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Exploring the Links Among Borderline Personality Disorder Symptoms, Trauma, and Pain in Patients with Chronic Pain Disorders

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Abstract

Objective: Chronic pain and borderline personality disorder (BPD) are commonly comorbid and jointly associated with increased symptoms of both disorders and clinical and functional impairment. Little is known, however, about specific links between these disorders. In a cross-sectional study of patients with chronic pain, we compared participants high or low on BPD symptoms on patterns of pain experience and types of child and adult traumas.

Methods: Adults (N = 181) with chronic pain completed self-reports of pain severity, dimensions of pain experiencing, body coverage of pain, and clinical indicators of central sensitization (i.e., chronic hypersensitivity of the central nervous system), as well as measures of child and adult physical abuse, sexual abuse, trauma, and neglect. Participants also completed the McLean Screening Instrument for BPD.

Results: Participants with clinically significant BPD symptoms (n = 32) reported more childhood sexual trauma, punishment, and neglect, as well as adult physical/sexual trauma, than those without elevated BPD symptoms. Among participants with clinically significant BPD symptoms, affective pain and central sensitization were elevated, potentially explained by heightened negative affect in BPD.

Conclusion: BPD symptoms are associated with increased clinical severity among patients with chronic pain as well as a unique manifestation of pain experiencing (i.e., increased affective pain

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and central sensitization in particular). Childhood trauma of all types is associated with chronic pain and BPD co-occurrence. Researchers and clinicians should assess for BPD in people with chronic pain to enhance conceptual models of the transaction between these disorders and to improve clinical care.

Keywords

BPD; central sensitization; chronic pain; fibromyalgia; trauma

Introduction

The comorbidity of borderline personality disorder (BPD) and chronic pain has garnered increased research attention in recent years. BPD, which affects 1-5% of the U.S. population, is a prevalent, debilitating, and costly disorder characterized by unstable mood, behavior, relationships, and sense of self, as well as self-harm and suicidality (1). Several recent studies suggest that chronic pain is more frequent and more severe in people with BPD than those without BPD (2-6), and up to 65% of patients with BPD have a lifetime chronic pain diagnosis (7). The presence of chronic pain also predicts increased medical complaints and other symptoms (e.g., anxiety) in BPD and decreased likelihood of BPD remission (8).

Likewise, patients with chronic pain have an increased prevalence of BPD symptoms and diagnoses, with up to 30% meeting BPD diagnostic criteria (9-11). In those with chronic pain, the presence of BPD is associated with increased pain symptoms (12-18), pain-related interference (12,18), and somatic and psychological symptoms (4,17,19), and a decreased likelihood of pain remission (20). The increased symptom severity and poor prognosis associated with this comorbidity has been widely demonstrated across community, non-clinical, and clinical samples (2,12,21-23).

Multiple theoretical models exist to explain the co-occurrence of BPD and chronic pain. These include a diathesis-stress model where liabilities in emotional regulation become taxed by chronic pain onset, thus contributing to personality pathology (24,25), pain as a consequence of chronic dysregulation in BPD (6), a focus on their shared neuroanatomical underpinnings (26), and acknowledging shared risk factors between the conditions.

Considerable research supports the view that trauma (especially childhood sexual and emotional abuse) plays an important role, along with other biological and psychosocial risk factors, in the development of BPD (27-30). Similarly, reports of trauma are elevated in patients with chronic pain, and trauma is a major etiological factor in central sensitization (31-33), that is, chronic hypersensitivity of the central nervous system, which predisposes individuals to increased pain experiencing and is an important maintaining factor in chronic pain conditions (34,35). Only one study, however, has examined the role of trauma in the manifestation of pain among patients with BPD, reporting that non-sexual childhood abuse and neglect was positively associated with average pain severity (2). No studies have examined associations between trauma and BPD symptoms in patients with chronic pain, and, despite a putative relationship (15), little is known regarding clinical manifestations of central sensitization, such as the prevalence of fibromyalgia—a prevalent pain disorder

characterized by central sensitization—in those with BPD or elevated BPD symptoms (36). Thus, the degree to which experiences of trauma and various pain manifestations are associated with BPD symptoms, and the extent to which trauma and BPD symptoms are interactive predictors of pain among patients with chronic pain, is unknown.

Despite clear evidence linking BPD and pain, questions remain about how BPD influences pain patterns and experiencing. For example, although the presence of BPD increases chronic pain risk, BPD may not increase risk for having multiple comorbid chronic pain diagnoses (37). In a non-clinical sample, BPD symptoms were associated with pain interference but not pain severity (38), contrary to findings from patients with chronic pain (14). One possible source of contradictory findings about pain in BPD has been labeled the "pain paradox" of BPD, in which those with BPD report greater chronic pain but attenuated acute pain response, such as to self-injury (39). Carpenter and Trull evaluated the pain paradox experimentally in undergraduates during a cold pressor task, finding a link between BPD symptoms and pain response only in the absence of a history of self-harm (40). Clearly, to provide a nuanced understanding of the complex relations between BPD and pain, multidimensional assessment of pain is essential, including distinct measurement of pain chronicity, pain type, pain severity, and central sensitization (41).

Aims and Hypotheses

In a sample of patients with chronic pain, we first aimed to replicate previous findings: BPD symptomatology is associated with greater clinical impairment (e.g., elevated symptoms of anxiety and depression) as well as greater pain severity. We also sought to answer three novel questions: Which types of pain experiencing are most elevated in the context of co-occurrence of chronic pain and BPD? Is a history of trauma associated with this co-occurrence? If so, what types of trauma are most likely to be associated with this co-occurrence? We hypothesized that central sensitization and fibromyalgia would be especially elevated in the context of co-occurring BPD symptoms and chronic pain (15), that childhood sexual abuse and neglect would be particularly associated with BPD symptoms (27,30), and that childhood—but not adult—trauma would be an independent correlate of pain, when included in models with BPD symptoms as a joint predictor (2). Furthermore, as exploratory aims, we tested the role of negative affect in the association between BPD symptoms and pain, given growing evidence of its role in helping to explain these associations (17,19,21,22), and we examined statistical interactions between trauma and BPD symptoms in predicting pain.

Method

Participants and Procedures

The institutional review board reviewed and approved study procedures, all participants provided informed consent, and the study was carried out in accordance with the Declaration of Helsinki. From January 2016 to March 2017, we recruited patients with chronic pain to complete a series of validated questionnaires (online at home, by paper, or at an on-site computer station) assessing BPD symptoms, child and adult trauma experiences, and current pain-related and emotional symptoms. We identified participants through a large university-

affiliated medical outpatient clinic, a hospital-wide research listserv, and locally via online advertisement through ResearchMatch[1] (42) with a recruitment radius in the hospital catchment area. Eligible participants were English-speaking adults (> age 18) who reported a chronic pain diagnosis, as indicated 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)" (43). Exclusion criteria included a diagnosis of cognitive or thought disorder, current substance dependence, or active suicidal ideation. Of 211 consenting participants, 181 (M_{age} = 44.62, 80.1% female) completed relevant measures and comprise the current sample. We determined specific pain diagnoses and comorbid psychiatric diagnoses via electronic health record review (Appendix). Four patients had insufficient medical record information (i.e., no diagnoses available) and 1 was external to the medical center system and had no medical record available. We reimbursed participants with a $20 gift card in exchange for study participation. Analyses from this data set have been previously published, examining the impact of posttraumatic stress disorder (PTSD) in patients with interstitial cystitis (44) and the association between trauma exposure and central sensitization in patients with chronic pain (41). The current analyses have not been previously reported.

Measures

McLean Screening Instrument for BPD (MSI-BPD)—The MSI-BPD (45) is a 10-item self-report questionnaire that screens for the presence of BPD. Dichotomous items assess each of the nine BPD criteria according to the Diagnostic and Statistical Manual-5th Edition (46) (the paranoia/dissociation criterion is assessed via two items). We categorized participants as likely or unlikely to meet BPD criteria using a cut score of 7, which has shown good sensitivity (.81) and specificity (.85) in predicting a BPD diagnosis as assessed by structured diagnostic interviews (45). For consistency and interpretability, all analyses utilize the dichotomized MSI-BPD.

Trauma—We assessed childhood trauma exposure with the *Childhood Abuse and Trauma Scale* (CATS) (47), a 38-item questionnaire with three subscales: sexual abuse, punishment, and neglect. Items rated from 0 (never) to 4 (always) are summed for each subscale. The CATS was originally developed on clinical adolescent inpatients but was later applied to non-clinical young adults. The subscales (α = .63 - .86) and total scale (α = .90) show acceptable to excellent internal consistency, except the punishment subscale (α = .63). In the present study, internal consistency was acceptable to excellent for all scales (α = .76 – .94). We also assessed adult trauma exposure with the *Trauma History Questionnaire* (THQ) (48), which consists of 24 yes/no items summed across three types of trauma: general disaster/ traumatic events, crime-related events, and physical/sexual traumas. The test-retest reliability of the THQ is acceptable (r = .70) (48). To obtain a measure of adult trauma only, we developed an adjusted THQ score by summing only traumas that occurred at age 18 or older.

Pain—In addition to the number of chronic pain diagnoses obtained from patients' medical records, we assessed eight pain-relevant variables capturing pain severity, location(s), the

quality and intensity of pain, degree of polysomatic complaints, and whether or not the individual met epidemiological criteria for a fibromyalgia diagnosis:

Current pain severity was assessed using the *Numeric Pain Rating Scale* (NRS-11) (49), an 11-point scale from 0 ("No pain") to 10 ("Pain as bad as you could imagine"). We averaged four ratings (current; and worst, least, and average in the past 24 hours) to create a single pain severity score (α = .90).

Widespread pain was assessed via the *Michigan Body Map – Revised Version* (MBM) (50), a two-sided body image with check-box responses for 35 body areas where chronic pain (i.e., longer than 3 months) might exist. We summed the number of pain areas endorsed for a measure of widespread pain. The 1-to-2-week test-retest reliability of the MBM is acceptable (r = .77) (50).

Quality and intensity of pain was assessed through the *McGill Pain Questionnaire-Short Form-Revised* (MPQ)(51). The MPQ is a comprehensive self-report measure capturing four dimensions of pain, rated on 22 different descriptive words on a 0-10 scale. The four pain dimensions are continuous (e.g. throbbing, gnawing, aching), intermittent (e.g. shooting, sharp, splitting), neuropathic (e.g. hot-burning, cold-freezing tingling), and affective (e.g. tiring-exhausting punishing-cruel, fearful). The four subscales have demonstrated good-to-excellent internal consistency (α = .84 – .92) (52) and acceptable-to-good reliability in our sample (α =.72 – .84).

Polysomatic complaints associated with central sensitization were assessed via Part A of the *Central Sensitization Inventory* (CSIA) (53). The CSIA assesses 25 health-related symptoms common in conditions involving central sensitization; ratings from 0 (never) to 4 (always) are summed. Part A of the CSIA has demonstrated an internal consistency of .88 (53), corroborated by excellent internal consistency in the present study (α = .91), and differentiates CS from non-CS groups (54).

Fibromyalgia diagnostic criteria.: To corroborate chart diagnoses of fibromyalgia, we generated provisional fibromyalgia diagnoses via the American College of Rheumatology's epidemiological criteria (55), which utilizes thresholds obtained from the Widespread Pain Index (WPI) to determine widespread pain and Symptom Severity (SS) scores to assess for fatigue, waking un-refreshed, 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. Thus, using ACR 2010 criteria, we classified participants who scored ≥ 7 on the WPI and SS ≥ 5, or WPI of 3-6 and SS ≥ 9 as meeting epidemiological criteria for fibromyalgia.

Emotional Distress—We used the *Hospital Anxiety and Depression Scale* (56) to assess current (past week) anxiety (HADS-A) and depression (HADS-D); the seven items in each subscale were rated 0 to 3 and summed. Both HADS subscales have generally shown good internal consistency (α = .82 – .83) (57), confirmed in our data ($\alpha_{\text{HADS-A}}$ = .87; $\alpha_{\text{HADS-A}}$ = .85).

Quality of Life—We used the *Satisfaction with Life Scale* (SWLS) (58) to assess quality of life, summing the ratings (1 = strongly disagree to 7 = strongly agree) of the five items. The SWLS has shown good internal consistency in medical outpatients (α = .87) (59), corroborated in our study (α = .89).

Data Analysis

We compared subgroups of participants with (BPD+) or without (BPD−) a probable BPD diagnosis (as assessed by the MSI-BPD) with respect to their pain-related variables, emotional distress, trauma, and trauma symptoms. Differences between groups were determined via analysis of covariance (ANCOVA), controlling for age and gender. We also evaluated the role of emotional distress (i.e., anxiety and depression) by conducting a set of analyses on significant group differences while controlling for anxiety and depression scores (simultaneously, alongside age and gender). Furthermore, when including anxiety/depression in the model resulted in the effect of BPD becoming non-significant, we tested the significant of the indirect effects of anxiety/depression scores through path analyses (PROCESS macro v. 3.4, Model 4) (60).

Finally, we explored the interactive associations of BPD symptoms (dichotomous) and trauma (centered) with pain outcomes through multiple linear regression analyses with 2-way interaction terms, controlling for age and gender. To reduce the number of analyses, CATS and THQ (Adult) total scores (rather than subscales) were used as predictors in two separate sets of models, resulting in 14 models (1_{BPD} x 2_{Trauma} x 7_{Pain}).

All analyses were conducted using SPSS Version 26, and results were interpreted using family-wise error correction (i.e., Bonferroni alpha correction = .05 / # of tests) for each group of tests (i.e., demographics [α = .006], pain diagnoses [α = .003], comorbid psychiatric conditions [α = .01], initial BPD group differences [α = .003], covariate-adjusted analyses [α = .006], and main effects of interaction models [α = .004]). However, we did not correct alpha level for the interaction terms in tests of moderation given that several factors tend to reduce the power of interaction tests (61), and, thus, used a traditional .05 cutoff for interpretation.

Results

Sample Characteristics

The sample of 181 patients was largely female (80.1%) and white (81.2%), with diverse educational and vocational statuses (Table 1). The majority of participants presented with diagnoses of back, head, or extremity pain, although many were diagnosed with diffuse musculoskeletal pain (e.g., fibromyalgia), pelvic pain, and abdominal pain (Table 1). Patients were diagnosed with an average of 2.69 chronic pain conditions (*Med.* = 2, range = 1-12) (see also Appendix).

On the MSI-BPD, 17.7% of the sample (n = 32) endorsed 7+ items, indicating the likely presence of BPD. As shown in Table 1, there were no significant differences (chi-square tests, after alpha correction) between BPD+ and BPD− participants in demographic variables except age; BPD+ participants were younger than BPD− participants. Although not

significant, the odds of being female were more than 5 times higher in the BPD+ group, and odds of being single, unemployed, and having an income less than $50K were each also more than 2 times higher in the BPD+ group. Although BPD+ participants did not differ significantly from BPD− participants on any category of medical chart pain diagnosis, BPD + participants were significantly more likely to meet the threshold of a provisional fibromyalgia diagnosis according to epidemiological criteria compared to BPD− participants ($\chi^2 = 19.32$, $p < .001$). In the case of both chart records and ACR criteria, fibromyalgia was twice as prevalent among BPD+ participants than BPD− participants ($PR_{CHART} = 2.13$; $PR_{ACR} = 2.24$).

Associations Among BPD Symptoms, Pain Diagnoses, Pain Symptoms, and Clinical Variables

Table 2 reports bivariate correlations among BPD symptoms (dichotomous) and the pain, emotional distress, and trauma variables. BPD symptoms were significantly correlated with most pain measures except current pain severity (NPRS-11) and intermittent pain (MPQ). BPD symptoms were associated with all categories of childhood trauma but with only adult physical/sexual trauma. All pain measures were intercorrelated (r's ≈ .3 - .6, p's < .01), and both anxiety and depression were associated with BPD symptoms and all pain measures (r's ≈ .2 - .6, p's < .01). Childhood neglect, in particular, was associated with all forms pain experiencing.

Table 3 presents the data on pain, trauma, and other clinical variables for the BPD+ and BPD − groups, and the results of ANCOVAs comparing the two groups, controlling for age and gender. These analyses revealed that the BPD+ group reported higher continuous and affective pain (MPQ) and polysomatic complaints associated with central sensitization (CSIA) than the BPD− group, but the groups did not differ on pain severity (NPRS-11), intermittent or neuropathic subtypes of pain (MPQ), or widespread pain (MBM). The BPD+ group also displayed higher anxiety and depression (HADS) than the BPD− group. All types of childhood traumas, but only adult physical/sexual abuse, were elevated in the BPD+ compared to BPD− group. All of these group differences were moderate or large in magnitude.

The Role of Negative Affect in the Associations of BPD Symptoms with Trauma and Pain-Related Measures

In ANCOVAs controlling for HADS anxiety and depression subscales (as well as age and gender), BPD+ patients still showed significant elevations in all forms of childhood trauma and adult physical/sexual trauma (p's < .002) compared to BPD− patients, but BPD group was no longer associated with affective pain ($p = .87$) or polysomatic complaints associated with central sensitization ($p = .17$). Path analysis confirmed that anxiety and depression explained the relations of BPD symptoms with affective pain (Indirect effects: $b_{ANX} = 0.88$, 95% CI [0.35, 1.41]; $b_{DEP} = 0.90$, 95% CI [0.50, 1.41]; Direct effect: $b_{BPD} = −.08$, 95% CI [−0.95, 0.79]) and central sensitization (Indirect effects: $b_{ANX} = 6.00$, 95% CI [2.32, 10.30]; $b_{DEP} = 7.83$, 95% CI [9.92, 18.34]; Direct effect: $b_{BPD} = 4.24$, 95% Ci [−1.78, 10.26]).

Interaction Between BPD Symptoms and Trauma in Predicting Pain

In the moderated regression models, there were no significant interactions between BPD symptoms and childhood trauma in predicting any pain outcome. BPD symptoms also did not interact with adult trauma to predict any pain outcome except widespread pain (b = −1.00, p = .03). Simple effects analysis revealed a positive association between adult trauma and widespread pain in patients who screened negative for BPD (B = .24, p = .01) but no association in patients who screened positive for BPD B = −.13, p = .40). Main effects were somewhat consistent with the pattern of results reported in Table 3, such that BPD symptoms were associated with affective pain (b = 1.58, p = .001) and central sensitization (b = 16.44, p < .001) in the context of adult trauma as a co-predictor, though these effects were attenuated in the context of childhood trauma (MPQ_{AFF}: b = 0.54, p = .38; CSIA: b = 11.41, p = .006). The magnitude of the BPD effect became trivial (B = .09) in predicting affective pain, but remained moderate (B = .26) in predicting central sensitization. Childhood trauma was significantly associated with both of these outcomes (MPQ_{AFF}: b = 0.02, p = .003; CSIA: b = 0.28, p < .001), as well as intermittent pain (b = 0.02, p = .001).

Discussion

We sought to replicate findings of BPD being associated with greater pain and clinical severity among patients with chronic pain, as well as to explore the role of child and adult trauma in BPD and pain co-occurrence. Although we found BPD symptoms to be associated with significantly greater clinical distress, findings among pain measures were mixed and in general suggested that BPD was associated only with affective pain and polysomatic complaints associated with central sensitization, but not with pain severity, widespread pain, or number of pain diagnoses. Notably, anxiety and depression accounted for the links between BPD symptoms and both affective pain and central sensitization. Patients meeting provisional BPD criteria were more likely to meet epidemiological criteria for fibromyalgia, which is the prototypical central sensitization disorder.

In addition, childhood trauma of all types appeared as a substantial correlate of BPD symptoms in our chronic pain sample, whereas of adult trauma exposures, only physical/sexual trauma was associated with BPD symptoms. Furthermore, BPD symptoms generally did not interact with trauma load to predict pain outcomes, except in the case of adult trauma predicting widespread pain, in which this association existed only in the absence of elevated BPD symptoms. Together, these results elucidate a picture of the complex interrelations of BPD features with various components of the pain experience and further highlight potential links between BPD, trauma, and pain.

The Multifaceted Relationship Between BPD and Pain

Almost one fifth of the patients in our chronic pain sample (17.7%) screened positive for likely BPD. This proportion is similar to past research (62) and may even underestimate the true number of pain patients who would meet criteria for BPD through formal assessment (45). Regardless, this prevalence emphasizes the importance of assessing for BPD among patients with chronic pain disorders, given the associations between BPD symptoms and various aspects of pain experiencing and general distress.

Chronic pain patients with elevated BPD symptoms reported greater affective pain and central sensitization. The affective pain subscale of the MPQ captures emotionally-tinged aspect of pain (e.g., fear, punishment, exhaustion) that are understandably elevated in BPD, given research suggesting individuals with BPD are prone to various forms of psychological pain, especially experiences of rejection or betrayal (63,64). The association between BPD symptoms and polysomatic complaints associated with central sensitization marks one of the first empirical findings supporting the theoretical link between BPD and chronic hypersensitivity of the central nervous system (36), which may help explain the established link between BPD and chronic pain (7) and reduced remission rates of chronic pain among pain patients with BPD symptoms (20). Furthermore, our findings provide some support for the pain paradox of BPD (39-40), in that BPD symptoms were associated with central sensitization, which produces vulnerability to chronic pain, but were not associated with acute pain in the past 24 hours (NPRS) or other forms of pain intensity (MPQ) besides affective pain. However, we were unable to directly assess the pain paradox without experimental manipulation of acute pain or a full assessment of self-harm history among these patients (40). Such research is important, given findings that BPD patients respond differently to physical pain induction (76-78), potentially due to the emotion and stress regulatory function of self-harm in this disorder (79).

We also found that fibromyalgia was more than twice as common among patients with elevated BPD symptoms than those without. Though the absolute prevalence of fibromyalgia depended on the diagnostic method (it was roughly twice as prevalent when assessed using self-reported epidemiological criteria than as indicated in medical records), this finding supports theoretical claims that individuals with BPD may be at particular risk for fibromyalgia (15,36), a disorder characterized by central sensitization.

Consistent with a growing body of literature exploring the paths by which BPD contributes to pain experiencing, the links between BPD and both affective pain and central sensitization were explained by a combination of depression and anxiety (17,19,21,22). This finding can be understood in multiple ways. First, it is possible that negative affect is a causal mechanism or mediator by which BPD contributes to pain. Individuals with BPD experience chaotic relationships, self-harm, and other detrimental experiences and behaviors that may give rise to chronic negative affect, which in turn puts these individuals at risk for heightened pain. Second, consistent with other research (64), BPD symptoms were strongly associated with both depression and anxiety in our study (Table 1). Thus, it is possible that the heightened and dysregulated negative affect that is endemic to BPD itself is the key feature of this disorder most associated with how individuals with BPD experience chronic pain and pain sensitivity. Third, negative affect may be a broadband, transdiagnostic contributor to chronic pain that is non-specific to BPD. Fourth, BPD may increase chronic pain risk directly, which in turn produces elevated negative affect. Fifth, pain and negative affect may be linked in a positive feedback loop among patients with BPD. Given the cross-sectional, survey-based nature of our study and most existing research on this topic, future research is needed to better understand the role of negative affect in BPD and pain experiencing.

Our finding that BPD symptoms were not associated with increased pain severity (assessed by the NPRS-11) is in contrast with that of Tragesser et al. (17) and Sansone et al. (16), who found significant correlations between BPD symptoms and pain severity among pain clinic and internal medicine patients, respectively. This discrepancy may be caused by differences in the timespan in which pain was assessed across these studies. We assessed pain severity within the past 24 hours, whereas Tragesser and colleagues assessed for the past month, and Sansone and colleagues assessed both current pain and pain in the past year. Notably, Sansone and colleagues found a non-significant correlation between the MSI-BPD and current pain, but a significant correlation with 12-month pain. Similarly, Dixon-Gordon et al. (38) found that BPD features (in an undergraduate sample) did not predict acute pain severity reported in daily life. On the other hand, Carpenter and colleagues (13) found higher momentary pain (i.e., assessed using ambulatory assessment) among individuals with BPD compared to a non-chronic pain comparison sample, though the absence of a chronic pain sample in this study makes comparisons to our results more challenging.

The Role of Trauma

As expected, childhood trauma was elevated among patients with elevated BPD symptoms. All forms of childhood trauma showed moderate-to-large associations with BPD symptoms, consistent with evidence that childhood trauma is a significant risk factor for BPD (27,28,30). Furthermore, accounting for childhood trauma in our interaction models reduced the strength of associations between BPD and pain outcomes, while childhood trauma remained a significant predictor of affective (and intermittent) pain experiencing and central sensitization, suggesting that childhood trauma may play a vital role in the symptomatic manifestation of pain disorders regardless of the presence of BPD. Interestingly, childhood trauma did not interact with BPD in predicting pain, suggesting that the presence of BPD does not appear to increase the risk for pain associated with childhood trauma, and vice versa. These risk factors may instead confer risk for pain, and in particular central sensitization, independently and both warrant attention in research and clinical care of patients with chronic pain and central sensitization disorders.

Notably, adult trauma did not show the same robust association with BPD, in that only adult physical/sexual, but not general or crime-related trauma was linked to BPD symptoms. Furthermore, after accounting for BPD, adult trauma was not associated with any pain outcomes. This discrepancy between child and adult traumas may suggest that childhood trauma, in particular, may produce vulnerability to the joint experience of BPD and chronic pain later in life, and only the experience of physical and/or sexual trauma in adulthood may confer increased risk for the development of BPD for those with chronic pain conditions. It is also possible that traumas that are interpersonal in nature and/or experienced vis-à-vis someone close to the victim (which largely characterizes the trauma types significantly associated with BPD) are particularly likely to confer risk for a co-occurring BPD and chronic pain disorder, though again it is notable that adult trauma did not predict increased pain symptoms in the context of BPD. Unfortunately, we were not able to more precisely discriminate between adult physical and sexual traumas in their link to BPD symptoms, as these were assessed in tandem in the THQ. Together, these findings point to the complex interrelations among trauma and BPD in patients with chronic pain and indicate the

importance of assessing both in order to more completely understand the various facets of pain experienced by these patients.

Clinical Practice Implications

Our findings may inform clinical care in three key ways. First, given both the prevalence of positive BPD screens in our sample and the increased clinical severity of these patients, assessment for BPD among chronic pain samples is warranted. Several methods are available to the clinician in order to provide at least preliminary identification of BPD among chronic pain patients, such as brief and psychometrically-sound screening tools like the MSI-BPD (45). A two-stage assessment process may also be beneficial, in which patients who screen positive for BPD are further assessed using more comprehensive interviews (e.g., the Structured Clinical Interview for DSM-5 PDs) (65), as such a two-step process balances diagnostic accuracy and resource utilization (66,67).

Second, the finding that both affective pain and polysomatic complaints associated with central sensitization were associated with anxiety and depression, which are elevated in BPD, suggests that the pain experienced in patients with co-occurring chronic pain and BPD may be directly linked to patients' psychological state (17). These patients may especially benefit from interventions that account for psychological aspects of pain experiencing, such as through increased exploration of the meaning and interpretation of pain among these patients (e.g., pain may be interpreted as punishing or exhausting among these patients). Also, several treatments for BPD suggest the importance of directly targeting maladaptive interpretative biases and the belief that one is irrevocably vulnerable or incapable of tolerating distress or pain (68,69). Treatments like these may be especially beneficial for patients who have comorbid BPD and chronic pain, in whom unresolved psychological trauma and the need for emotion regulation is common (32,70). When BPD is present, referring patients to concurrent treatment particularly focused on emotion regulation may increase the likelihood that these patients remit through pain treatment.

Third, taking a thorough history that includes possible experiences of childhood trauma and neglect may provide valuable insight into the pain experience. Although adult physical trauma, such as accidents and injuries, is commonly assessed prior to chronic pain treatment, it is less common for childhood trauma to be assessed. Accounting for childhood trauma may improve pain interventions and provide a more thorough understanding of the range of symptoms experienced by patients with chronic pain. Some preliminary evidence suggests that trauma-focused psychotherapies, including Emotional Awareness and Expression Therapy (EAET) (71,72) and Eye Movement Desensitization and Reprocessing (73-75) can be helpful for patients with comorbid chronic pain and trauma.

Limitations and Future Directions

Our study was limited by reliance on self-reported screening for BPD; replicating this study using structured clinical interviews to diagnosis BPD would increase confidence in the findings. Nevertheless, our results may generalize to other situations in which only BPD screening is available, such as in the presence of significant time constraints common to medical settings. We also relied on cross-sectional data, thus limiting causal claims that

could be made regarding risk for BPD or pain conditions. Longitudinal exploration of proximal and distal risk factors for the development of comorbid chronic pain and BPD is important. Furthermore, incorporating a group of patients with BPD and no comorbid chronic pain disorder would have enhanced our ability to examine which variables are associated with each condition in isolation versus their co-occurrence. Additionally, information regarding psychotropic medication and opioid use was not available in this study and thus we could not account for the potential confounding influence of analgesic medications on reports of pain. Finally, clinical practice recommendations remain largely untested empirically, and research that examines the clinical utility of these suggestions is needed.

Conclusion

Our study provides one of the first explorations of the interplay between child and adult trauma, borderline personality disorder symptoms, and various manifestations of pain among patients with chronic pain disorders. BPD symptoms appear to be meaningfully associated not only with increased clinical severity among patients with chronic pain, but also with a unique manifestation of certain aspects of pain, particularly affective pain, polysomatic complaints associated with central sensitization, and fibromyalgia. Childhood trauma may be especially important in the co-occurrence of BPD and chronic pain conditions. We suggest that researchers and clinicians assess for BPD in chronic pain patients, both to enhance conceptual models of the transaction between symptoms of these disorders and to improve clinical care and treatment outcomes.

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Appendix

Clinical Diagnoses Across the Entire Sample

Pain Diagnoses	**Total Sample (*N* = 202)**	
	n	**%**
Diffuse Musculoskeletal Pain	55	27.2
Myofascial pain syndrome	2	1.0
Fibromyalgia	42	20.8
Complex regional pain syndrome-I/II	5	2.5
EDS/hypermobility syndrome	8	4.0
Spasticity	1	0.5
Back Pain	81	40.1
Low back pain	38	18.8

Pain Diagnoses	Total Sample (*N* = 202)	
	n	**%**
Chronic back pain	12	5.9
Thoracic back pain	6	3.0
Herniated disc	2	1.0
Dorsalgia	1	0.5
Scoliosis	4	2.0
Spondylosis	4	2.0
Thoracic outlet syndrome	1	0.5
Failed back syndrome/post-laminectomy syndrome	2	1.0
Degenerative disc disease	12	5.9
Radiculopathy	7	3.5
Sacral joint pain	1	0.5
Spondylolisthesis	1	0.5
Stenosis	6	3.0
Osteoporosis	6	3.0
Neck Pain	20	9.9
Cervical dysplasia	1	0.5
Cervical radiculopathy	2	1.0
Neck pain	17	8.4
Head Pain	60	29.7
Temporomandibular joint syndrome	2	1.0
Chronic headaches	11	5.4
Chronic migraine	47	23.3
Facial pain	3	1.5
Trigeminal neuralgia	1	0.5
Tinnitus	1	0.5
Extremity Pain	68	33.7
Arthritis	10	5.0
Osteoarthritis	19	9.4
Joint pain	4	2.0
Shoulder pain	12	5.9
Hand pain	2	1.0
Carpal tunnel	5	2.5
Ankle/foot pain	11	5.4
Plantar fasciitis	1	0.5
Hip pain	14	6.9
Bursitis	2	1.0
Knee pain	22	10.9
Chest pain	9	4.5
Wrist pain	3	1.5
Elbow pain	3	1.5
Arm pain	1	0.5

Pain Diagnoses	Total Sample (*N* = 202)	
	n	**%**
Neuropathic Pain	13	6.4
Peripheral neuropathy	4	2.0
Polyneuropathy	4	2.0
Diabetic neuropathy	2	1.0
Diabetic lumbosacral plexopathy	1	0.5
Ulnar neuropathy	1	0.5
Transverse myelitis	1	0.5
Abdominal Pain	45	22.3
Abdominal pain	14	6.9
Gastroparesis	1	0.5
Gastritis	1	0.5
Crohn's disease	3	1.5
Ulcerative colitis	5	2.5
Sclerosing cholangitis	1	0.5
Irritable bowel syndrome	17	8.4
Diverticulosis/diverticulitis	2	1.0
Colonic disorder/colonic inertia	1	0.5
Dysphagia	4	2.0
Pelvic Pain	49	24.3
Dysmenorrhea	5	2.5
Menorrhagia	1	0.5
Endometriosis	21	10.4
Polycystic ovary syndrome	4	2.0
Pelvic pain	8	4.0
Interstitial cystitis/bladder pain syndrome	19	9.4
Chronic UTI	1	0.5
Chronic cystitis	1	0.5
Vaginal pain	1	0.5
Prostatitis	2	1.0
Testicular pain	1	0.5
Chronic Fatigue-Related Pain	12	5.9
Postural orthostatic tachycardia syndrome	5	2.5
Chronic fatigue syndrome	8	4.0
Autoimmune Inflammatory Pain	22	10.9
Systemic lupus erythematosus	2	1.0
Lyme disease	2	1.0
Rheumatoid arthritis	6	3.0
Ankylosing spondylosis	2	1.0
Sjögren's syndrome	1	0.5
Inflammatory polyarthropathy	1	0.5
Polymyalgia rheumatica	1	0.5

Pain Diagnoses	Total Sample (*N* = 202)	
	n	**%**
Raynaud's syndrome	3	1.5
Multiple sclerosis	1	0.5
Idiopathic proliferative fibrosing mediastinitis	1	0.5
Bechet's disease	1	0.5
Erdheim-Chester disease	1	0.5
Retroperitoneal fibrosis	1	0.5
Cancer pain (stage IV pancreatic)	1	0.5
Metabolic Pain	4	2.0
Chronic kidney disease (stage III/IV)	3	1.5
Mitochondrial myopathy	1	0.5
Chronic Pain Not Otherwise Specified	13	6.4
Chronic pain	3	1.5
Chronic pain, unspecified	1	0.5
Chronic pain syndrome	9	4.5
	M	**SD**
Number of pain diagnoses	2.69	1.84
	n	*%*
Comorbid Psychiatric Conditions	94	46.5
Anxiety disorder	36	17.8
Depressive disorder	52	25.7
Mixed anxiety/depressive disorder	15	7.4
Posttraumatic stress disorder	11	5.4
Adjustment disorder	1	0.5

Note. Psychiatric conditions are not comprehensive as they were not formally assessed and represent only those psychiatric conditions comorbid to pain that were considered clinically relevant with regards to treatment planning and that were indicated in medical records. *n* = frequency of patients with clinical diagnosis; *%* = percentage of subgroup; *M* = mean; *SD* = standard deviation.

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Highlights

- BPD symptoms are linked with affective pain in patients with chronic pain
- BPD symptoms are linked to central sensitization and fibromyalgia in chronic pain
- Negative affect mediates the link between BPD symptoms and pain experiencing
- Childhood trauma is associated with risk for BPD/chronic pain co-occurrence
- Assessing for BPD among patients with chronic pain is clinically recommended

Table 1

Demographic characteristics and pain diagnoses by BPD screen on the MSI-BPD

Group	**Total Sample (*N* = 181)**		**BPD+ (*n* = 32)**		**BPD− (*n* = 149)**				
Demographics	**M**	**SD**	**M**	**SD**	**M**	**SD**	**t**	**P**	***d*[95% CI]**
Age	44.62	14.06	36.56	10.75	46.37	14.12	**4.40**	**<.001**	**0.78 [0.41, 1.15]**
	n	**%**	**n**	**%**	**n**	**%**	**χ^2**	**P**	***OR* [95% CI]**
Gender							5.60	.02	5.60 [1.06, 61.67]
Women	145	80.1	30	93.8	115	77.2			
Men	32	17.7	1	3.1	31	20.8			
Unknown	4	2.2	1	3.1	3	2.0			
Race							0.86	.46	0.86 [0.22, 2.13]
African American, Black	13	7.2	2	6.3	11	7.4			
Asian/Pacific Islander	4	2.2	0	0.0	4	2.7			
Hispanic, Latinx	5	2.8	0	0.0	5	3.4			
Native American/American Indian	1	0.6	0	0.0	1	0.7			
White	148	81.8	28	87.5	120	80.5			
Multiracial	5	2.8	2	6.3	3	2.0			
Other	4	2.2	0	0.0	4	2.7			
Unknown	1	0.6	0	0.0	1	0.7			
Relationship Status							5.11	.03	2.47 [1.11, 5.48]
Single	51	28.2	12	37.5	39	26.2			
Married/Domestic Partnership	95	52.5	11	34.4	84	56.4			
Divorced	31	17.1	8	25.0	23	15.4			
Separated	3	1.7	1	3.1	2	1.3			
Widowed	1	0.6	0	0.0	1	0.7			
Educational Attainment							4.50	.05	2.36 [1.05, 5.27]
High school diploma or equivalent	13	7.2	4	12.5	9	6.0			
Vocational or technical school	7	3.9	2	6.3	5	3.4			
Some college	33	18.2	8	25.0	25	16.8			
Bachelor’s degree	65	35.9	10	31.3	55	36.9			
Master’s degree	42	23.2	5	15.6	37	24.8			

Group	Total Sample (*N* = 181)		BPD+ (*n* = 32)		BPD− (*n* = 149)				
Demographics	**M**	**SD**	**M**	**SD**	**M**	**SD**	**t**	**P**	***d*[95% CI]**
Doctorate or professional degree	14	7.7	1	3.1	13	8.7			
Other	6	3.3	2	6.3	4	2.7			
Unknown	1	0.6	0	0.0	1	0.7			
Employment Status							3.14	.11	2.09 [0.91, 4.77]
Full-time	96	53.0	15	46.9	81	54.4			
Part-time	16	8.8	3	9.4	13	8.7			
Self-employed	9	5.0	2	6.3	7	4.7			
Unemployed	9	5.0	2	6.3	7	4.7			
Retired	18	9.9	0	0.0	18	12.1			
Unable to work	32	17.7	10	31.3	22	14.8			
Unknown	1	0.6	0	0.0	1	0.7			
Income							3.40	.09	2.13 [0.94, 4.80
Under $10,000	8	4.4	1	3.1	7	4.7			
$10,000-19,999	13	7.7	5	15.6	9	6.0			
$20,000-$50,000	42	23.2	10	31.3	32	21.5			
$50,000-$100,000	50	27.6	7	21.9	43	28.9			
$100,000-$150,000	31	17.1	3	9.4	28	18.8			
$150,000 or higher	15	8.3	3	9.4	12	8.1			
Unknown	21	11.6	3	9.4	18	12.1			
Disability Status							0.37	.65	1.31 [0.55, 3.11]
Currently receiving disability	29	16.0	5	15.6	24	16.1			
In the process of applying for disability	14	7.7	5	15.6	9	6.0			
Not receiving or applying for disability	101	55.8	19	59.4	82	55.0			
Unknown	37	20.4	3	9.4	34	22.8			
Pain Diagnoses From Medical Records									
Back pain	73	40.3	13	40.6	60	40.3	0.01	1.00	0.96 [0.44, 2.09]
Neck pain	17	9.4	2	6.3	15	10.1	0.52	.74	0.86 [0.23, 3.16]
Head pain	50	27.6	11	34.4	39	26.2	0.68	.40	1.41 [0.62, 3.19]
Extremity pain	58	32.0	5	15.6	53	35.6	5.32	.02	0.32 [0.12, 0.88]
Diffuse musculoskeletal pain	50	27.6	15	46.9	35	23.5	6.56	.02	2.75 [1.24, 6.07]

Group	Total Sample (*N* = 181)		BPD+ (*n* = 32)		BPD− (*n* = 149)				
Demographics	**M**	**SD**	**M**	**SD**	**M**	**SD**	**t**	**P**	***d*[95% CI]**
Fibromyalgia	38	21.0	12	37.5	26	17.4	5.85	.03	2.72 [1.18, 6.26]
Neuropathic pain	13	7.2	3	9.4	10	6.7	0.23	.71	1.39 [0.36, 5.35]
Abdominal pain	40	22.1	11	34.4	29	19.5	3.02	.10	2.08 [0.90, 4.79]
Pelvic pain	46	25.4	12	37.5	34	22.8	2.62	.12	1.94 [0.86, 4.37]
Chronic fatigue-related pain	10	5.5	2	6.3	8	5.4	0.02	1.00	1.13 [0.23, 5.61]
Autoimmune/inflammatory pain	18	9.9	1	3.1	16	11.4	1.91	.32	0.24 [0.03, 1.90]
Cancer pain (stage IV pancreatic)	1	0.6	0	0.0	1	0.7	0.22	1.00	--
Metabolic pain	4	2.2	0	0.0	4	2.7	0.91	1.00	--
Chronic pain, not otherwise specified	10	5.5	0	0.0	10	6.7	2.36	.21	
Comorbid Psychiatric Conditions									
Anxiety disorder	33	18.2	9	28.1	24	16.1	2.26	.14	2.04 [0.84, 4.94]
Depressive disorder	46	25.4	11	34.4	35	23.5	1.38	.27	1.71 [0.75, 3.88]
Mixed anxiety/depressive disorder	13	7.2	5	15.6	8	5.4	3.88	.06	3.26 [0.99, 10.74]
Posttraumatic stress disorder	8	4.4	0	0.0	8	5.4	1.86	.35	--
Adjustment disorder	1	0.6	0	0.0	1	0.7	0.22	1.00	--
	Med	**Range**	**Med**	**Range**	**Med**	**Range**	**Z**	**P**	**r**
Number of pain diagnoses	2.0	1-12	2.0	1-6	2.0	1-12	−0.39	.70	.002

Note. Statistical tests evaluate differences in distributions of each variable by BPD risk group. Significant tests at $p < .006$ (.05/8 alpha correction) for demographic variables, $p < .003$ (.05/15 alpha correction) for pain diagnoses, and $p < .01$ (.05/5 alpha correction) for psychiatric conditions are denoted by **bold** font. Chi-square conducted on collapsed demographic categories, resulting in more robust 2x2 comparisons, as follows: gender = women vs. men, race = non-white vs. white, relationship status = not married/partnered vs. married/partnered, education = no college degree vs. college degree, employment status = unemployed vs. employed, income = income <$50K vs. income ≥$50K, and disability status = receiving or applying for disability vs. not. Chi-square analyses exclude "Unknown" group participants. Percentages across pain diagnosis categories sum to greater than 100% as patients could be diagnosed with multiple pain disorders. Fibromyalgia diagnoses are a subset of diffuse musculoskeletal pain disorders. Comorbid psychiatric conditions are not comprehensive as they were not formally assessed and represent only those psychiatric conditions comorbid to pain that were considered clinically relevant with regards to treatment planning and that were indicated in medical records. BPD = borderline personality disorder; MSI-BPD = McLean Screening Instrument for BPD; BPD+ = screened positive for BPD; BPD− = screened negative for BPD; *M* = mean; *SD* = standard deviation; *t* = *t*-statistic; *p* = *p* value; *d* = Cohen's *d* effect size metric; 95% CI = 95% confidence interval; χ^2 = chi-square statistic; *n* = frequency of demographic characteristic; % = percentage of subgroup; *OR* = odds ratio; *Med* = median; *Z* = Mann-Whitney *U*-test *Z*-statistic; *r* = *r* effect size metric ($z/\sqrt{N}$).

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Table 2

Descriptive statistics of and correlations among the BPD, pain, emotional distress, and trauma variables.

	%		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. BPD (MSI-BPD)	17.7%																
Pain	*Mean*	*SD*															
2. Pain Severity (NPRS-11)	4.57	1.95	.03														
3. Widespread Pain (MBM)	9.22	7.09	.14	.32***													
4. Continuous Pain (MPQ)	3.98	2.10	.21**	.70***	.47***												
5. Intermittent Pain (MPQ)	3.02	2.31	.04	.62	.22**	.57***											
6. Neuropathic Pain (MPQ)	2.36	1.88	.14	.52***	.46***	.58***	.53***										
7. Affective Pain (MPQ)	3.15	2.23	.29***	.55***	.37***	.69***	.58***	.56***									
8. Central Sensitization (CSIA)	47.14	17.29	.41***	.40***	.52***	.59***	.35***	.50***	.63***								
Emotional Distress																	
9. Anxiety (HADS-A)	9.32	4.72	.50***	.25**	.22**	.41***	.30***	.32***	.55***	.61***							
10. Depression (HADS-D)	6.88	4.42	.31***	.33***	.26	.42***	.23**	.34***	.54***	.60***	.65***						
Trauma																	
11. Childhood Sexual Abuse (CATS)	1.66	2.78	.31***	.09	.10	.19*	.21**	.24***	.23**	.32***	.19*	.14					
12. Childhood Punishment (CATS)	9.20	4.74	.20***	.16*	.13	.30***	.24**	.20**	.28***	.44***	.29***	.36***	.37***				
13. Childhood Neglect (CATS)	17.69	13.75	.42***	.19*	.23**	.30***	.26***	.31***	.32***	.50***	.33***	.25***	.46***	.62***			
14. Adult General Disaster/ Traumatic Experience (THQ-Adult)	2.37	2.07	−.09	.16*	.06	.08	.12	.04	.05	.04	−.05	.16*	.24**	.09	.05		
15. Adult Crime-Related Trauma (THQ-Adult)	0.47	0.73	.07	.17*	.18*	.20**	.11	.09	.10	.14	.06	.13	.13	.15*	.10	.08	
16. Adult Physical/Sexual Trauma (THQ-Adult)	0.35	0.66	.34***	.01	.12	.23**	.16*	.15	.13	.18*	.16*	.13	.28***	.26***	.19*	.17*	.30***

Note. Pearson product-moment correlations, two-tailed tests. BPD = borderline personality disorder; MSI-BPD = McLean Screening Instrument for BPD (dichotomized at a cutoff of 7 or more criteria); NPRS-11 = Numerical Pain Rating Scale-11; MBM = Michigan Body Map; MPQ = McGill Pain Questionnaire; CSIA = Central Sensitization Inventory—Part A; HADS = Hospital Anxiety and Depression Scale; CATS = Child Abuse and Trauma Scale; THQ-Adult = revised Trauma History Questionnaire after removing childhood trauma exposure.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 3

Pain, emotional distress, and trauma by BPD screening status

Group	BPD+ (*n* = 32)		BPD− (*n* = 149)				
Pain	**M**	**SD**	**M**	**SD**	**F**	**P**	**η^2**
Pain Severity (NPRS-11)	4.68	1.72	4.55	2.01	0.85	.36	.005
Widespread Pain (MBM)	11.38	6.42	8.75	7.15	2.61	.11	.01
Continuous Pain (MPQ)	4.96	1.82	3.78	2.10	8.29	.005	.05
Intermittent Pain (MPQ)	3.25	2.07	2.97	2.36	0.68	.41	.004
Neuropathic Pain (MPQ)	2.92	1.86	2.24	1.86	3.38	.07	.02
Affective Pain (MPQ)	4.63	2.43	2.87	2.08	**12.76**	**<.001**	**.07**
Central Sensitization (CSIA)	62.55	11.97	43.83	16.48	**27.26**	**<.001**	.15
Emotional Distress							
Anxiety (HADS-A)	14.39	3.43	8.23	4.23	**48.00**	**<.001**	**.22**
Depression (HADS-D)	9.80	3.22	6.25	4.40	**24.75**	**<.001**	**.13**
Trauma							
Childhood Sexual Abuse (CATS)	3.40	3.66	1.29	2.42	**16.80**	**<.001**	**.09**
Childhood Punishment (CATS)	12.47	5.17	8.49	4.35	**23.44**	**<.001**	**.12**
Childhood Neglect (CATS)	30.20	11.43	15.07	12.74	**33.55**	**<.001**	**.16**
Adult General Disaster/Traumatic Experience (THQ-Adult)	1.97	1.82	2.46	2.11	0.24	.63	.001
Adult Crime-Related Trauma (THQ-Adult)	0.58	0.89	0.45	0.70	2.62	.11	.02
Adult Physical/Sexual Trauma (THQ-Adult)	0.84	0.90	0.25	0.55	**24.33**	**<.001**	**.13**

Note. All group comparisons conducted using univariate analysis of covariance, with age and gender as covariates. Descriptive statistics are raw, not covariate adjusted, for interpretability. Significant tests at $p < .003$ (.05/15 alpha correction) are denoted by **bold** font. BPD+ = screened positive for BPD; BPD− = screened negative for BPD; *M* = mean; *SD* = standard deviation; *F* = *F*-statistic; *p* = *p* value; η^2 = partial eta-squared effect size metric (small = .01, medium = .06, large = .14); NPRS = Numeric Pain Rating Scale; MBM = Michigan Body Map; MPQ = McGill Pain Questionnaire; CSIA = Central Sensitization Inventory—Part A; HADS = Hospital Anxiety and Depression Scale; CATS = Child Abuse and Trauma Scale; THQ-Adult = revised Trauma History Questionnaire after removing childhood trauma exposure.