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Situational vs. dispositional measurement of catastrophizing: Associations with pain responses in multiple samples

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Abstract

Catastrophizing is widely recognized as an important risk factor for adverse pain-related outcomes. However, questions remain surrounding the details of its assessment. In particular, recent laboratory studies suggest that evaluation of “situational” catastrophizing (i.e., catastrophizing measured during or directly after the administration of noxious stimulation) may provide information distinct from that obtained by standard, or “dispositional” measures which assess individuals’ recall of catastrophizing in daily life. However, comparatively little research has systematically investigated the inter-relationships and properties of these two different forms of pain-related catastrophizing. The current study evaluated both situational and dispositional catastrophizing measures within multiple samples: healthy individuals (N=84), patients with painful temporomandibular joint disorders (TMD; N=48), and patients with painful arthritis (N=43). All participants first completed the Pain Catastrophizing Scale (PCS)⁴⁹, and then underwent psychophysical pain testing, which included heat, cold, and pressure pain. Participants then completed a situational catastrophizing measure with reference to the laboratory pain he/she had just undergone. Situational catastrophizing scores were not significantly correlated with dispositional PCS scores in the healthy participants and arthritis patients, though they were associated in TMD patients. Situational catastrophizing was more strongly associated with experimental pain responses than dispositional PCS scores for the healthy subjects and arthritis patients. In general, higher levels of situational catastrophizing were associated with lower pain thresholds and higher pain ratings across all 3 samples. The findings highlight the importance of multi-dimensional assessment of pain-related catastrophizing, and suggests a role for measuring catastrophizing related to specific, definable events.

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Perspective

This study adds to a growing literature examining catastrophizing. Our findings highlight the potential importance of the multi-dimensional assessment of pain-related catastrophizing, and suggest a role for measuring catastrophizing related to specific, definable events.

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Keywords

Situational Catastrophizing; Dispositional Catastrophizing; TMD; Arthritis; experimental pain

Introduction

A substantial literature has demonstrated that catastrophizing, a set of negative emotional/cognitive processes (such as magnification of pain, rumination and pessimism about pain, and feelings of helplessness when in pain) is a critically-important risk factor for adverse pain-related outcomes. It has been positively associated with elevated indices of pain severity, pain behaviors, analgesic use,³⁴ interleukin-6 (a pro-inflammatory cytokine) reactivity,²⁰ length of hospital stay, postoperative recovery time,⁴⁸ suicidal ideation,²² and physical disability (see 5:50 for reviews of the catastrophizing literature), as well as with the prospective development of pain conditions such as spinal pain and phantom limb pain.^{7,43,45}

In some studies, catastrophizing has also been related to experimental pain responses, with those reporting increased catastrophizing generally showing more sensitivity to calibrated noxious stimuli administered in a controlled setting.⁵⁰ For example, Edwards and colleagues²² found that increased pain-related catastrophizing was associated with greater supra threshold ratings and elevated temporal summation of noxious thermal stimuli. Moreover, several studies have shown that catastrophizing was associated with greater sensitivity to cold pain (e.g., individuals high in catastrophizing had a lower tolerance for cold pain and rated cold pain intensity more highly).^{13,17} Two studies of electrical pain responses also found that catastrophizing related to elevated pain sensitivity, though in both cases, only self-reported pain and not nociceptive muscle reflexes, were affected by an individual's degree of catastrophizing.^{27,44}

One interesting feature of some of these experimental pain studies is that they have assessed catastrophizing specifically with respect to the noxious stimuli that are delivered in the laboratory (i.e., "situational catastrophizing"). In these studies, catastrophizing is measured immediately after a specific pain experience that is shared by all participants. It seems natural to expect that two different measures of pain catastrophizing, a construct with some trait-like characteristics and that is fairly stable over time,⁵³ would be highly inter-correlated. However, previous reports have documented, at best, moderate correlations between scores on a dispositional catastrophizing measure such as the Pain Catastrophizing Scale (PCS; which cues respondents to recall prior pain experiences and then queries their typical degree of catastrophizing) and situational catastrophizing scores.^{13,17} Moreover, when the two measures of catastrophizing have been directly compared, situational catastrophizing has been shown to correlate significantly more highly with experimental pain responses. This is consistent with classic research on interactions between individual trait characteristics and environmental circumstances in which expression of a behavioral disposition, such as catastrophizing, depends on existing situational demands.⁴⁰ Similarly, early coping research has suggested that coping disposition was only weakly associated with coping behaviors in a given situation.^{9,36}

Collectively, these findings suggest that situational measurement of pain-related processes such as catastrophizing, which are captured during or immediately after a specific pain induction procedure, may have greater accuracy and be more relevant to an individual's experience of pain than dispositional measures of how much an individual recalls responding in general. The PCS⁴⁹ measure relies on the respondent's choice of a pain referent (i.e., some previously painful event), and the recall of their response. This can create an important bias, as memory is known to be influenced by emotional processes^{37,39} and personality factors.⁸

Recently, findings have emerged in the depression literature, where day-to-day variability in “state” measures of depressed mood was associated with physiologic markers of stress where as a “trait” measure of depressive symptomatology was not.⁴⁶

However, to date the associations between different modes of assessing catastrophizing have only been examined in student samples, not in clinical populations in whom catastrophizing might be relatively more important in shaping their experiences of pain. The current analyses combined data from two separate studies, one described in a previous publication¹⁹ and one not yet published. Both sought to elucidate the associations of situational and dispositional catastrophizing measures with a variety of experimental pain responses among healthy adults, patients with arthritis pain and patients with temporomandibular joint disorders (TMD), all of whom underwent a standardized array of experimental pain induction procedures. It was hypothesized that measures of situational and dispositional catastrophizing would be more strongly inter-correlated with one another in the patient groups, and that situational catastrophizing would be more strongly associated with experimental pain responses (e.g., pain thresholds, ratings of standardized noxious stimuli) than dispositional catastrophizing across all of the groups. These results were presented in abstract/poster form at the annual meeting of the American Pain Society.⁶

Materials and Methods

Data from a total of 175 individuals (84 healthy individuals, 48 temporomandibular joint disorder patients, and 43 patients with arthritis pain) were analyzed in this study. Data were pooled from a previous study of sleep and TMD pain¹⁹ and a study of stress-immune system responsiveness in arthritis patients (unpublished). Approximately half of the healthy individuals and all of the TMD participants included here were part of the pain and sleep study¹⁹ and only baseline pain induction procedures (prior to sleep disturbance) were used in the current analyses. The arthritis patients and the other half of the healthy individuals participated in the stress-immune system study; again only baseline pain procedures were included in the current analyses. All study-related procedures were approved by the Johns Hopkins Hospital Institutional Review Board. All participants were recruited through posted advertisements throughout the medical institution and undergraduate/graduate schools. Potential participants contacted the research coordinators who conducted an extensive telephone screening. Eligibility criteria for healthy participants included having no pain condition or medical disorders. All participants were excluded for an active alcohol or drug abuse problem, or use of narcotics, antidepressants, anticonvulsants, and muscle relaxants. For the two patient groups, individuals reporting primary pain conditions or serious medical disorders other than TMD or arthritis were excluded. Both patient groups were required to report a typical pain severity ≥ 2 out of 10 and minimum symptom duration ≥ 6 months. Interested and potentially eligible individuals were scheduled for a physician assessment of additional eligibility criteria. Potential TMD participants were scheduled to meet with an oral medicine specialist who conducted the research diagnostic criteria [RDC for TMD¹⁴] and confirmed primary diagnosis of TMD (Axis I [group I, II, and/or III]). Osteoarthritis (OA) and rheumatoid arthritis (RA) patients also had a physician-confirmed arthritis diagnosis (i.e., many had been evaluated at the Johns Hopkins Arthritis Center). OA of the knee was confirmed using ACR Clinical Classification Criteria (e.g., pain in the knee and radiographic evidence of joint space narrowing or osteophytes). RA was diagnosed using ACR Clinical Classification Criteria, and both groups were combined into one group to generate an adequate sample size for data analyses. Previous catastrophizing work has also combined OA and RA patients,³⁸ and catastrophizing is clinically relevant in both groups.¹⁶ In the TMD group, six individuals were deemed ineligible based on RDC/TMD criteria, nine participants were lost to follow-up between their RDC/TMD visit and pain testing sessions and an additional five participants had incomplete pain testing data due to equipment malfunctioning and were thus excluded from

these analyses. Three arthritis patients and two healthy participants from the sleep disturbance study did not complete all pain testing measures, again due to equipment issues, and were therefore not included in the final analysis.

Verbal and written informed consent was obtained upon arrival, after which participants completed the Pain Catastrophizing Scale (PCS)⁴⁹ and the other self-report psychological questionnaires in paper and pencil format. Next, participants underwent psychophysical pain testing, following which he/she completed the situational catastrophizing questionnaire. The laboratory pain induction procedures included pressure, thermal, and cold pressor pain assessment. The pressure and thermal procedures were conducted first in randomized order, followed by the cold pressor procedure.

Psychophysical Pain Testing

Pressure Pain Procedure—A Somedic algometer (Sollentuna, Sweden) was used to assess responses to mechanical pressure using a 0.503-cm² probe covered with a 1-mm polypropylene material.³⁵ Pressure was gradually increased at an application rate of 30 kPa per second until the subject reported pain threshold (i.e., “first felt pain”), at which point stimulation was terminated. Pressure pain threshold was assessed twice each at the right and left upper trapezius muscles. The four pressure pain thresholds were averaged together.

Thermal procedures

Heat Pain Threshold (HPTh): A computer-controlled Medoc Thermal Sensory Analyzer (TSA-2001, Ramat Yishai, Israel), a peltier-element-based stimulator with a 30×30 mm surface area, was used to deliver contact heat stimuli. HPTh was assessed three times on the left ventral forearm using an ascending method of limits. The temperature increased from a baseline of 32 °C with a 0.5 °C/s rate of rise, until the participant responded by pressing a button (when he/she first noticed a painful sensation). When the button was pressed, the temperature of the thermode was recorded. Between trials, the positioning of the thermode was moved up the arm slightly to avoid overlapping the testing sites. A 30-s inter-stimulus interval was maintained. The cutoff temperature for all trials was 52 °C to avoid tissue damage. The HPTh was determined as the average of these trials.

Ratings of Supra Threshold Heat Stimuli: Two series of brief, repetitive noxious thermal stimuli were administered in ascending order to the left ventral forearm. Within each series there were 10 stimuli. The target temperatures were 49 and 51 °C. Each stimulus was delivered for a 0.5-s duration, with a 3-s inter-pulse interval. Participants provided intensity ratings for each of the ten stimuli using a 0–100 scale (where 0 = no pain and 100 = the most intense pain imaginable). The trial was terminated if the subject said “stop” at any point, or if he/she provided a rating of 100. Additionally, participants were asked to rate painful after-sensations 15 seconds following the final pulse.

Cold Pressor Pain: Cold pressor pain was assessed by having the subjects immerse their right hand up to the wrist in 4 °C water. The water temperature was maintained (± 0.1 °C) by a refrigeration unit (Neslab, Portsmouth, NH), and was constantly circulated to prevent local warming around the submerged hand. The participant was prompted to rate their hand pain on the 0–100 scale at 20 seconds post-submersion. Participants were instructed to remove their hand from the water following this brief procedure (or at any time if intolerable). Participants also rated their maximum level of hand pain during this procedure. Cold water immersion was repeated twice and ratings were averaged together.

Psychological measures

Depression Measures

The Beck Depression Inventory (BDI) is one of the most commonly-used measures of depressive symptomatology. It assesses the frequency and severity of a variety of cognitive, affective, physiological, and motivational symptoms of depression.² Each item is scored on a four-point scale (0–3) with higher scores reflecting more severe symptoms. The BDI has been shown to be a reliable and valid index of depressive symptomatology in general and clinical populations.¹ It correlates highly with the diagnosis of depression using structured clinical interviews.²⁸

The Brief Symptom Inventory (BSI) is a well-normed, 53-item self-report measure of multiple dimensions of psychological symptoms.¹² Each item is scored on a 5-point Likert scale, with responses ranging from 0 (not at all) to 4 (extremely). The six-item depression subscale was used in the current analysis. This measure is widely used and has been shown to be reliable and valid in both general and clinical populations.¹¹ Data for the current analysis is a combination of two studies in which one (the immunity study) used the BDI and the other used the BSI-depression subscale (sleep study). Therefore, these measures were z-scored within their respective samples. The subsequent Z-scores were used in the regression analyses

Catastrophizing Measures

The Pain Catastrophizing Scale (PCS)⁴⁹ consists of 13 items rated on a 5-point scale ranging from 0 (not at all) to 4 (all the time). Participants are instructed to indicate the degree to which he/she has specific thoughts and feelings when experiencing pain. The measure assesses three dimensions of catastrophizing: rumination, magnification, and helplessness. The PCS has been validated for both clinical and nonclinical samples.⁴¹⁻⁴⁹

The Situational Catastrophizing Questionnaire (SCQ) is a six question adaptation of the PCS (described above). It has been used by our group in other studies and has been described more fully by Edwards et al.²² It is included in Appendix 1. Participants completed this questionnaire immediately following the pain induction procedures described above and were instructed to reference the pain procedures while answering. On the other hand, the depression measure and PCS were completed prior to conducting pain testing.

Data Analysis

All analyses were conducted using SPSS 16. Pain testing measures were z-scored separately in each participant group and combined into components based on Principal Component analysis with varimax rotation which yielded 3 factors: pain threshold (including thermal and pressure pain thresholds), supra threshold pain ratings (ratings of 49 and 51 °C stimuli, and cold pressor pain ratings), and painful after-sensations (following both supra threshold heat stimulations) see supplementary Table 1. Chi-Square tests were used to compare demographic variables between the three participant groups. To examine the relationship between situational and traditional catastrophizing measures with the other variables of interest, Pearson partial correlations were conducted (controlling for sex, age) to examine the associations between psychological variables and experimental pain components. A sensitivity analysis was also conducted to examine whether combining data from the two studies was appropriate.

A series of hierarchical linear regressions were then performed to examine the relationship between catastrophizing measures and each pain testing component for each of the three participant groups. Hierarchical regression was chosen as the most appropriate assessment, given our goal of elucidating the association between pain responses and catastrophizing measures within groups. To control for potential confounds related to demographic variables

and overlap between depression and catastrophizing characteristics, sex, age and ethnicity (coded as white/nonwhite for data analysis purposes) were entered into the first block, the depression z-score was entered into the second, and both catastrophizing measures were entered into the third block simultaneously.

In order to statistically test whether the two measures of catastrophizing (dispositional [PCS] and situational [SCQ]) differed significantly in their unique association with pain responses, the difference in unstandardized beta coefficients between the dispositional PCS and the SCQ was calculated via the studentized nonparametric bootstrap method. This technique^{10,23} allows statistical comparison of the difference in beta weights between two independent (predictor) variables in a regression equation, permitting conclusions regarding whether the observed associations with the dependent variable are significantly different.

Results

Chi-Square tests revealed that the sex and age distribution differed significantly between groups. More women were in the TMD and arthritis groups than the healthy participants, and arthritis patients were generally older than the other two groups. Demographic information, means (SD), Cronbach's alpha coefficients for situational and dispositional catastrophizing, and the raw pain data for each of the participant groups are presented in Table 1. Overall, situational catastrophizing scores ($F = 4.2$, $p = .02$) and PCS scores ($F = 5.1$, $p = .007$) were significantly different between the three groups. Situational catastrophizing differed significantly between arthritis patients and healthy participants ($p = .006$) as well as arthritis and TMD patients ($p = .015$), controlling for age and sex. Situational scores did not differ between healthy and TMD participants ($p > .05$). A significant difference on PCS scores emerged between healthy participants and both of the patient groups (TMD, $p = .01$; arthritis, $p = .009$), controlling for age and sex. No difference was observed between TMD and arthritis patients on the PCS. Internal consistency was adequate for both the situational and dispositional PCS measures for each group. Sensitivity analysis revealed that healthy individuals recruited to each study differed on age, as the immune response study sought to recruit arthritis patients and it was important that the healthy sample be more representative and comparable in age with the arthritis patients. No differences emerged between the two healthy groups on any experimental pain measures controlling for age.

Associations Between Measures

As reported in Table 2, situational catastrophizing scores were not correlated with dispositional PCS scores in the healthy participants and arthritis patients, though there was a significant positive association in the TMD patients. The dispositional PCS was significantly associated with depression scores in the healthy group and arthritis patients, while situational catastrophizing was correlated with depression in TMD patients. Moreover, at a univariate level, situational catastrophizing was generally more strongly associated with experimental pain responses (e.g. pain threshold, supra threshold pain ratings) than dispositional PCS scores, though these results varied by pain testing modality (see Table 2).

Hierarchical Regressions

Hierarchical linear regressions revealed that, after controlling for sex, age, ethnicity, depression and the dispositional catastrophizing measure, situational catastrophizing predicted pain threshold in healthy participants and arthritis patients (Tables 3a and 3b respectively) but not in TMD patients (Table 3c), though a trend toward significance was observed in this group ($p = .07$). Dispositional PCS did not significantly predict pain threshold after adjusting for these variables in any of the three participant groups. Additionally, beta weight calculations revealed that situational catastrophizing was a significantly better predictor than the dispositional PCS

measure in both the healthy and arthritis groups ($p < .05$). The situational measure also predicted supra threshold responses in healthy participants (Table 4a) after controlling for sex, age, ethnicity, depression and dispositional PCS, while the dispositional PCS measure did not (after controlling for demographics and situational catastrophizing). A significant difference between dispositional and situational catastrophizing measures observed in healthy participants ($p = .05$). This relationship was not found in the arthritis patients (Table 4b). The situational measure did predict supra threshold responses in TMD patients (Table 4c) after controlling for sex, age, ethnicity, depression and dispositional PCS, while the dispositional PCS measure did not; however a significant difference between dispositional and situational catastrophizing measures was not observed in this group. Interestingly, situational catastrophizing predicted after-sensation pain ratings in each of the three groups (Tables 5a, 5b and 5c) controlling for demographics as well as depression and dispositional PCS; again however, the situational measure was only significantly different from the dispositional PCS in healthy participants ($p = .04$). Dispositional PCS scores were not predictive of any pain responses measured for any of the three groups of participants although PCS beta weights approached significance as predictors among the TMD patients (threshold $p = .08$; supra threshold $p = .06$; after-sensation $p = .1$). The variance in pain responses accounted for by the construct of catastrophizing (including both situational and dispositional) ranged between 6 – 13% for healthy participants, 15 – 21% for TMD patients, and 3 – 16% for arthritis patients.

Discussion

The current study sought to extend recent work on the assessment of catastrophizing by examining both dispositional and situational responses in different populations. The situational catastrophizing measure (reported in 22) uses questions derived from PCS items, which were modified to apply to laboratory-based pain testing. In general, findings indicate that the situational catastrophizing was significantly associated with experimental pain measures, though these relationships evidenced a variable response pattern across participant groups. Situational catastrophizing significantly correlated with threshold (healthy and arthritis), supra threshold (TMD), and after-sensations (healthy, arthritis, and TMD), whereas the dispositional PCS measure did not significantly add to the models (adjusted for situational catastrophizing) in any participant group. Statistical trends were observed for associations between the dispositional PCS and pain thresholds and supra threshold ratings in TMD participants. The magnitude of the association between situational catastrophizing and pain responses is likely to be clinically significant. For the regressions in which situational catastrophizing is a significant predictor of pain responses, the betas range in absolute value from .05 to .08. In our previous work, we have reported that individuals who are low in situational catastrophizing differ from those who are high in situational catastrophizing by 8–10 points on this scale. 22 To simplify, by taking a 10-point “change” in that situational catastrophizing scale, going from low to high in situational catastrophizing is associated with a “unique change” in pain responses of between $\frac{1}{2}$ and $\frac{3}{4}$ of a standard deviation in pain response. This is true after accounting for the influence of demographic factors, depressive symptoms, and dispositional catastrophizing. We have previously noted that individual differences in pain sensitivity have important clinical implications for the development of clinical pain,¹⁵⁻²¹ and the present data suggest that individual differences in situational catastrophizing may be an important contributor to that variability.

Our findings parallel those of Dixon et al¹³ and Edwards and colleagues¹⁷ who found that situational reports of catastrophizing were more strongly associated with experimental pain report as compared to dispositional assessment of catastrophizing. Geisser and colleagues²⁹ modified the Coping Strategies Questionnaire (CSQ) in a similar fashion (i.e., changing questions to past tense and excluding inappropriate items) and administered the questionnaire following a cold pressor task. While the study did not include a comparison of the standard

CSQ measure to the situational version, pain tolerant individuals had significantly lower catastrophizing subscale scores than a pain sensitive group. Bruehl and colleagues³ also used a modified CSQ questionnaire (which they termed “CSQ-acute”); however, they did not examine the catastrophizing subscale. Clearly, situational and dispositional assessment of catastrophizing yield somewhat different information.

Potential mechanisms underlying these differences include a distinction between “trait” measurement (i.e., the dispositional PCS measures a person’s general tendency to catastrophize when in pain) and “state” measurement (i.e., the situational scale measures only an individual’s degree of catastrophizing in a specific situation, over an approximately 1-hour time span). While the state/trait distinction is to some degree artificial (e.g., recent findings suggest that purportedly “trait” measures of personality change as a function of treatment in patients with chronic pain),²⁵ it is likely that the PCS assesses a more broad, dispositional construct than the presumably situation-specific catastrophizing measure. It is also likely that the dispositional PCS measures different pain experiences than the situational scale, since there is no standard referent or time frame for subjects completing the PCS to use when thinking about their pain experience. These pain experiences may have occurred in the distant past in the case of healthy subjects and may include a summary of multiple pain experiences, including recent pain, in the clinical groups. Thus, dispositional PCS scores may relate only minimally to experimental pain responses because of the artificiality of the laboratory environment; that is, standard PCS scores may be driven by past experiences with clinical pain in daily life, whereas in a novel laboratory environment, participants have no relevant prior experience to draw on as they undergo the pain induction procedures. Additionally, the dispositional PCS may be more subject to distortion as a function of memory biases, which have been well-documented in the field of pain research.³¹ While this issue is perhaps less likely to apply to clinical populations, as a number of studies have shown strong associations between standard PCS scores and pain outcomes in the context of chronic pain, it may be relevant for healthy participants who have no experience with daily pain or whose pain experience is not known to the researcher. Finally, the possibility exists that situational catastrophizing scores are driven by individual differences in pain sensitivity, such that those who are more pain-sensitive experience more pain from the procedures and catastrophize more. Future studies may wish to examine how potential mechanisms influence the relationships between situational and dispositional catastrophizing for a better conceptual understanding of these measures.

It is interesting to note the differences in catastrophizing and its associations across groups after statistically controlling for sex, age, and ethnicity. Consistent with prior research, both groups of patients with persistent pain had higher dispositional PCS scores than generally healthy adults free from chronic pain. This is a common finding^{4,26,54} in cross-sectional comparisons of patients and healthy participants, although it is unclear whether such differences are due to catastrophizing acting as a risk factor for the development of pain or whether catastrophizing generally increases after a pain condition develops. While situational catastrophizing scores were highest in the arthritis patients, associations between situational catastrophizing and dispositional PCS scores were most strongly related among TMD patients. Thus, our original hypothesis that measures of situational and dispositional catastrophizing would be more strongly inter-correlated with one another in the patient groups was only partially confirmed. Although the cause of this variability is unknown, one potential explanation may be differences in the degree or nature of clinical pain experiences in the two groups. While ethnicity was controlled within the analyses, ethnic differences within the samples may represent another potential explanatory factor.

While bi-directional associations between catastrophizing and nociceptive processing have been proposed,⁵⁰ the mechanisms underlying catastrophizing’s effects on pain are yet unknown. One theory proposes that catastrophizing amplifies pain processing in the central

nervous system⁴⁷ by facilitating sensitization and/or interfering with pain inhibition. Our group has shown that situational measures of catastrophizing are associated with increased temporal summation of pain, a primary marker for central nervous system sensitizability associated with endogenous opioid function.²² We have also recently found that situational catastrophizing is associated with greater IL-6 reactivity, suggesting that catastrophizing may influence the pro-inflammatory immune system.²⁰ Some preliminary data may suggest that catastrophizing correlates with a dysregulation of opioid-based pain-control systems.^{24,34,55} Greater catastrophizing has been associated with reduced analgesic benefit of an IV kappa opioid agonist.²⁴ This hypothesis is additionally supported by an experiment in which high-catastrophizing participants demonstrated less effective functioning of endogenous pain-inhibitory systems, measured using a Diffuse Noxious Inhibitory Controls (DNIC) paradigm.⁵⁵ While these studies use indirect measures of the endogenous opioid system, they suggest a link between the use of certain pain-coping techniques and opioid-mediated endogenous pain modulation. Catastrophizing does not appear to engage regulatory mechanisms that alter pain transmission in the spinal cord prior to brain processing, as it has no influence on the nociceptive flexion reflex, a spinally mediated withdrawal reflex.^{27,44} However, catastrophizing predicts increased myocardial contractility after a cold pressor task,¹⁸ influencing the relationship between muscle tension, cardiovascular stress, and pain responses.⁵⁶

Several limitations should be considered when interpreting these findings. First, two datasets were combined in this study in order to compare findings across participant groups. This could be problematic due to minor variations in location and timing of testing. However, the questionnaires and psychophysical tests were the same for all participants. Second, our analysis did not assess fear of pain or other potentially overlapping constructs. A recent study by Hirsh and colleagues,³³ examined dispositional and situational measures of both PCS and CSQ catastrophizing as well as fear of pain in a sample of healthy volunteers undergoing a cold pressor test. They found that pain-related fear was a stronger predictor of cold pressor pain response than either dispositional or situational catastrophizing. While the current assessment did not evaluate fear of pain, considerable evidence has demonstrated that both catastrophizing and fear of pain participate in shaping the pain response.^{32,42} In contrast, other previous evidence suggests that catastrophizing is a stronger predictor than fear of pain in influencing pain responses⁵¹, although this is not consistent across studies.³³ Third, we did not probe participants' dispositional PCS responses to determine what referent they used when indicating how much they "typically" catastrophized. Thus, it is difficult to determine why the correlations between situational and dispositional PCS scores are weak, particularly for the arthritis patients who experience ongoing pain. Fourth, our sample sizes, particularly in the TMD and arthritis groups were small and it is unclear how these findings generalize to other samples of patients with arthritis and TMD or other pain populations. Fifth, we are unable to definitively determine the direction of the association between pain responses and situational catastrophizing in this study. This final point is especially important for future studies of situational catastrophizing in order to begin to establish the causal pathway linking catastrophizing to heightened pain responses. Future studies in this area may benefit from the manipulation of either the degree of pain experienced during laboratory testing (and the impact of this manipulation on situational catastrophizing) or the degree of catastrophizing during a standardized pain experience (and the impact on pain report).

These limitations notwithstanding, the current findings emphasize the importance of assessing catastrophizing related to proximal, specific and known, painful events. Such measures may produce more relevant information, at least when examining laboratory pain responses, particularly with healthy volunteers for whom pain is not a current complaint. The construct of situational catastrophizing is particularly useful when comparing healthy participants to patient groups, as is frequently done in experimental pain studies.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Demographic Information, Cronbach's Alpha for situational catastrophizing and PCS Questionnaires and raw experimental pain scores for each group.

Group	Healthy (n=84)	TMD (n=48)	Arthritis (n=43)
Sex (%female)	38.80%	85.70%	61.90%
Age in years, Mean (SD)	34.00 (14.59)	33.86 (12.03)	54.57 (9.73)
Ethnicity	African American: 24.7%	African American: 10.2%	African American: 34.9%
	Asian American: 17.6%	Asian American: 8.2%	Asian American: 0%
	non-Hispanic white: 51.8%	non-Hispanic white: 77.6%	non-Hispanic white: 51.3%
	Other: 5.9%	Other: 4%	Other: 13.8
Pain Duration	N/A	9.0 (8.1) yrs	8.3 (6.4) yrs
Depression Measure (SD)	BSI(n=45): 0.55 (1.1) BDI(n=40): 2.23 (3.2)	BSI: 2.41 (2.96)	BDI: 8.27 (7.08)
Situational Catastrophizing Mean (SD) [range = 0–24]	4.93 (3.89)	5.21 (3.68)	6.84 (6.40)
PCS Mean (SD) [range = 0–52]	9.53 (8.98)	14.29 (9.20)	15.4 (11.95)
Cronbach's Alpha Situational	0.87	0.81	0.94
Cronbach's Alpha PCS	0.93	0.90	0.95
Heat Pain Threshold Mean (SD) Measured in °C	43.99 (3.87)	43.29 (3.83)	41.74 (4.52)
Pressure Pain Threshold Mean (SD) Measured in Kpascals	441.13 (209.65)	453.84 (236.98)	385.11 (146.16)
Mean Heat Pain Intensity Ratings (SD) Ratings of 49 and 51°C	62.56 (29.96)	58.34 (28.57)	73.06 (23.52)
Mean Cold Pressor Pain Ratings (SD) Ratings of cold pain intensity	71.10 (24.09)	66.42 (23.90)	80.51 (17.39)
After-Sensation Pain Ratings Mean (SD) Rating 15 seconds after 49 and 51°C heat stimuli	9.85 (16.47)	12.05 (17.99)	13.71 (22.94)

Pearson Partial Correlation Matrix (controlling for sex and age) between questionnaire data and experimental pain measures for each participant group.

Table 2

Healthy (n=84)	SCQ	PCS	Depression	Pain Threshold	Supra-Threshold	After Sensation
SCQ	1.0					
PCS	.007	1.0				
Depression	.19	.36**	1.0			
Pain Threshold	-.38**	.15	-.02	1.0		
Supra threshold	.30**	.08	-.004	-.39**	1.0	
After Sensation	.33**	.07	.17	-.25**	.15	1.0
TMD (n=48)	SCQ	PCS	Depression	Pain Threshold	Supra-Threshold	After Sensation
SCQ	1.0					
PCS	.45**	1.0				
Depression	.40*	.28	1.0			
Pain Threshold	-.30*	-.31*	.03	1.0		
Supra threshold	.43**	.43**	.08	-.48**	1.0	
After Sensation	.50**	.46**	.21	-.24	.49**	1.0
Arthritis (n=43)	SCQ	PCS	Depression	Pain Threshold	Supra-Threshold	After Sensation
SCQ	1.0					
PCS	.22	1.0				
Depression	.08	.32*	1.0			
Pain Threshold	-.42**	.01	-.27	1.0		
Supra threshold	.13	.14	.01	-.29	1.0	
After Sensation	.32*	-.10	-.15	-.44**	.28	1.0

Note – the depression measure is a z-scored combination of two different depression inventories and pain measures are z-scored components of experimental pain stimuli.

*
p < .05;

**
p < .01

Table 3**Table 3a. Healthy Participants: Pain Threshold**

Step Variables	β	SE	Beta	<i>t</i>	R^2	ΔR^2	P value for Δ in unstandardized beta coefficient
1. Sex	-.13	0.19	0.20	-0.07			
Age	.02	0.01	-0.16	.37*			
Ethnicity	-0.21	0.19	-0.03	-0.12	0.19	0.19**	
2. Depressive symptoms	-0.04	0.09	-0.44	-.43	0.19	0.002	
3. Standard PCS	0.02	0.01	.19	1.83			
Situational PCS	-0.07	0.02	-0.32	-3.21**	0.32	0.13**	0.01**

Table 3b. Arthritis Participants: Pain Threshold

Step Variables	β	SE	Beta	<i>t</i>	R^2	ΔR^2	P value for Δ in unstandardized beta coefficient
1. Sex	-.22	0.25	-0.14	-0.89			
Age	-0.03	0.01	-0.33	-1.97			
Ethnicity	-0.51	0.27	-0.32	-1.91	0.13	0.13	
2. Depressive symptoms	-0.03	0.02	-0.26	-1.58	0.19	0.06	
3. Standard PCS	0.02	0.01	.23	1.47			
Situational PCS	-0.05	0.02	-0.4	-2.75**	0.35	0.16*	0.008

Table 3c. TMD Participants: Pain Threshold

Step Variables	β	SE	Beta	<i>t</i>	R^2	ΔR^2	P value for Δ in unstandardized beta coefficient
1. Sex	-.31	0.37	-0.13	-0.84			
Age	0.17	0.01	0.23	1.57			
Ethnicity	0.12	0.31	0.06	0.37	0.07	0.07	
2. Depressive symptoms	0.003	0.02	0.03	0.17	0.07	0.001	
3. Standard PCS	-0.03	0.02	-0.28	-1.78			
Situational PCS	-0.07	0.04	-0.3	-1.83	0.25	0.18*	.35

Final Model: $F(6, 76) = 5.99, p = 0.001$

Final Model: $F(6, 35) = 3.09, p = 0.015$

Final Model: $F(6, 41) = 2.26, p = 0.05$

*
p < .05;

**
p < .01

Table 4**Table 4a. Healthy Participants: Supra Threshold ratings**

Step Variables	β	SE	Beta	<i>t</i>	R^2	ΔR^2	P value for Δ in unstandardized beta coefficient
1. Sex	0.21	0.2	0.12	1.07			
Age	-0.004	0.01	-0.07	-0.63			
Ethnicity	0.54	0.19	0.31	2.86*	0.13	0.13	
2. Depressive symptoms	0.01	0.09	0.01	0.09	0.13	0.00	
3. Standard PCS	0.004	0.01	0.04	0.33			
Situational PCS	0.06	0.02	0.26	2.34*	0.19	0.06	0.05

Table 4b. Arthritis Participants: Supra Threshold ratings

Step Variables	β	SE	Beta	<i>t</i>	R^2	ΔR^2	P value for Δ in unstandardized beta coefficient
1. Sex	-0.05	0.2	-0.04	-0.25			
Age	0.002	0.01	0.03	0.16			
Ethnicity	0.01	0.21	0.01	0.07	0.002	0.002	
2. Depressive symptoms	<-0.001	0.02	0.0	-0.002	0.002	0.00	
3. Standard PCS	0.01	0.01	0.14	0.73			
Situational PCS	0.01	0.02	0.11	0.62	0.03	0.03	0.95

Table 4c. TMD Participants: Supra Threshold ratings

Step Variables	β	SE	Beta	<i>t</i>	R^2	ΔR^2	P value for Δ in unstandardized beta coefficient
1. Sex	-0.38	0.39	-0.15	-0.98			
Age	-0.003	0.01	-0.03	-0.23			
Ethnicity	0.48	0.32	0.22	1.49	0.09	0.09	
2. Depressive symptoms	0.01	0.02	0.09	0.59	0.09	0.01	
3. Standard PCS	0.03	0.02	0.3	1.97			
Situational PCS	0.08	0.04	0.33	2.09*	0.3	0.21*	0.4

Final Model: $F(6, 76) = 2.9, p = 0.01$

Final Model: $F(6, 35) = 0.2, p = 0.98$

Final Model: $F(6, 41) = 2.98, p = 0.016$

*
p < .05

Table 5

Table 5a. Healthy Participants: After-sensation

Step Variables	β	SE	Beta	t	R^2	ΔR^2	P value for Δ in unstandardized beta coefficient
1. Sex	0.08	0.24	0.04	0.33			
Age	-0.01	0.01	-0.15	-1.21			
Ethnicity	0.34	0.23	0.17	1.49	0.07	0.07	
2. Depressive symptoms	0.17	0.11	0.17	1.53	0.1	0.03	
3. Standard PCS	0.001	0.01	0.01	0.09			
Situational PCS	0.08	0.03	0.29	2.59*	0.17	0.08*	0.04*

Table 5b. Arthritis Participants: After-sensation

Step Variables	β	SE	Beta	t	R^2	ΔR^2	P value for Δ in unstandardized beta coefficient
1. Sex	0.41	0.31	0.2	1.29			
Age	0.01	0.02	0.07	0.42			
Ethnicity	0.64	0.34	0.33	1.92	0.11	0.11	
2. Depressive symptoms	-0.03	0.02	-0.2	-1.18	0.15	0.03	
3. Standard PCS	-0.02	0.01	-0.17	-1.05			
Situational PCS	0.05	0.02	0.31	2.0*	0.24	0.10	0.14

Table 5c. TMD Participants: After-sensation

Step Variables	β	SE	Beta	t	R^2	ΔR^2	P value for Δ in unstandardized beta coefficient
1. Sex	-0.22	0.39	-0.08	-.57			
Age	0.01	0.01	0.08	0.62			
Ethnicity	1.05	0.33	0.44	3.22**	0.22	0.22*	
2. Depressive symptoms	0.03	0.02	0.23	1.69	0.27	0.05	
3. Standard PCS	0.02	0.02	0.22	1.59			
Situational PCS	0.08	0.04	0.31	2.12*	0.42	0.15**	0.18

Final Model: $F(6, 76) = 2.49, p = 0.03$

Final Model: $F(6, 35) = 1.88, p = 0.11$

Final Model: $F(6, 40) = 4.83, p = 0.001$

* $p < .05$;

** $p < .01$