evidence of more widespread impairment, then benign cognitive lapses may similarly indicate damage to the brain (Boone, 2009). Perceived changes in attention, memory, and other problems may be interpreted as fearful or catastrophic cues of cognitive impairment, signaling that the mind—like the body—cannot be trusted. As a result, rigid beliefs about dysfunction persist. Related appraisals such as anxiety sensitivity and catastrophizing may underlie this finding, and future research should investigate these proposed mechanisms. Anxiety sensitivity is related to both perceptions of somatic burden and cognitive functioning. Individuals high in anxiety sensitivity may report fear of being unable to keep their minds on a task, for instance, or that something is wrong if their mind goes blank (Reiss, Peterson, Gursky, & McNally, 1986). Research on cognitive concerns related to anxiety sensitivity has been mixed, but generally the symptoms appear to encompass fear of developing a mental disorder (Petrocchi, Tenore, Couyoumdjian, & Gragnani, 2014; Reiss et al., 1986). When individuals experience high somatic burden they are unable to understand or explain, they may view it as a sign they are going crazy or losing their minds. Individuals with PTSD or pain, which are associated with cognitive changes, may be oriented to this altered cognitive capacity, particularly in the acute stages.

Several limitations of this study should be considered. Most importantly, the data are cross-sectional and as such, causal relationships and therefore mediation cannot be indisputably established (Kraemer, Kiernan, Essex, & Kupfer, 2008). Second, neuropsychological test data were not collected, which could help elucidate the nature of the link between perceptions of cognitive impairment and objective cognitive performance. Finally, this study was limited to a trauma-exposed sample with or without self-endorsed pain, so it is unclear whether these findings extend to samples with medically established chronic pain. Since these data have not been collected in clinical samples, the model may not hold in patients medically diagnosed with PTSD or chronic pain. No information was collected on whether participants were treatment-seeking, which could further influence results. Future research should attempt to replicate this model in other populations, especially clinical, and consider other variables (e.g., trauma type, depression, etc.). In addition, inclusion of other appraisals such as those related to anxiety sensitivity, pain- and health-related anxiety, and pain catastrophizing, may elucidate how perceived somatic and cognitive impairment influence functioning.

Patients with pain and PTSD are challenging to treat due to symptom overlap, comorbidity, and interactive effects (Sharp & Harvey, 2001). Our findings demonstrate that the perception of cognitive impairment in patients with PTSD and pain is driven by perceived somatic burden, which some researchers contend has often been neglected in research, treatment, and diagnostic considerations (McFarlane, 2017). As trauma-exposed patients with PTSD may incur somatic pathologies (e.g., metabolic syndrome, autoimmune diseases), somatic symptoms should be considered an integral component of trauma treatment (Carvajal, 2018; McLeay et al., 2017). Because PTSD is a “systemic disorder” (McFarlane, 2017) affecting critical biological functions, somatic symptoms are a direct result rather than secondary byproduct of trauma exposure. It is important to treat somatic symptoms, including pain, not as pathological conversion but legitimate sequelae of PTSD.

Importantly, this study does not suggest that patients with high somatic symptom burden are malingering or exaggerating cognitive symptoms, but rather that somatic distress plays an important role on their perceptions of cognitive functioning. We postulated that high somatic burden may reflect an underlying appraisal about somatic
cues, which extend in part to interpretation of cognitive cues. These findings suggest that treatment focus should not necessarily remain on rehabilitating somatic and cognitive changes themselves but targeting appraisals about the changes, particularly following a traumatic event. Moreover, the discrepancy between subjective and objective cognitive performance may actually reflect increased attunement to or preoccupation with troubling symptoms since the trauma. Given the strength of appraisals, the inclusion of cognitive behavioral therapy may prove to be an impactful element in the treatment of both pain and PTSD. In fact, in patients with somatizing tendencies, cognitive-behavioral treatments have found success in reducing self-report of symptoms and healthcare use, suggesting symptom-related appraisals may be a driving mechanism (Allen, Woolfolk, Escobar, Gara, & Hamer, 2006; Kroenke & Swindle, 2000; Namenek, 2007). This growing body of research suggests that the wide-ranging effects of trauma exposure and subsequent appraisals are evident across psychological and physical domains, and requires that clinicians implement more integrated and holistic approaches to treatment.

CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

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