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Posttraumatic Stress Symptoms Mediate the Effects of Trauma Exposure on Clinical Indicators of Central Sensitization in Patients with Chronic Pain

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Abstract

Objective: Evidence supports high rates of co-occurrence of posttraumatic stress disorder (PTSD) and chronic pain disorders involving central sensitization (CS). The nature of this relationship, however, remains relatively unexplored. In this study, we aimed to (1) assess how both trauma exposure and current PTSD symptoms are related to clinical manifestations of CS, and (2) test whether PTSD symptoms explain the relationship between trauma exposure and CS. Because experiential avoidance has been shown to impact the relationship between trauma and health outcomes, we (3) explored experiential avoidance as a possible mediator or moderator of the trauma-CS relationship.

Methods: A sample of 202 adult patients (79% female) with chronic pain completed validated self-report measures of trauma exposure, current PTSD symptoms, experiential avoidance, and three manifestations of CS: widespread pain, greater pain severity, and polysomatic symptom reporting. We used path analysis and multivariate regression to assess our study aims.

Results: Both trauma exposure and PTSD symptoms were significantly associated with all three clinical indicators of CS. PTSD symptoms partially explained the relationship between trauma exposure and widespread pain, pain intensity, and polysomatic symptoms. Experiential avoidance did not mediate or moderate the trauma-CS relationship.

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Conclusion: Our findings suggest that trauma exposure is linked to elevated clinical markers of CS, but a critical factor in this relationship is the mediating effect of current PTSD symptoms.

Keywords

central sensitization; experiential avoidance; fibromyalgia; PTSD; trauma; COPC

Introduction

Central sensitization (CS)—the amplification of neural signaling in the central nervous system contributing to hyperalgesia[1]—is a significant problem in chronic pain and affects up to 44 million Americans.[2] CS represents an underlying abnormality in pain processing involving both pain hypersensitivity and its underlying processes that contribute to a wide range of chronic and often overlapping pain conditions such as fibromyalgia, irritable bowel syndrome, and low back pain.[3] Rates of psychosocial trauma and lifetime adversity are substantially elevated in patients with pain disorders involving CS,[4,5] with posttraumatic stress disorder (PTSD) prevalence estimated at 20.5% in patients with chronic widespread pain,[6] and those with a trauma history being approximately three times more likely to develop pain conditions involving CS later in life than those without a trauma history.[4] Individuals with trauma histories tend to have worse pain and health outcomes, including more severe symptom presentation, increased disability and likelihood of unemployment, and higher healthcare utilization.[7,8] A more comprehensive understanding of the link between trauma and CS could improve treatment for many chronic pain patients.[2]

CS can be assessed under laboratory conditions using evoked pain protocols designed to tap into temporal summation.[9–11] Recent work suggests that CS as indexed by temporal summation is associated with potential clinical markers of CS, for example greater pain intensity[12,13] and extent of pain.[14,15] Indeed, diffuse or widespread pain, sensory amplification, and disproportionate pain intensity have been explored as three clinical markers to capture CS or centrally-maintained pain in clinical settings.[16–18] Based on perhaps the largest study to-date, Shrepf and colleagues[19] provided support for the multidimensional nature of CS, showing that CS is characterized by generalized sensory sensitivity or increased sensitivity to external stimuli, heightened somatic awareness and experience of widespread pain, and also disruptions in constitutional symptoms such as heightened pain severity, cognitive disturbance, and sleep difficulties. These multiple domains of CS are reflected in revisions to the definition of one of the common centrally-maintained pain disorders, fibromyalgia,[20,21] moving from a purely pain-focused phenotype toward a broader symptom-focused condition that includes pain and fatigue, unrefreshing sleep, and somatic and cognitive symptoms. Manifestations of CS beyond pain severity are rarely investigated in chronic pain samples. It is recommended to consider multiple dimensions of CS when characterizing CS-related pain, which falls on a spectrum of severity.[19,22]

Higher rates of trauma exposure have been noted in pain disorders involving CS,[4,8] yet not all patients exposed to trauma develop chronic pain or CS. This suggests that the relationship between trauma exposure and pain disorders might be better explained by a third variable,

such as PTSD symptoms, which reflects the psychological impact of the trauma. This idea is supported by research in PTSD samples evaluating response to experimental pain. Evidence suggests that both exposure to trauma and PTSD symptoms separately contribute to health outcomes.[23] In non-pain samples, preliminary evidence demonstrates that individuals with PTSD have increased sensitivity to painful stimuli[24] and are more likely to have acute CS as indexed by quantitative sensory testing and temporal pain summation following exposure to prolonged experimental pain.[25] Moreover, individuals who develop PTSD after exposure to torture, combat, and intimate partner violence show evidence of altered neurological response and disrupted pain regulation in response to painful stimuli.[26–28] Evidence suggests this phenomenon is driven by avoidance of trauma cues, which predisposes PTSD sufferers to pain chronification.[27,29] In the chronic pain population, one study with adolescents[30] found that individuals exposed to trauma had heightened CS as reflected in elevated temporal summation; however, that study did not evaluate PTSD symptoms or diagnoses. In the context of chronic pain populations and CS, the relationships between both trauma exposure and PTSD symptoms as they relate to CS symptomology remain relatively unexplored and require further study.[31]

Although PTSD and CS tend to co-occur, they are often not examined concurrently in the same patients, leaving the many ways in which they could potentially be associated unexplored. Multiple theoretical models exist to explain the relationship between trauma and pain.[32–34] For example, the mutual-maintenance model[35] emphasizes several pathways that reinforce and maintain both PTSD and pain, including shared mechanisms such as experiential avoidance.[36] Experiential avoidance is an individual's attempt to avoid or not experience aversive thoughts, feelings, and sensations, and it is implicated in a broad range of psychosocial problems.[37,38] It is comprised of multiple interrelated distress-management processes, such as attempting to ignore, suppress, or avoid distress, as well as holding negative attitudes toward, delaying approach of, distancing oneself from, or persisting through one's distress.[38] In chronic pain and trauma populations, the broad construct of experiential avoidance has seldom been investigated, but specific avoidance processes sometimes have. For example, actively suppressing negative emotions has been associated with elevated pain intensity in people with chronic back pain[39] and greater physiological arousal in people with PTSD.[40] Experiential avoidance has been identified as both a mediator[41] (coping strategy) and moderator[42,43] (dimension of personality) of the relationships among trauma exposure and health outcomes and is often associated with impaired recovery and poor health. Understanding how experiential avoidance may contribute to the trauma-CS relationship could provide guidance on addressing this topic therapeutically with highly complex patients.

To capture clinical markers of CS in the current study, we operationalized CS as having three manifestations: widespread pain, elevated pain severity, and polysomatic symptom reporting. With this study, we aimed to (1) evaluate the relations among cumulative exposure to trauma and three CS-related clinical markers and (2) assess whether PTSD symptoms explained the trauma-CS relationship. We specifically hypothesized that a history of trauma exposure would be associated with greater pain intensity, more widespread pain, and polysomatic complaints (our three operationalized clinical indicators of CS). Furthermore, we hypothesized that there would be an indirect relationship between trauma load and CS

indicators through PTSD symptoms. Because experiential avoidance may be either a mediator or moderator of the trauma-CS relationship, as an exploratory aim (3), we tested both possibilities and evaluated the level of support for each.

Method

Participants and Procedure

The university institutional review board approved all study procedures. We recruited participants with chronic pain to complete a series of validated questionnaires from January 2016 to March 2017. We identified potential participants primarily via large university-affiliated outpatient medical clinics, a hospital-wide research listserv, and online through ResearchMatch,[44] a national clinical research registry. All interested individuals were screened for eligibility via a structured questionnaire. Eligible participants were English-speaking adults (> age 18) reporting a chronic pain diagnosis, indexed by the patient or referring provider responding "yes" to the question "do you have a medical diagnosis involving chronic pain (for 6 months or longer")[45]. We confirmed the presence of chronic pain via electronic health record review. Exclusion criteria included a diagnosis of cognitive or thought disorder, current substance dependence, or significant emotional distress (e.g., active suicidal ideation) as assessed by referring medical providers, the principal investigator (PI, a licensed psychologist), or a research assistant supervised by the PI. Of the 211 participants initially recruited, 202 completed the study protocol and comprise the analyzed sample.

Following screening and consenting, participants completed a battery of validated questionnaires assessing trauma-related experiences, PTSD symptoms, experiential avoidance, and manifestations of CS (online at home, at an on-site computer station, or on-site via paper-and-pencil). We reimbursed participants with a $20 gift card in exchange for study participation.

Measures

Demographics.—Data on age, gender, race, relationship status, educational attainment, employment status, income, and disability status were obtained.

Trauma-Related Measures

Trauma History Questionnaire (THQ[46]): The THQ is a 24-item self-report measure that assesses experiences with potentially traumatic events such as crime, general disaster, and sexual and physical assault using a yes/no format. A total trauma load score is calculated by summing the overall number of endorsed traumatic experiences. The THQ has been determined to be reliable and valid in clinical and non-clinical samples[47].

PTSD Checklist—*DSM-5* Version (PCL-5[48]): The PCL-5 is a 20-item self-report measure corresponding to the *DSM-5*[49] criteria for PTSD. Participants indicate whether symptoms have bothered them in the past month, rating each item on a scale from 0 (not at all) to 4 (extremely). A total score of current PTSD symptoms can be calculated by summing all 20 ratings, and a provisional PTSD diagnosis can be given using DSM-5 PTSD

diagnostic criterion rules.[50] The PCL-5 has excellent internal consistency (α = .94[48], α = .95 in the present study) and good convergent/discriminant validity in past research.

Multidimensional Experiential Avoidance Questionnaire (MEAQ[38]): The MEAQ is a 62-item self-report questionnaire measuring experiential avoidance on six subscales that comprise different aspects of the broader concept of experiential avoidance: distress aversion, behavioral avoidance, distraction/suppression, repression/denial, procrastination, and distress endurance. Items are rated from 1 (strongly disagree) to 6 (strongly agree), and a total score is obtained by summing each of the subscales (distress endurance reverse scored). The measure has demonstrated discriminant and convergent validity[20] and good reliability and consistency,[38] and in the present study internal consistency was high for all subscales (α = .82 – .89) and the overall score (α = .93).

Indicators of Central Sensitization

To operationalize central sensitization, we used the measures below that tap into the well-described CS constructs of extent of bodily pain, pain intensity, and polysomatic complaints:

Michigan Body Map – Revised Version (MBM[51,52]): The MBM is a self-report measure used to assess the location(s) of chronic pain complaints and widespread body pain. It is a two-sided body image with check-box responses for 35 potential body areas where chronic pain (defined as pain experienced for longer than 3 months) might exist, and a box for “no pain.” In this study, the measure was used to indicate widespread pain related to CS by summing the number of pain areas endorsed. The MBM has acceptable test-retest reliability and face, convergent, and discriminant validity as an index of widespread pain.[30]

McGill Pain Questionnaire-Short Form-Revised (SF-MPQ-2[53]): The SF-MPQ-2 is a self-report measure assessing various dimensions of pain (continuous, intermittent, neuropathic, and affective pain). It consists of 22 pain descriptions that are rated from 0 (none) to 10 (worst possible). An overall pain score is computed by averaging all 22 ratings; higher scores indicate increased pain intensity.[54] The revised measure has demonstrated excellent reliability, validity, and excellent total score internal consistency (α = .91/.96),[53,55] corroborated by the present study (α = .91).

Central Sensitization Inventory (CSI[56]): The CSI is a self-report screening measure that assesses key polysomatic complaints often associated with a CS disorder. The CSI contains two sections: Part A (CSIA; used in the present study) assesses 25 health-related symptoms common in conditions involving CS, with responses rated on a scale from 0 (never) to 4 (always). The CSIA has demonstrated an internal consistency of Part A of .88,[56] corroborated by excellent internal consistency in the present study (α = .91), and has been effective at differentiating CS from non-CS groups[34]. The CSI is a reliable and valid measure assessing the severity of CS-related symptomology[57] and recommended for use in evaluating clinical indicators of CS such as generalized sensory sensitivity.[16]

Descriptive Variables for Clinical Interpretability

ACR Epidemiological Criteria for Fibromyalgia (FM-Dx): For clinical interpretative purposes, we used study variables to operationalize fibromyalgia. To do so, we followed epidemiological criteria[21] and collapsed and scaled data from our sample into these criteria. Published criteria include thresholds obtained from the Widespread Pain Index (WPI) to assess widespread pain, and Symptom Severity (SS) scores to assess for fatigue, waking unrefreshed, and cognitive symptoms. We collapsed responses on 29 regions of the MBM into the 19 bodily pain areas on the WPI (excluding facial, pelvic and head pain). For the SS score, we utilized items from the CSIA that ask the same questions and scaled responses to a 4-point Likert-type scale. All study participants had symptoms for over three months. Thus, consistent with ACR criteria, participants who scored ≥ 7 on the WPI and SS ≥ 5 or WPI of 3–6 and SS ≥ 9 were categorized as meeting epidemiological criteria for fibromyalgia.

Data Analytic Plan

All analyses of study hypotheses were conducted using R software.[58] We sought to determine the specific relations of trauma load and PTSD symptoms with each of the three CS-related clinical features (MBM, MPQ, and CSIA); however, theoretical and quantitative overlap exists in the various indicators of CS. Thus, we used multivariate regression and tested mediation via path models in which all three CS variables were included simultaneously as dependent variables to account for residual covariance among them. Analyses were conducted using the R package "lavaan."[59] Given the positively skewed (CSIA, MPQ) or Poisson (e.g., the MBM is a count measure) distribution of all CS outcome variables, we used weighted least squares estimation with mean and variance adjustments for all models.[60] We computed scaled scores only when at least 80% of the relevant item-level data was available. To maximize available data, we used pairwise (rather than listwise) deletion during estimation, resulting in a final sample size for our path models of $N = 191$. Model fit information was generated using robust standard errors which are relatively unaffected by non-normality. Model fit is reported for unsaturated models and evaluated using a combination of CFI > .95, TLI > .95, and RMSEA < .05 indicating good model fit. Significance of model paths was examined using unstandardized path coefficients ($p < .05$).

Results

Participant demographics are reported in Table 1. The sample ($M_{age} = 44.89$, $SD = 14.23$) was largely female (79.7%) and White (81.2%), although with diverse educational and vocational statuses. Men and women did not differ in reported trauma exposure, current PTSD symptoms, or pain intensity (MPQ), but women reported higher levels of widespread pain (MBM), $t(57.60) = 3.19$, $p = .002$, $d = .42$, and polysomatic complaints (CSIA; $M_{men} = 39.13$, $M_{women} = 49.42$), $t(195) = 3.02$, $p = .003$, $d = .46$. Women also reported greater use of emotional suppression (MEAQ-Distraction & Suppression scale), $t(190) = 3.25$, $p = .001$, $d = .47$, but did not differ from men on any other MEAQ subscale. Given the associations among gender and CS indicators, we included gender as a covariate in all analyses.

Supplemental Table 1 provides a list of documented pain-related conditions of the analyzed sample as indicated in medical records (as reviewed by LJC and LCM), organized by

diagnosis type and pain region for increased interpretability. Commonly-diagnosed conditions included chronic low back pain, neck pain, chronic migraine, osteoarthritis, fibromyalgia, irritable bowel syndrome, endometriosis, and interstitial cystitis/bladder pain syndrome. On average, participants had 2.69 (*SD* = 1.84) diagnosed pain-related conditions.

Descriptive statistics of and correlations among the variables of interest are reported in Table 2. Of note, the moderate correlation (*r* = .32) between trauma exposure (THQ) and PTSD symptoms (PCL) indicates that these measures do in fact measure at least partially distinct constructs. The correlations among the three CS clinical indicators (widespread pain, pain severity/intensity, and polysomatic complaints) are higher but reflect only 22% to 38% shared variance, indicating overlapping but distinguishable constructs.

Are trauma exposure and PTSD symptoms related to CS outcomes?

Trauma exposure was significantly (and similarly) associated with all three CS indicators, explaining 12–14% of the variance of each CS outcome. Figure 1 displays the associations between trauma exposure (THQ) and the three CS outcomes (MBM, MPQ, and CSIA), taking into account the covariance among these outcomes. Zero-order correlations between PTSD symptoms (PCL) were moderately-to-highly correlated with CS outcomes (Table 2). Together, these results confirm the significant and meaningful associations of both trauma exposure and PTSD symptoms with all three CS indicators, suggesting the importance of further delineating potential indirect links among these constructs.

Do PTSD symptoms explain the trauma exposure-pain relationship?

PTSD symptoms significantly, partially mediated the relationship between trauma exposure and all three CS outcomes ($\chi^2(1) = 0.37$, CFI = 1.00, TLI = 1.04, RMSEA = .00) (Figure 2). Trauma exposure directly explained 1.3% (CSIA) to 4.5% (MBM, MPQ) of the variance in CS outcomes, whereas PTSD symptoms proved to be much more strongly related to CS, accounting for 4.9% (MBM) to 40% (CSIA) of the variance in these outcomes. In terms of the mediated path, 0.5% (MBM), 1.9% (MPQ), and 3.7% (CSIA) of the variance in CS outcomes was explained indirectly by the effect of trauma exposure through PTSD symptoms (i.e., mediated effects). Together, trauma exposure and PTSD (and gender as a covariate) explained 16% of the variance in widespread pain, 33% in pain intensity, and 50% in polysomatic complaints. These findings indicate that, although trauma exposure is associated with indicators of CS, a significant portion of this influence is conveyed via greater PTSD symptoms. Of note, we compared our hypothesized model (THQ->PCL->CS) to a reverse mediation model in which CS indicators mediated the trauma-PTSD relationship (THQ->CS->PCL). Results suggested a poorer practical fit of this alternate model on two of three fit indices ($\chi^2(1) = 3.96$, CFI = .99, TLI = .82, RMSEA = .13),[61] providing further support for our primary mediation model.

Does experiential avoidance moderate or mediate the relations among trauma exposure, PTSD symptoms, and CS?

Total experiential avoidance did not significantly mediate or moderate either the relation between trauma and PTSD symptoms or between PTSD symptoms and CS outcomes (*p*s > .11). Examination of specific subscales of experiential avoidance indicated that results were

largely identical when testing only the theory-relevant MEAQ subscale emotional suppression ($p > .10$); however, emotional suppression partially mediated the relationship between PTSD symptoms and pain intensity ($b = .006$, $z = 2.99$, $p = .003$). This finding may suggest a potential path in which trauma exposure is linked to PTSD symptoms, which are in turn linked to emotional suppression that adversely impacts pain intensity.

Population trauma characteristics and clinical application:

Table 3 indicates trauma characteristics of the sample, including various forms of trauma exposure (THQ General Disaster and Trauma, Crime-Related, and Physical/Sexual Abuse subscales) and PTSD symptomology (PCL Intrusion, Avoidance, Cognition/Mood, and Arousal/Reactivity subscales). Overall, participants reported exposure to an average of 5.75 traumatic events in their lifetime ($SD = 3.95$), and 28.2% of the overall sample met provisional criteria for PTSD. To demonstrate the clinical relevance of these trauma-related concepts, we examined trauma characteristics in those who met ACR epidemiological criteria for fibromyalgia ($n = 83$, 42.6% of the sample) to those not meeting epidemiological criteria for FM ($n = 107$, 54.9%). When compared to those without FM, individuals with a probable fibromyalgia diagnosis[11] had significantly higher levels of all forms of trauma exposure, PTSD symptoms, and rates of provisional PTSD diagnosis (44.6% vs. 16.8%, $p < 0.001$), indicating that trauma and its psychological sequelae are highly associated with the paradigmatic CS condition, FM.

Discussion

We examined how trauma exposure and PTSD symptoms are related to different clinical indicators of CS in a sample of chronic pain patients. Specifically, we were interested first in confirming that trauma exposure was related to clinical indicators of CS, and then whether these effects were directly due to trauma exposure itself, or instead were better explained by the extent of PTSD symptoms related to the trauma. Results indicate that as exposure to trauma increases, patients experience greater CS, that is, widespread pain, pain intensity, and polysomatic complaints. Importantly, when including current symptoms of PTSD in the model, the relations between trauma exposure and measures of CS weakened. Findings from mediation analyses indicated that this diminution of trauma-CS links was due to the fact that PTSD symptoms partially explained the effect of trauma exposure on widespread pain, pain intensity, and polysomatic symptoms. These results suggest that both exposure to trauma and PTSD symptoms are relevant to CS outcomes, and that whereas trauma exposure predicts clinical indicators of CS, a significant portion of this influence is conveyed via greater PTSD symptoms. Notably, PTSD symptoms explained up to four times as much variance in CS outcomes as trauma exposure itself, suggesting that it is most important to consider PTSD symptoms (as opposed to trauma exposure per se) in the assessment, treatment, and conceptualization of CS.

Prior work reported associations between trauma exposure and chronic pain generally, and CS specifically.[24,30] For example, abuse exposure has been associated with heightened temporal summation of pain[30] and longer ischemic pain response,[62] and PTSD is associated with abnormal painful reactions to stimuli and reduced pain thresholds.[24] The current

findings expand upon this prior work, indicating that trauma exposure may not be the only trauma-relevant factor in determining the extent of CS-related phenomena, but rather, that the degree of ongoing PTSD symptoms resulting from the trauma exposure may play a larger role. Although intriguing, these preliminary findings need to be replicated in future work, ideally incorporating objective laboratory markers of CS (i.e., temporal summation of pain).

With this study, we also aimed to delineate the role of experiential avoidance, specifically emotional suppression, as either a mediator or moderator of trauma and CS symptoms. Surprisingly, overall experiential avoidance neither moderated nor mediated the relationships between trauma and indications of CS, either at the trauma-PTSD symptom path, or the PTSD-CS path. However, when we examined emotional suppression specifically, analyses indicated that emotional suppression partially mediated the relationship between PTSD symptoms and pain intensity. This suggests that for those people exposed to trauma, experiential avoidance in general does not explain the relations between trauma and CS, but that active suppression of emotion may partially explain why patients with PTSD symptoms experience greater pain levels.

Our findings indicate only partial mediation by PTSD symptoms on the relationship between trauma exposure and CS, suggesting that other mediators stemming from trauma exposure or PTSD may be important in fully explicating the trauma exposure-CS relation. An individual exposed to trauma may undergo physical or neurological changes as a result of trauma exposure that precede CS. For example, repeated physical injury may result in increased inflammation, or excessive central nervous system activation following nociceptive input, which have been cited as factors influencing the development of CS.[63,64] Further, the mutual-maintenance model proposes several additional pathways linking PTSD and pain. For example, attentional biases, anxiety sensitivity, and the cognitive burden resulting from both PTSD and pain have all been proposed as reinforcing both conditions.[35] Further, other processes not measured in this study such as shared neurophysiological mechanisms between PTSD and CS may play a role in maintaining this relationship. For example, neurochemical factors such as glutamate or NMDA receptor dysregulation have been cited as present in both CS[65,66] and PTSD.[67]

Clinical Implications

It is important to assess current levels of PTSD symptoms to better understand, predict, and potentially treat clinical manifestations of CS among patients presenting with chronic pain. Our findings indicate that it is not only trauma exposure itself that may be critical, but rather associated PTSD symptoms linked to that exposure may help explain CS.

Overall, 28.2% of our chronic pain sample met provisional criteria for PTSD. Further, among patients likely meeting criteria for fibromyalgia, a prototypical CS condition, the number of patients meeting provisional PTSD criteria was substantial—44.6%—compared to a much lower prevalence (16.8%) among those patients with chronic pain not meeting FM criteria. Assuming that observed associations between PTSD symptoms and CS are causal, it would be important to routinely assess and treat posttraumatic stress in patients with chronic pain, and in particular those with symptoms of CS. That is, treating the PTSD or otherwise

attenuating symptoms of posttraumatic stress could have a marked impact on the severity of pain and CS-related symptoms, quality of life, and day-to-day functioning of these patients. We encourage providers to interview for trauma-related symptoms, if not both trauma exposure and symptoms, when evaluating patients with chronic pain, or at a minimum, to use brief measures, such as the publicly-available 4-item primary care PTSD screening tool[68] or the PCL-5 used in this study, to allow the provider to assess the potential impact of PTSD on symptom presentation and to provide motivation, education, and validation to the patient.[68]

Further, there are several efficacious treatments for PTSD[69–71] and good reason to believe that this proposed symptom maintenance mechanism (i.e., symptoms of PTSD) could be adequately targeted in clinical trials with patients who have CS-related pain conditions. Several pilot studies suggest the efficacy of trauma-focused psychotherapies for patients with co-morbid trauma and either fibromyalgia[72] or chronic back pain.[73] If larger trials confirm these results, and if reductions in PTSD symptoms predict reductions in CS indicators, this would be compelling evidence of the causal role of trauma symptoms in driving CS and the value of assessing and treating trauma symptoms directly. Relatedly, directly targeting emotional suppression by enhancing emotional awareness and expression is intended to increase the acceptance and experience of trauma- and conflict-related emotions and eventually their resolution, and this approach has recently been shown efficacious with heterogeneous chronic pain populations[74] and those with fibromyalgia.[75] Addressing emotional suppression in psychotherapy, particularly among patients with CS and PTSD, may be an avenue for future intervention and study.

Strengths and Limitations

This study has multiple strengths to note. First, we examined a relatively large clinical sample with a variety of chronic pain conditions, which helps to capture the breadth of symptoms across the chronic pain spectrum. To our knowledge, this is the first study to ascertain how traumatic stress is related to different patient-reported clinical manifestations of CS in adults with chronic pain. We gathered information on multiple dimensions of CS that exist in parallel with chronic pain. We also included both trauma exposure and current symptoms of posttraumatic stress in our study design—a discernment not often made but known to affect symptom presentation.

The study of trauma in clinical populations is a challenge and often relies on retrospective self-report of traumatic events. This raises concerns of recall bias and subjective interpretation of events. Ideally, we would have evaluated participants in-person with structured interviews for medical diagnoses and the presence of PTSD; however, we used validated self-report measures and clinical cutoffs consistent with psychological and medical diagnoses to improve the validity of our categorizations and interpretations. Further, the validity of self-reported experiential avoidance is complicated, as individuals who are avoidant may be unaware of or underreport this behavior. We suggest using a behavioral task to capture this construct in future work. Our data are cross-sectional; to make more definitive assertions about the development of CS following trauma and other causal implications, longitudinal designs are needed. The results of our study should therefore be

interpreted as elucidating patterns of overlap among these constructs relevant to CS, and further research is required to explain the temporal mechanisms of such overlap. For example, following a sample with exposure to traumatic events or experiencing acute pain, but without CS manifestations, would allow researchers to determine whether PTSD symptoms predict the development of chronic pain and CS and may be in part responsible, at least in some individuals, for centrally-maintained pain. Last, the incorporation of objective laboratory markers of CS, such as temporal summation, in addition to clinical measures of CS would enhance the validity of findings in future research.

Conclusions

In conclusion, our findings suggest that whereas trauma exposure is relevant to CS-related clinical outcomes, PTSD symptoms account for a significant portion of the relation between traumatic experiences and CS. Given that patients with CS disorders are often characterized by complicated clinical presentations without promising prognoses, it is worthwhile to identify potential treatment targets that may often be overlooked. We suggest that PTSD symptoms might be one such variable, and we encourage clinical researchers to consider PTSD symptoms as a potential mechanism of CS development, as well as a target of treatment in chronic pain conditions.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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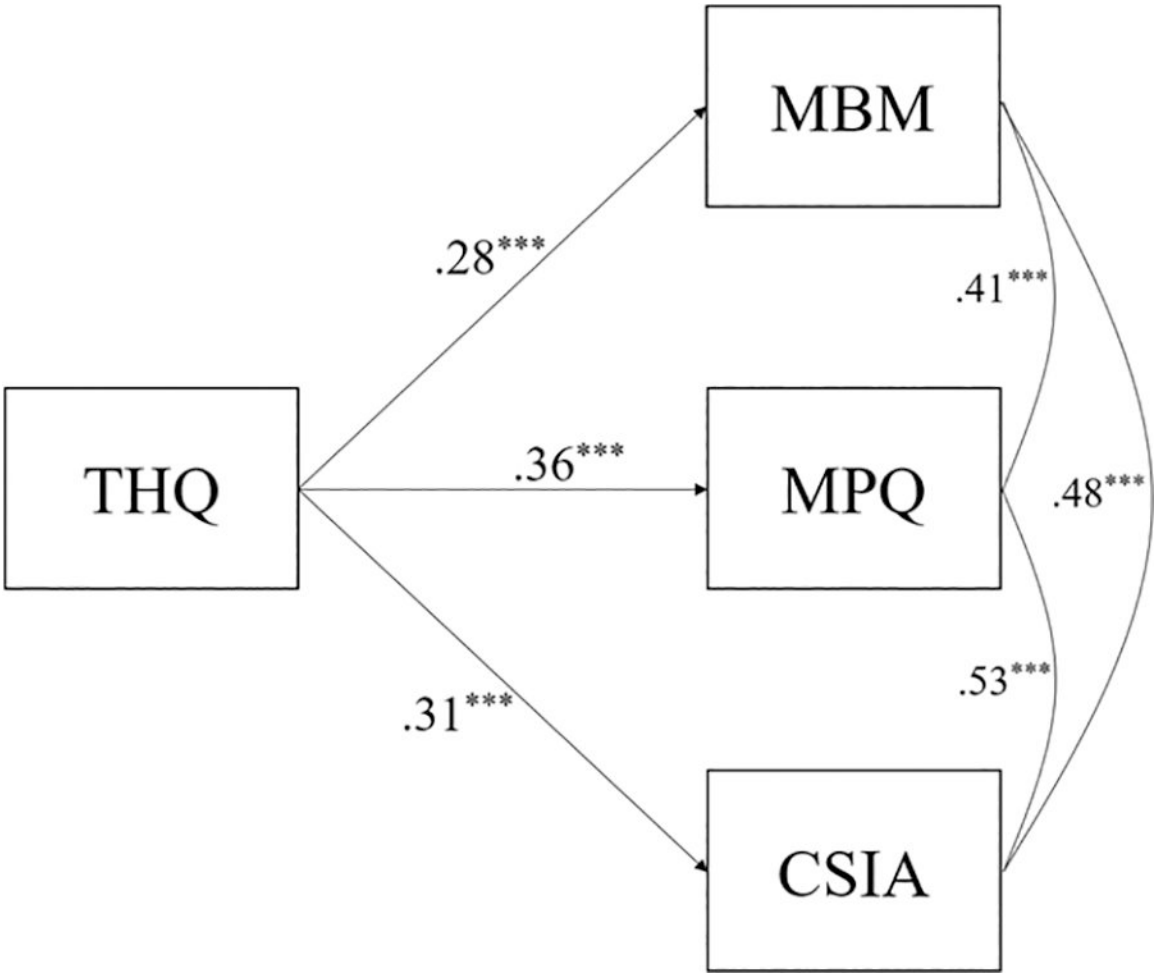

Figure 1.

Trauma exposure as a predictor of CS indicators, controlling for gender. All regression and covariance parameters are standardized. $N = 191$. THQ = Trauma History Questionnaire; MBM = Michigan Body Map; MPQ = McGill Pain Questionnaire; CSIA = Central Sensitization Inventory, Part A.

* $p < .05$

** $p < .01$

** $p < .001$

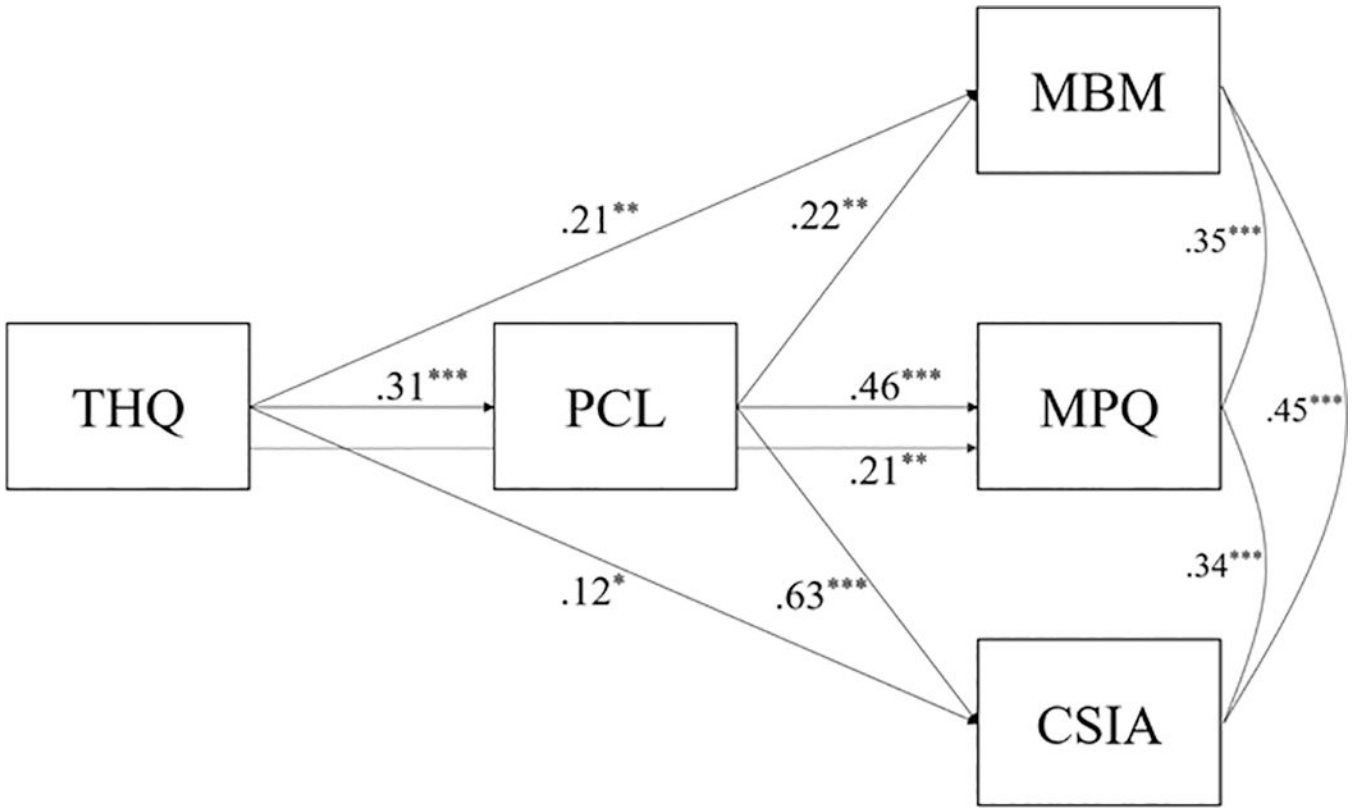

Figure 2.

Posttraumatic symptoms as a partial mediator of the trauma exposure-CS outcome relationship, controlling for gender. All regression and covariance parameters are standardized. PCL partially mediates the THQ to MBM, MPQ, and CSIA relations. $N = 191$. THQ = Trauma History Questionnaire; PCL = PTSD Checklist—DSM-5 Version; MBM = Michigan Body Map; MPQ = McGill Pain Questionnaire; CSIA = Central Sensitization Inventory, Part A.

* $p < .05$

** $p < .01$

** $p < .001$

Table 1:

Demographic characteristics (N = 202)

	M	*SD*
Age	44.89	14.23
	n	%
Gender		
Women	161	79.7
Men	36	17.8
Not reported	5	2.5
Race		
African American, Black	17	8.4
Asian/Pacific Islander	4	2.0
Hispanic, Latina/o	5	2.5
Native American/American Indian	1	0.5
White	164	81.2
Multiracial	6	3.0
Other	4	1.9
Unknown	1	0.5
Relationship Status		
Single	53	26.2
Married/Domestic Partnership	108	53.5
Divorced	36	17.8
Separated	4	2.0
Widowed	1	0.5
Educational Attainment		
High school diploma or equivalent	15	7.4
Vocational or technical school	8	4.0
Some college	40	19.8
Bachelor’s degree	70	34.7
Master’s degree	44	21.8
Doctorate or professional degree	17	8.4
Other	7	3.5
Employment Status		
Full-time	106	52.5
Part-time	18	8.9
Self-employed	9	4.5
Unemployed	10	5.0
Retired	20	9.9
Unable to work	37	18.3
Income		
Under $10,000	8	4.0
$10,000–19,999	16	7.9

	M	*SD*
$20,000–$50,000	48	23.8
$50,000–$100,000	55	27.2
$100,000–$150,000	36	17.8
$150,000 or higher	15	7.4
Preferred not to respond	24	11.9
Disability Status		
Currently receiving disability	33	16.3
In the process of applying for disability	16	7.9
Not receiving or applying for disability	113	55.9
Unknown	40	19.8

M = mean; *SD* = standard deviation.

Table 2:

Descriptive statistics of and correlations among the trauma, pain, and experiential avoidance measures (N = 175 – 202).

	M	*SD*	1	2	3	4	5	6	7	8	9	10	11
Trauma													
1. THQ	5.75	3.95											
2. PCL	23.13	17.81	.32***										
Pain													
3. MBM	9.42	7.20	.32***	.32***									
4. MPQ	3.16	1.77	.28***	.51***	.47***								
5. CSIA	47.59	17.68	.28***	.65***	.55***	.62***							
Experiential Avoidance													
6. MEAQ Total	190.80	39.75	.09	.59***	.22**	.36***	.42***						
7. Behavioral Avoidance	34.60	10.85	−.03	.41***	.14*	.26***	.34***	.85***					
8. Distress Aversion	45.25	11.11	.07	.54***	.18*	.30***	.33***	.77***	.63***				
9. Procrastination	22.25	7.74	.07	.47***	.17*	.24***	.37***	.72***	.52***	.36***			
10. Distraction and Suppression	27.11	6.53	.07	.31***	.23**	.38***	.34***	.60***	.51***	.52***	.26***		
11. Repression and Denial	33.31	10.87	.18*	.40***	.09	.22**	.20**	.71***	.45***	.38***	.50***	.31***	
12. Distress Endurance	48.84	8.01	.06	−.40***	−.02	−.09	−.22**	−.59***	−.44***	−.28***	−.50***	−.08	−.25***

Note. Spearman rank-order correlation coefficients (ρ) reported to account for skewed data, except for relations among normally distributed PCL and MEAQ scales), for which Pearson product-moment correlation coefficients (*r)* are reported. THQ = Trauma History Questionnaire; PCL = PTSD Checklist; MBM = Michigan Body Map; MPQ = McGill Pain Questionnaire; CSIA = Central Sensitization Inventory-Part A; MEAQ = Multidimensional Experiential Avoidance Questionnaire; *M* = mean; Med. = median; *SD* = standard deviation.

* $p < .05$;

** $p < .01$;

*** $p < .001$

Table 3

Trauma characteristics of the total sample and by fibromyalgia diagnosis

Group	***Total***		***FM-Dx+***		***FM-Dx−***				
***N* (%)**	**195**		**83 (42.6%)**		**107 (54.9%)**				
Trauma Characteristics	***M***	***SD***	***M***	***SD***	***M***	***SD***	***t***	***p***	***d***
Trauma Exposure									
Total	5.75	3.95	6.90	3.86	4.80	3.85	3.73	<.001	0.54
General Disaster and Traumatic Events	3.54	2.54	3.98	2.50	3.19	2.57	2.12	.04	0.31
Crime-Related	0.82	0.97	1.09	1.03	0.59	0.87	3.58	<.001	0.52
Physical and Sexual	1.06	1.31	1.43	1.35	0.78	1.23	3.43	<.001	0.50
Posttraumatic Symptoms									
Total	23.13	17.81	31.78	18.65	16.44	13.81	6.01	<.001	0.93
Intrusion	5.25	4.93	6.70	5.38	4.15	4.28	3.48	<.001	0.52
Avoidance	2.43	2.36	3.34	2.56	1.73	1.94	4.71	<.001	0.71
Cognition/Mood	8.27	7.18	11.65	7.52	5.74	5.70	5.84	<.001	0.89
Arousal/Reactivity	6.99	5.39	9.94	5.61	4.73	3.93	7.06	<.001	1.08
	n	%	*n*	%	*n*	%	χ^2	*p*	φ
Provisional PTSD Diagnosis	55	28.2	37	44.6	18	16.8	15.89	<.001	0.29

Note. Statistical difference tests compare Fibromyalgia+ and Fibromyalgia- groups. Five participants are missing Fibromyalgia diagnosis information, but are included in the Total column. *df*-adjusted *t*- and *p*-values reported for comparisons with unequal variances determined unequal by Levene's test (i.e., all except THQ Total, General, and Crime). Trauma exposure data drawn from the Trauma History Questionnaire (THQ). Posttraumatic symptom data drawn from the PTSD Checklist—DSM-5 Version. FM-Dx+ = probable fibromyalgia diagnosis present[11]; FM-DX- = probable fibromyalgia diagnosis absent; *M* = mean; *SD* = standard deviation; PTSD = posttraumatic stress disorder