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The relationship between catastrophizing and altered pain sensitivity in patients with chronic low back pain

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Abstract

Changes in central pain processing have been shown in patients with chronic low back pain (cLBP). We used quantitative sensory testing (QST) methods to identify differences in pain sensitization between patients with cLBP (N=167) and healthy controls (N=33). Results indicated that, compared to healthy pain-free controls, cLBP patients showed increased sensitivity and greater painful aftersensations for mechanical pressure and pin prick stimuli and lower tactile spatial acuity in the two-point discrimination task ($ps<.05$). Then, we examined the role of pain catastrophizing as a mediator of the group differences in pain sensitization. We found that catastrophizing partially accounted for group differences in pressure required to produce moderate pain. Finally, we examined the relationship between pain sensitization, catastrophizing, and clinical pain among patients with cLBP. We found that catastrophizing and deep-tissue pressure pain were associated with greater pain intensity in the past month, week, and at the visit as well aslow back pain bothersomeness. Further, deep-tissue pressure pain mediated the associations between catastrophizing and both pain in the past month and low back pain severity. Taken together, these results indicate that not only do patients with cLBP demonstrate increased pain sensitization and decreased sensitivity to innocuous stimuli, but these changes are also linked with increased catastrophizing. Furthermore, both catastrophizing and sensitization are associated with increased clinical pain among cLBP patients.

Summary

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cLBP patients demonstrate greater pain sensitization and poorer spatial acuity, changes linked with greater catastrophizing. Catastrophizing and sensitization are also associated with greater clinical pain.

INTRODUCTION

Chronic low back pain (cLBP) affects 15–30% of the U.S. population [14] and is the leading cause of disability worldwide [20]. Unfortunately, treatment options for cLBP often yield only limited relief. However, the development of more efficacious, better-targeted treatments for cLBP requires a better understanding of the mechanisms that contribute to the onset, maintenance, and impact of this chronic condition [14].

One proposed mechanism that may contribute to the development and maintenance of cLBP is pain sensitization [2, 11]. Pain sensitization involves increased responsiveness of central and/or peripheral nervous system circuits, resulting in pain hypersensitivity (e.g., allodynia, hyperalgesia) [27, 62, 67] and potentially poor pain-related outcomes (see Figure 1) [11].To date, however, there is equivocal evidence for sensitization among patients with cLBP [59]. Some studies have found widespread pain sensitization among patients with cLBP across various pain modalities [10, 22, 23, 37] while other studies show minimal sensory differences between cLBP patients and healthy controls [34, 36, 45; 52, 53]. Other studies report mixed findings with cLBP patients demonstrating pain sensitivity for some modalities, but not others [3, 15, 38, 49]. Given the considerable individual differences in pain sensitization even within this disease state, understanding the factors contributing to sensitization and the relationship between pain sensitization and pain-related outcomes is warranted.

A related mechanistic contributor to the experience of cLBP is pain catastrophizing. Pain catastrophizing, a pattern of negative cognitive-emotional responses to pain that includes rumination, magnification, and helplessness [63], has been shown to be associated with pain severity, disability, and poor outcomes for patients with cLBP [26, 46, 48]. It has also been shown to predict the development of chronic pain in previously pain-free individuals, and the chronification of acute back pain [60, 68]. Pain sensitization and pain catastrophizing may also be inter-related. For example, some prior studies suggest that catastrophizing is a contributor to pain sensitivity through aberrant central nervous system processing of pain-related information [24, 49]. However, as noted by Curatolo & Arendt-Nielsen, the role of psychosocial factors, such as catastrophizing, in the development of pain sensitization remains unclear with additional research needed to identify such mechanisms [11].

Collectively, there is mixed evidence supporting pain sensitization as a pathophysiological mechanism of cLBP. While, the role of catastrophizing as a psychosocial factor contributing to the development of chronic pain and poor pain-related outcomes has been documented, the role of catastrophizing as a mechanism of pain sensitization as well as the effect of pain sensitization on pain-related outcomes among cLBP patients remain unclear. In the current study, we use a large sample and formal mediation analyses to better understand the relationships between pain sensitization, catastrophizing, and pain outcomes in cLBP. We first aimed to identify differences in pain sensitivity between patients with cLBP and

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healthy, pain-free controls using quantitative sensory testing (QST) procedures [2, 11]. Then, we examined the role of pain catastrophizing as a mediator of the group differences in pain sensitization. Finally, we examined the relationship between pain sensitization, catastrophizing, and clinical pain among patients with cLBP.

METHODS

Participants and Design

The current study uses only the baseline behavioral and clinical data collected between 2013–2017 from a large single-site neuroimaging-based longitudinal treatment study of acupuncture in patients with cLBP (P01-AT006663; clinicaltrials.gov/ct/show/NCT01614639). Upon arrival at the laboratory, participants provided informed consent. They then completed a series of baseline questionnaires assessing their demographic information, pain, depression, and catastrophizing. Participants subsequently underwent QST as described below. Upon completion of the study, participants were debriefed and compensated. All procedures were approved by the Partners Healthcare Institutional Review Board.

Participants were 167 adults (age 18–60) with idiopathic cLBP and 33 healthy, pain-free controls. Participants were recruited via email, internet, and bulletin board advertisements in Boston, MA as well as through electronic medical records-based databases from Brigham and Women's and Massachusetts General Hospitals. All participants, both those with cLBP and healthy controls, were included if they were right handed, acupuncture-naïve, and were able to complete self-report measures of pain, psychosocial functioning, and medical history. Potential participants from either group (cLBP or healthy controls) were excluded if: (1) they had a history of cardiac, respiratory, or nervous system disease that may impact MRI, (2) acupuncture or MRI were contraindicated, (4) they had systemic diseases, (5) they had a history of head injury or coma, or (6) they had active substance abuse disorder within the past two years. Inclusion criteria for patients with cLBP required: (1) meeting Quebec Task Force Classifications System symptom categories I-II [i.e., unlikely to have significant nerve root involvement, stenosis, or mechanical instability (Abenhaim et al., 2000; Loisel et al., 2002)] diagnosed by a clinical evaluation including the use of X-ray/MRI reports when available, (2) low back pain duration greater than 6 months, (3) average low back pain intensity rating ≥ 4 on a 0–10 numeric rating scale (0: no pain, 10: most intense pain imaginable) over the two-week period prior to enrollment, and (5) the ability to temporarily exacerbate cLBP with calibrated physical maneuvers. Patients with cLBP were excluded if: (1) back pain manifested with one or more specific causes (e.g., cancer, fracture, infection), (2) radicular pain radiated below the knee, (3) back problems were complicated (e.g., medicolegal issues), (4) they had undergone prior back surgery, or (5) they were on daily high-dose opioids [> 60 milligrams morphine equivalents(MME)].

Measures

The Pain Catastrophizing Scale (PCS) [63] is a 13-item self-report measure of pain-related catastrophizing comprised of rumination, magnification, and helplessness [12, 51]. To complete this measure, participants were asked to identify how frequently they experience

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catastrophic cognitions in response to pain using a scale from 0 (not at all) to 4 (all the time). Item scores were then summed to determine an aggregate score of pain catastrophizing. The PCS has been shown to be valid and reliable among patients with chronic pain [66].

The Beck Depression Inventory-II (BDI-II) [4] is a 21-item self-report measure of depressive symptoms. Participants rated the severity with which they experienced depressive symptoms over the past two weeks on a scale from 0 (not present) to 3 (severe). Item scores were then summed to determine an aggregate score of depressive symptoms. The BDI-II has been validated among patients with chronic pain [25].

Low back pain bothersomeness was assessed only in the cLBP group with a single-item question asking, “How bothersome has your low back pain been during the past week?” Participants rated the bothersomeness of their back painon a 0–10 visual analog scale (VAS) ranging from “not at all bothersome” to “extremely bothersome”. This widely accepted assessment has been shown to be both valid and reliable [5, 8, 13]. Average pain intensity in the past week and month were assessed using an eleven-point numeric rating scale (NRS) ranging from 0 (no pain) to 10 (worst pain imaginable). Pain at the beginning of the visit was rated on a 0–100 NRS ranging from “no pain” to “worst pain imaginable”. The NRS has been validated for specificity and use in chronic pain research [21, 28]. Pain interference was assessed using the pain interference items from the Patient-Reported Outcomes Measurement Information System (PROMIS-29) [1]. Pain duration was also measured in years.

Quantitative Sensory Testing

Two-point Discrimination: Two-point discrimination thresholds are a measure of tactile acuity for non-painful mechanical sensation [31, 33]. Using a two-point aesthesiometer (Mitutoyo Digital Caliper, Mitutoyo), subjects completed a series of ascending and descending trials in which they indicated whether they felt “one distinct point” or “two points separated by a distance.” This task was performed at two body sites: the right side of the lower back (over the erector spinae muscles, medial to PSIS, level with the lumbar vertebrae) and the right index finger (middle phalanx, palmar surface). Participant eyes were closed throughout the testing session and finger testing followed back testing. The right index finger served as a “control” body site to the lower back (affected body site due to pain pathology). For stimulations following a descending order, the experimenter started with a large separation distance and decreased the distance every three stimulation trials (distance was decreased by 1cm for the lower back and 1mm for the index finger). After every three stimulation trials at a particular separation distance, the experimenter verbally asked the participant if they felt one or two points. The separation distance at which two points shifted to one point was repeated twice, and if subject reported the change consistently, the distance was recorded. The same procedure was repeated with the ascending order, but the starting separation distance was zero, and gradually increased from that distance. Ascending and descending order administration was counterbalanced across subjects. Results of the ascending and descending orders were then averaged to determine the two-point discrimination threshold for each body site.

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Deep-tissue Pressure Pain: Cuff pressure algometry (CPA) was used to determine responses to deep pressure pain [17, 32, 41, 55]. Using a Hokanson rapid cuff inflator, tonic, deep-tissue, mechanical stimulation was applied to the left gastrocnemius muscle using a standard blood pressure cuff. To begin, the cuff was inflated to 30 mmHG, a non-painful pressure for all participants which was verbally confirmed by the experimenter. Using a method of limits, the cuff pressure was increased at a rate of approximately 5 mmHg per second. The experimenter asked the participant to provide a verbal prompt when the stimulus first transitioned from being non-painful to painful. When the participant prompted the experimenter that the stimulus became painful, the experimenter increased cuff pressure in steps of 15 mmHg. At each 15mmHg increase, the participant provided a verbal rating of pain intensity and unpleasantness from 0 (no pain; not at all unpleasant) to 100 (worst pain imaginable; most unpleasant imaginable). When pain intensity reached a level of ~40/100, the experimenter recorded the pressure in mmHg (P40) and kept that pressure level constant for the remainder of the experiment. This P40 was the target pressure also used as a metric of pain sensitivity. At this steady P40 pressure, the participant provided verbal ratings of pain and unpleasantness every 30 seconds for 2 minutes. After 2 minutes, the cuff was deflated. Fifteen seconds following cuff deflation, participants provided verbal ratings of any painful aftersensations [61]. The mean ratings were calculated by averaging the 30, 60, 90, and 120 ratings separately for pain intensity and pain unpleasantness. Participants were unable to see the display on the Hokanson rapid cuff inflator device to minimize any anchoring effects in pain/unpleasantness ratings.

Mechanical Punctate Pain: Mechanical punctate pain was assessed using weighted pinprick stimulators [16, 17, 19]. Participants used a 0 (no pain) to 100 (worst pain imaginable) numeric rating scale (NRS) to rate the sensation of pain produced by 64mN, 128mN, and 256mN stimulators. The lowest-force stimulator that produced a painful sensation (128 or 256mN for most participants) was then used to apply a train of 10 stimuli to the skin on the dorsum of the right middle finger (middle phalanx) at a rate of 1 pinprick per second. Participants provided pain ratings for the first, fifth, and tenth stimulus. Fifteen seconds after the end of the stimulus train, participants rated painful aftersensations on the same 0–100 scale [18, 19]. To calculate temporal summation, the pain intensity rating after the first stimulus was subtracted from the rating after the tenth stimulus. The pain ratings from the initial three stimulators (64mN, 128mN, and 256MN) were averaged to determine mean mechanical punctate pain intensity.

Data Analysis

Differences between cLBP and HC Groups: We used independent samples t-tests and chi-square analyses to determine differences in demographic variables and pain variables between the cLBP and HC groups. We ran independent samples t-tests to identify differences between cLBP and HC groups on the following QST outcomes: two-point discrimination on the finger, two-point discrimination on the back, P40 pressure, mean deep-tissue cuff pressure pain intensity, mean deep-tissue cuff pressure pain unpleasantness, deep-tissue cuff pressure painful aftersensations, pain rating for 1st mechanical punctate stimulus, pain rating for 10th mechanical punctate stimulus, mean pain ratings for pinprick probes, temporal summation of mechanical punctate pain, and mechanical punctate painful

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aftersensations. We then examined the relationship between catastrophizing and QST outcomes in the entire sample using Pearson correlations. Finally, using the Process Macro for SPSS [56, 57], we conducted bias-corrected bootstrapped mediation analyses using 10,000 bootstrapped resamples to examine the role of catastrophizing as a potential mediator of the group differences in QST responses controlling for depression and opioid use [56, 57]. Bootstrapping is a nonparametric procedure that does not assume indirect effects are normally distributed. For these analyses, we only conducted mediation models for QST variables when (1) there were significant group differences in the QST response and (2) the QST response was significantly correlated with catastrophizing ($p<.05$). Estimates of indirect effects were considered significant in the case that zero was not included in the 95% confidence intervals [56, 57].

Associations between Catastrophizing, Pain Outcomes, and QST in cLBP Patients: To examine the associations between catastrophizing, pain outcomes, and QST variables, we first conducted Pearson correlations within the cLBP sample. We then conducted bias-corrected bootstrapped multiple mediation analyses using 10,000 bootstrapped resamples to examine the role of pain sensitization (as measured by QST) as mediators of the relationship between catastrophizing and clinical pain outcomes controlling for depression and opioid use. For these analyses, we only restricted QST variables to only those correlated with both catastrophizing and pain outcomes ($p<.05$); likewise we restricted outcome variables to only those correlated with both catastrophizing and QST variables ($p<.05$). Again, estimates of indirect effects were considered significant in the case that zero was not included in the confidence intervals [56, 57]. All statistical analysis was conducted used SPSS 24 (Chicago, IL, USA).

RESULTS

Participant Characteristics

The sample consisted of 200 participants in total (167 cLBP; see Table 1). There were no significant differences between the groups with respect to age, gender, or race. Compared to healthy controls (who did not use opioid pain medications), approximately 16% of cLBP patients used them ($X^2_1=4.97$, $p<0.05$). Patients with cLBP also reported greater overall depressive symptoms ($t_{198}=-5.77$, $p<0.01$) and pain catastrophizing ($t_{80.8}=-8.95$, $p<0.01$).

QST Differences between HC and cLBP Groups

Results of a series of independent samples t-tests comparing pain sensitivity between patients with cLBP and healthy controls indicated significant differences across several QST modalities (see Table 2). Patients with cLBP demonstrated lower spatial acuity in the low back area ($t_{195}=-2.26$, $p<0.05$) compared to healthy controls; however, there were no differences in spatial acuity at the finger, a pain-free control site (i.e., finger; $p>0.05$). Compared to healthy controls, cLBP patients required less cuff inflation pressure to produce moderate deep-tissue pain (P40; $t_{198}=2.83$, $p<0.01$) and provided higher pain intensity ratings for mechanical punctate probes of various forces ($t_{114.3}=-5.91$, $p<0.01$), offering evidence for hyperalgesia in cLBP patients. Both cLBP patients and healthy controls demonstrated temporal summation of mechanical punctate pain, and there were no group

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differences in temporal summation. Patients with cLBP did, however, rate aftersensations from the mechanical punctate stimulus as more painful ($t_{64.2}$=−2.65, p<0.05) than their pain-free counterparts.

Bivariate Associations Between Catastrophizing and QST Responses among all Participants

Results of Pearson correlations indicated that greater catastrophizing was associated with decreased spatial acuity on the lower back, as reflected by a larger two-point discrimination threshold (*r=.161; p*<0.05; see Table 3). Greater catastrophizing was also associated with lower P40 cuff inflation pressure (r=−.196; p<0.01), as well as higher ratings for pressure pain aftersensations (r=.199; p<0.01) and mean pressure pain unpleasantness (r=.259; p<0.01).

Catastrophizing as a Mediator of Group Differences in QST

As indicated in the analysis section, mediation analyses were only conducted (1) there were significant group differences in the QST response (i.e., low back spatial acuity, P40 pressure, mean mechanical punctate pain ratings, and mechanical painful aftersensations) and (2) the QST response was significantly correlated with catastrophizing (low back spatial acuity, P40 pressure, and average pressure pain unpleasantness and aftersensations). This resulted in mediation analyses for low back spatial acuity and P40 pressure. Results of the first mediation analysis indicated that catastrophizing accounted for 23% of the variance in P40 cuff inflation pressure and significantly mediated the group differences in P40 cuff inflation pressure. Patients with cLBP endorsed higher levels of catastrophizing which were associated with requiring less pressure to produce moderate deep-tissue pain (see Figure 2). Catastrophizing did not mediate the group differences in low back spatial acuity (CI includes 0).

Relationships Between Catastrophizing, Pain Outcomes, and QST in cLBP Participants

Results of Pearson correlations used to examine the relationships between catastrophizing, clinical pain outcomes, and QST variables within the cLBP group indicated that greater catastrophizing was associated with pain in the past month (r=.359, p<0.01), pain in the last week (r=.407; p<0.01), pain at the visit (r=.333, p<0.01), low back pain bothersomeness (r=.429, p<0.01), and pain interference (r=.521, p< 0.01); see Table 4). Within the cLBP group, greater catastrophizing was also associated with lower P40 cuff inflation pressure (r=−.152, p<0.05) and greater pressure pain unpleasantness (r=.256, p<0.01) and aftersensations (r=.188, p<0.05). Lower P40 cuff inflation pressure was associated with greater pain in the past month (r=−.215, p<0.01), pain in the past week (r=−.163, p<0.05), pain at the visit (r=−.233, p<0.01), and backpain bothersomeness (r=−.221, p<0.01). Further, greater pain in the past month was associated with greater painful pressure aftersensations (r=.246, p<0.01) while greater pain the last week was associated with greater pressure pain unpleasantness (r=.164, P<0.05). We conducted mediation analyses only when relationships between catastrophizing and pain outcomes (i.e., pain in the last month, pain in the last week, pain at the visit, pain bothersomeness, and pain interference), catastrophizing and QST (i.e., P40 pressure and pressure pain unpleasantness and aftersensations), and QST and pain outcomes (i.e., P40 with pain in the past month, pain in the past week pain at the visit, and pain bothersomeness;

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pressure pain unpleasantness and pain in the last week; pressure pain aftersensations and pain in the past month) were all significant ($p<.05$). Thus we ran 4 separate models. In the first model, P40 cuff inflation pressure and ratings of pressure pain aftersensations jointly accounted for 19% of the variance in self-reported pain in the past month. However, only P40 cuff inflation pressure mediated the relationship between catastrophizing and pain in the past month. That is, individuals who endorse greater catastrophizing also demonstrate deep-tissue hyperalgesia which is associated with greater self-reported pain in the past month (see Figure 3). In the second model, cuff inflation pressure accounted for 11% of the variance in pain rating at the time of the visit and significantly mediated the relationship between catastrophizing and pain at the time of the visit (see Figure 4). Greater catastrophizing was associated with lower P40 cuff inflation pressure which, in turn, accounted for greater pain ratings at the time of the visit. In the third model, cuff inflation pressure accounted for 9% of the variance in low back pain bothersomeness, and significantly mediated the relationship catastrophizing and low back pain bothersomeness such that individuals who endorsed greater catastrophizing demonstrated deep-tissue hyperalgesia, which was further associated with greater low back pain bothersomeness (see Figure 5). In the final model, P40 cuff inflation pressure and pressure pain unpleasantness did not mediate the relationship between catastrophizing and pain in the past week.

DISCUSSION

In the current study, we examined the association between two key putative contributors to cLBP: pain sensitization and pain catastrophizing. We found that patients with cLBP demonstrated increased pain sensitivity compared to healthy, pain-free controls for various somatosensory measures, and that catastrophizing partially mediated the group differences in the pressure necessary to produce moderate deep-tissue pain. Additionally, we found that greater catastrophizing was associated with both experimental pain sensitivity and clinical pain among patients with cLBP, with deep-tissue hyperalgesia mediating the relationship between catastrophizing and clinical pain among cLBP patients.

Consistent with our hypothesis, we found that patients with cLBP demonstrated greater deep-tissue hyperalgesia as well as increased sensitivity for mechanical punctate pain compared to pain-free controls. While previous studies have reported that cLBP patients exhibit increased pain sensitivity compared to pain-free controls [23, 30, 49, 50, 58], there is considerable heterogeneity in both the QST methodology used in these studies and the nature of their findings. For example, Giesecke and colleagues found that pressure pain thresholds on the thumb were greater among cLBP patients compared to healthy controls [23]. On the other hand, O'Neill and colleagues found that pressure pain thresholds differed between groups only on the infraspinatus muscle (i.e., shoulder) but not the tibialis anterior (i.e., shin) but also that cLBP patients exhibited higher pain intensity of longer duration for saline-induced muscle pain [49]. The present study builds upon previous work by using novel methodology (e.g., cuff pressure algometry) while also assessing a variety of QST outcomes to highlight individual differences in pain sensitization while still demonstrating a general pattern of increased sensitization among cLBP patients.

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In addition to group differences in pain sensitization, we found that, compared to pain-free controls, cLBP patients demonstrated decreased spatial acuity for an innocuous somatosensory stimulus at the pain site (i.e., back) but not at a non-painful control site (i.e., finger). This aligns with previous findings [43, 47] suggesting an impairment in somatosensory processing at the site of back pain. There were no differences in the spatial acuity at the hand, signifying that spatial acuity may be pain-site specific and possibly not global in cLBP patients. However, it is important to note that there was a small to moderate effect size for spatial acuity on the hand. Thus additional research is warranted to better characterize somatosensory perception, especially for non-noxious stimuli, among patients with cLBP. Taken together, these findings suggest that patients with cLBP demonstrate localized hyposensitivity to non-noxious stimuli despite their increased sensitivity to noxious stimuli overall.

Results of the current study also suggest that catastrophizing may play a role in pain sensitization among patients with cLBP. We found that patients with cLBP endorsed greater levels of catastrophizing, with greater catastrophizing accounting for the group differences in deep-tissue hyperalgesia. This finding supports the extant theory suggesting that psychosocial factors such as catastrophizing may may serve as a mechanism in central pain sensitization [9, 44]. Further, Taub and colleagues found that experimental manipulation of catastrophizing among women with cLBP was associated with greater pain sensitivity to punctate mechanical pain, as well as mechanical allodynia [64], lending further support to the hypothesis that catastrophizing may be causally linked to increased pain sensitization. In fact, this association may be supported by increased functional connectivity between the brain's primary somatosensory (S1) cortical representation of the body site for evoked pain and right anterior insula cortex, as demonstrated by our recent study in chronic musculoskeletal pain patients [35].

We also sought to examine the relationship between pain sensitization, catastrophizing, and clinical pain outcomes among patients with cLBP. Consistent with previous studies [7, 11, 29, 42, 54], our results indicated that catastrophizing was associated with both pain sensitization and clinical pain outcomes, while pain sensitization (i.e. deep-tissue hyperalgesia) was also associated with clinical pain outcomes as proposed in Curatolo & Arendt-Nielsen model [11]. Furthermore, the relationships between catastrophizing and clinical pain outcomes were driven, in part, by differences in deep-tissue hyperalgesia. That is, cLBP patients who exhibited greater deep-tissue hyperalgesia also reported greater pain intensity in the past month, pain at the time of the visit, and low back pain bothersomeness, and these associations partly accounted for the deleterious effect of catastrophizing on back pain intensity and bothersomeness. While the central nervous system mechanisms supporting these links between catastrophizing and clinical pain are not well understood, our recent study found that engaging in a catastrophizing task activates the brain's posterior cingulate cortex (PCC), and that greater clinical pain severity was specifically associated with greater activation of the dorsal PCC [40].

Collectively, these results support the notion that central sensitization of pain may be one of the mechanisms implicated in the development and maintenance of idiopathic chronic low back pain, and that this sensitization may be partially driven by negative affective and

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cognitive responses to pain, as instantiated by elevations in pain-related catastrophizing. Moreover, our findings suggest that greater catastrophizing and the resulting pain sensitization are both associated with greater pain intensity and low back pain bothersomeness among patients with cLBP. These results have important clinical implications for clinicians involved in the management of patients with cLBP. Instead of focusing on localized pain treatment (e.g., injections, topical analgesics, TENS), clinicians may consider targeting systemic sensitization processes via reductions in catastrophizing. Indeed, there is evidence suggesting that Cognitive Behavior Therapy for Pain is effective in reducing catastrophizing, and that these reductions in catastrophizing are associated with reduced pain and improved functioning among patients with chronic pain [6, 39, 65].

Several limitations should be considered when interpreting these findings. First, our sample is disproportionally weighted with cLBP patients compared to healthy, pain-free controls. As a result of this numeric imbalance in group sizes, we may have been underpowered to examine the mediation of group differences in pain sensitivity. Despite this reduced power, we found meaningful group differences, which suggests that the magnitude of these differences was considerable. An additional limitation is the cross-sectional nature of this study. As such, a prospective, longitudinal approach should be considered for future studies to examine catastrophizing as a predictor of the development of sensitization prior to the onset of cLBP. Although we did identify participants using opioids at the time of the study, we did not account for the frequency, dosing, or duration of opioid use. Given concerns with opioid-induced hyperalgesia, future studies should measure more detailed patterns of opioid use. Finally, although pain sensitization at sites distal to the primary source of pain (e.g., the back) suggests a central sensitization of pain, future studies should utilize neuroimaging methodology to better understand the neural mechanisms involved in both catastrophizing and pain sensitization among patients with cLBP.

In conclusion, the current study uses novel QST modalities, a large clinical sample, and formal mediation analysis to provide a better understanding of pain sensitization among patients with cLBP. Not only did patients with cLBP demonstrate increased pain sensitization and decreased spatial acuity, but these somatosensory changes were also linked with increased catastrophizing. Furthermore, both catastrophizing and sensitization were associated with increased clinical pain among cLBP patients. These findings have important clinical implications and provide us with potential treatment targets for patients with cLBP.

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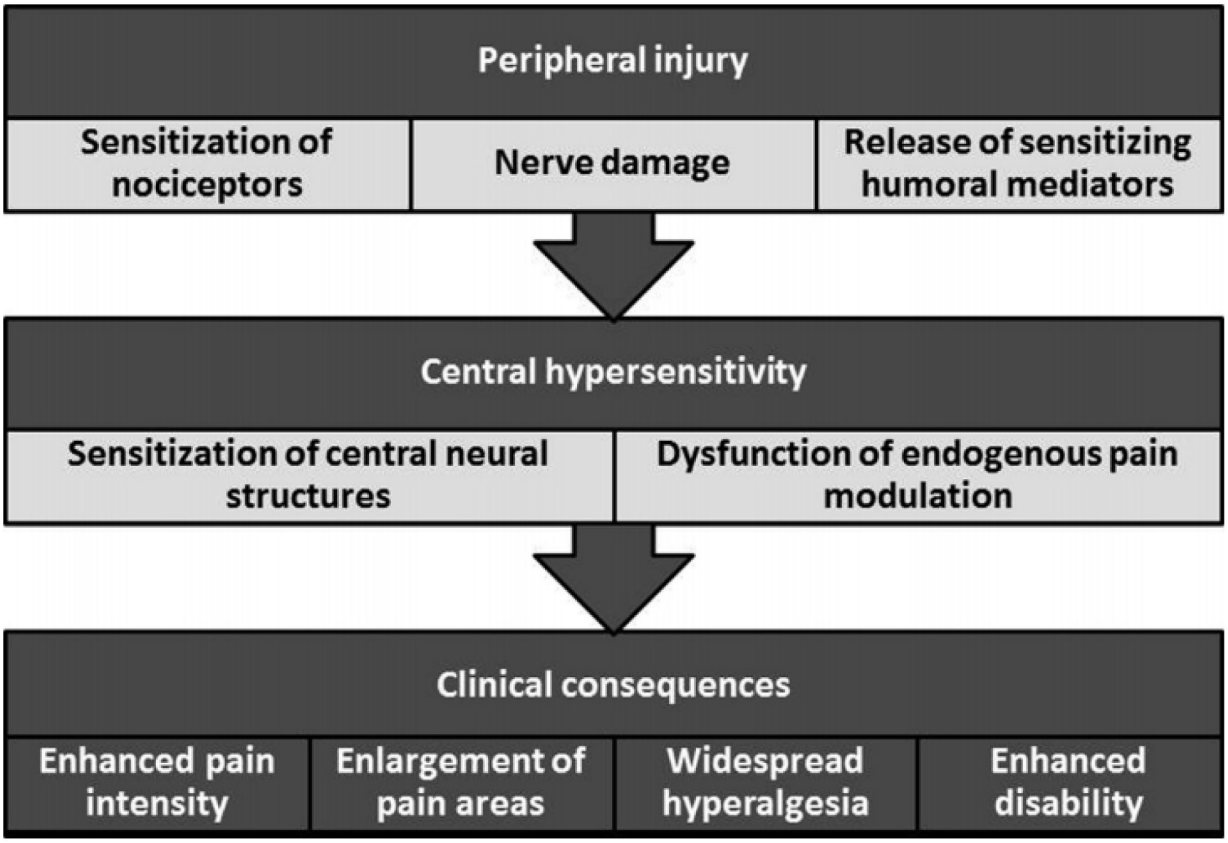

Figure 1.
Curatolo & Arendt-Nielsen (2015) model of path between peripheral injuries, central hypersensitivity, and clinical consequences.

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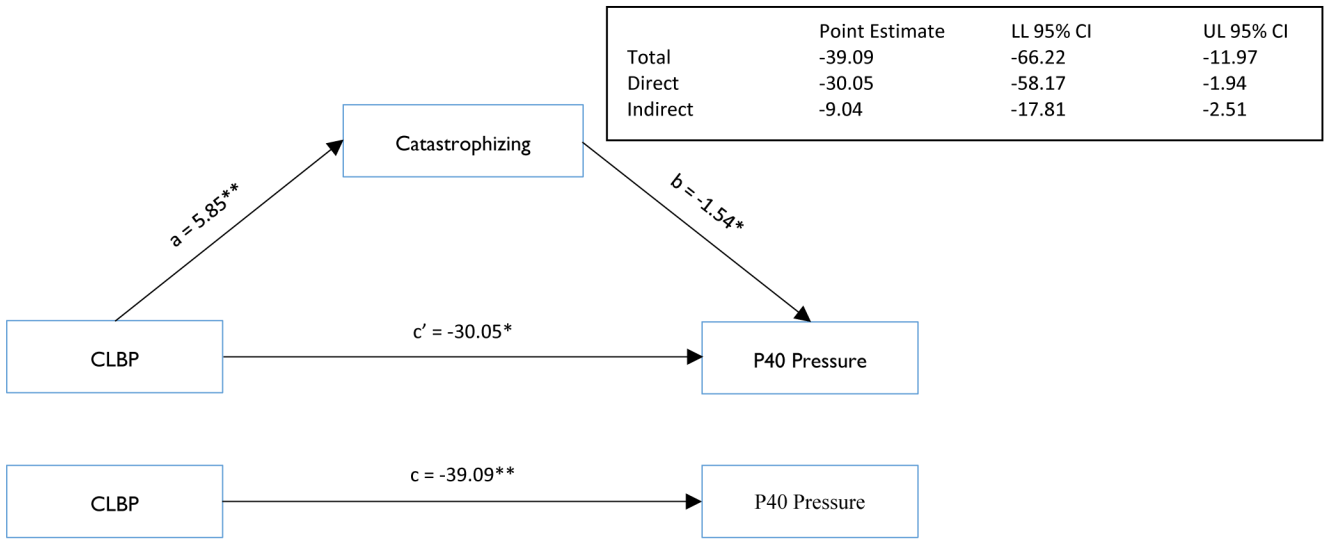

Figure 2.

The mediating effect of catastrophizing in the relationship between back pain and P40 cuff inflation pressure. *p<.05; **p<.01

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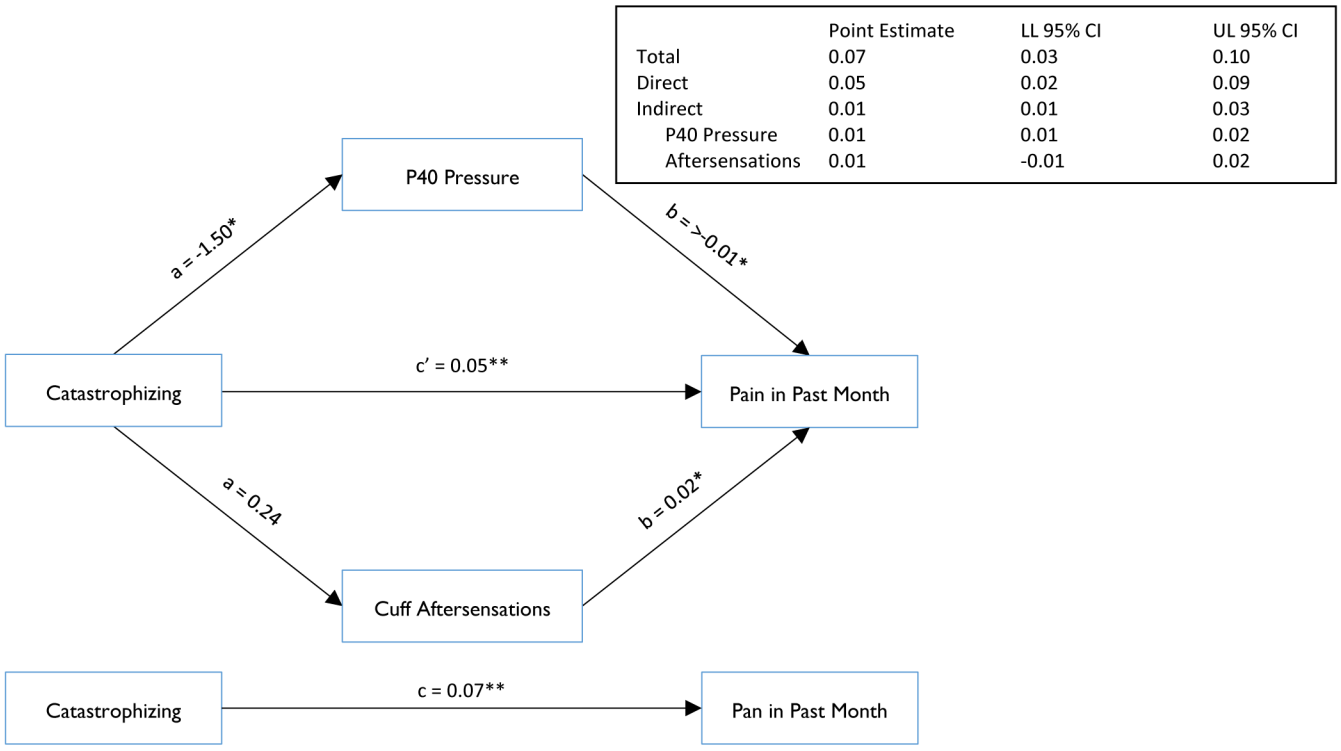

Figure 3.

The mediating effect of pain sensitization (as measured by P40 cuff inflation pressure and cuff aftersensations) in the relationship between catastrophizing and pain in the past month controlling for opioid use and depression. *p<0.05; **p<0.01

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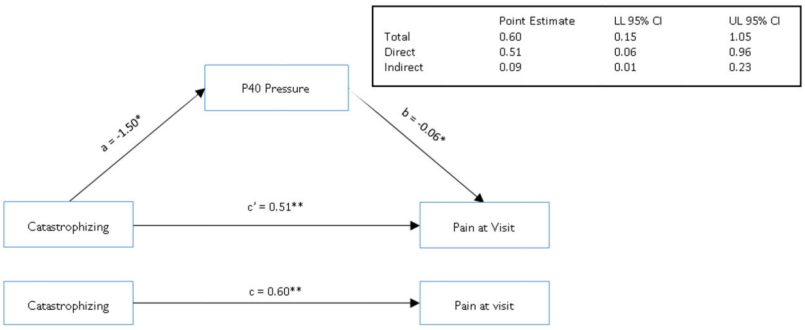

Figure 4.

The mediating effect of pain sensitization (as measured by P40 cuff inflation pressure) in the relationship between catastrophizing pain rating at time of visit controlling for opioid use and depression. *p<0.05; **p<0.01

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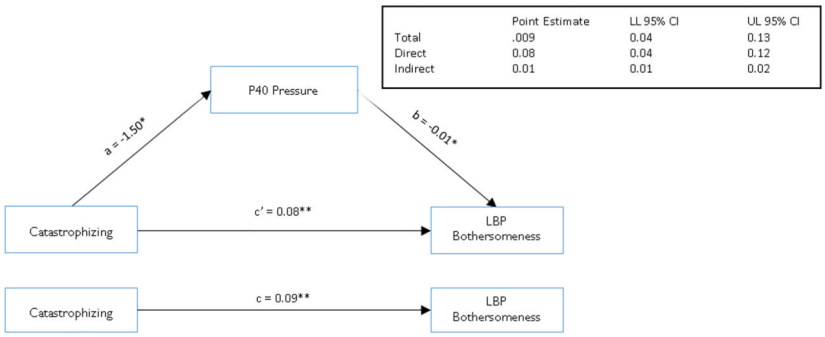

Figure 5.

The mediating effect of pain sensitization (as measured by P40 cuff inflation pressure) in the relationship between catastrophizing and low back pain bothersomeness controlling for opioid use and depression. *p<0.05; **p<0.01

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Table 1.

Participant Characteristics

Characteristics	HC(n=33)	CLBP(n=167)	t or X^2 statistic
Age, mean (SD)	43.35 (10.84)	40.77 (12.29)	1.22
Sex, n (%)			0.14
Male	15 (45)	70 (42)	
Female	18 (55)	97 (58)	
Race, n (%)			2.62
American Indian / Alaska Native	0 (0)	1 (<1)	
Asian	2 (6)	8 (5)	
Native Hawaiian or Other Pacific Islander	0 (0)	1 (<1)	
Black or African American	4 (12)	17 (10)	
White	26 (79)	122 (73)	
More than one race	1 (3)	12 (7)	
Unknown	0 (0)	6 (4)	
Opioid Use, n (%)			4.97*
Yes	0 (0)	22 (13)	
Depression, mean (SD)	1.7 (3.7)	6.6 (6.8)	−5.77**
Pain in the past month, mean (SD)	.1 (.4)	5.9 (1.8)	−36.39**
Pain in the past week, mean (SD)	.2 (.9)	5.6 (1.7)	−27.10**
Pain at visit, mean (SD)	.3 (1.7)	43.4 (21.7)	−25.27**
Low back pain bothersomeness, mean (SD)	- (−)	6 (2)	-
Pain interference, mean (SD)	- (−)	11 (4)	-
Pain duration in years, mean (SD)	- (−)	8 (7)	-
Catastrophizing, means (SD)	3 (5)	13 (9)	−8.95**

CLBP: Chronic low back pain

HC: Healthy pain-free control

** p<0.01

* p<0.05

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Table 2.

Independent t-tests for QST Outcomes

Outcome	Group	Mean	Std. Deviation	t	d
Two-point Discrimination-Finger	HC	3.16	1.53	−1.83	0.37
	CLBP	3.78	1.83		
Two-point Discrimination-Back	HC	34.53	15.42	−2.26*	0.45
	CLBP	41.73	16.69		
P40 Pressure	HC	201.94	79.81	2.83**	0.50
	CLBP	165.69	64.66		
Cuff Mean Pain Intensity	HC	44.89	18.73	−1.21	0.21
	CLBP	48.53	15.10		
Cuff Mean Pain Unpleasantness	HC	49.75	21.91	−1.15	0.21
	CLBP	54.27	20.44		
Cuff Painful Aftersensations	HC	3.85	7.31	−1.66	0.25
	CLBP	6.61	13.90		
1st Probe Pain Rating	HC	10.94	11.85	−1.95	0.40
	CLBP	16.08	13.98		
10th Probe Pain Rating	HC	25.58	25.14	−1.58	0.28
	CLBP	32.05	20.49		
Mean Pain Rating 64-256mN Probes	HC	3.09	4.56	−5.91**	0.82
	CLBP	10.06	11.11		
Temporal Summation of Probes	HC	14.64	16.73	−0.40	0.08
	CLBP	15.97	17.57		
Probe Painful Aftersensations	HC	2.87	7.00	−2.65*	0.43
	CLBP	6.84	10.92		

** $p<0.01$

* $p<0.05$

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Table 3.

Pearson Correlations Between Catastrophizing, and QST Measurements in Entire Sample

	Catastrophizing
Two-point Discrimination-Finger	0.078
Two-point Discrimination-Back	0.161 *
P40 Pressure	−0.196 **
Cuff Mean Pain Intensity	0.120
Cuff Mean Pain Unpleasantness	0.259 **
Cuff Painful Aftersensations	0.199 **
1st Probe Pain Rating	0.129
10th Probe Pain Rating	0.077
Pain Rating 64-256mN Probes	0.122
Temporal Summation of Probes	−0.007
Probe Painful Aftersensations	0.134

** $p<0.01$

* $p<0.05$

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Table 4.

Pearson Correlations Between Clinical Outcomes and QST Measurements in CLBP Participants

	Sex	Age	Opioid Use	Depression	Catastrophizing	Pain in Past Month	Pain in Last Week	Pain at Visit	LBP Bothersomeness	Pain Interference	Pain Duration
Sex	1	−0.150	−0.097	−0.148	0.015	0.202**	0.140	0.062	0.078	0.004	−0.039
Age	−0.150	1	0.164*	0.094	0.050	0.058	0.022	0.045	−0.014	0.122	0.220**
Opioid Use	−0.097	0.164*	1	0.185*	0.236**	0.111	0.155*	0.163*	0.088	0.239**	−0.161*
Depression	−0.148	0.094	0.185*	1	0.547**	0.276**	0.307**	0.252**	0.310**	0.460**	0.014
Catastrophizing	0.015	0.050	0.236**	0.547**	1	0.359**	0.407**	0.333**	0.429**	0.521**	−0.029
Two-point Discrimination-Finger	0.03	0.120	0.127	0.051	0.053	0.142	0.042	−0.039	0.082	0.076	0.071
Two-point Discrimination-Back	−0.023	0.172*	0.123	0.129	0.143	0.117	0.072	0.026	0.082	0.145	0.081
P40 Pressure	−0.225**	−0.035	0.005	−0.005	−0.152*	−0.215**	−0.163*	−0.233**	−0.221**	0.067	0.067
Cuff Mean Pain Intensity	−0.023	0.066	−0.53	−0.101	0.112	0.153*	0.162*	0.034	0.101	0.022	0.022
Cuff Mean Pain Unpleasantness	0.094	−0.039	−0.093	0.015	0.256**	0.14	0.164*	0.071	0.144	0.023	0.023
Cuff Painful Aftersensations	0.104	−0.022	−0.072	0.133	0.188*	0.246**	0.15	0.082	0.131	−0.02	−0.02
1st Probe Pain Rating	0.124	−0.169*	−0.078	−0.027	0.102	0.163*	0.078	0.016	0.131	−0.012	−0.004
10th Probe Pain Rating	0.125	−0.205**	−0.129	−0.076	0.054	0.191*	0.116	0.126	0.131	0.19	−0.093
Pain Rating 64-256mN Probes	0.092	−0.220**	−0.105	0.057	0.024	0.051	0.051	0.098	−0.005	−0.147	−0.147
Temporal Summation of Probes	0.046	−0.105	−0.088	−0.067	−0.019	0.095	0.073	0.134	0.119	0.032	−0.105
Probe Painful Aftersensations	0.144	−0.064	−0.122	−0.050	0.1	0.233**	0.187*	0.078	0.184*	0.045	−0.46

** p<0.01

* p<0.05