Disease-Related, Nondisease-Related, and Situational Catastrophizing in Sickle Cell Disease and Its Relationship With Pain

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Abstract: Catastrophizing is a potent psychological modulator of pain across several chronic pain populations; yet despite evidence that patients with sickle cell disease (SCD) catastrophize more than patients with other chronic pain conditions, previous research indicates that catastrophizing is not related to sickle cell pain after controlling for relevant covariates such as depression. Recent research suggests that pain-related catastrophizing should be assessed across pain contexts (eg, dispositional and situational). In this study, we measured disease-specific, general non-disease-related, and situational catastrophizing and assessed the relationship between these contextual dimensions of catastrophizing and laboratory and clinical pain among patients with SCD. Results revealed differential catastrophizing across pain contexts, with patients reporting greater catastrophizing about SCD-specific pain compared with non-SCD pain and laboratory pain. SCD-specific and non-SCD catastrophizing were associated with clinical pain outcomes, and situational catastrophizing with markers of central sensitization and laboratory pain. Further examination of the time course of laboratory responses revealed that increases in situational catastrophizing were associated with subsequent increases in laboratory pain sensitivity. Taken together, results show the relevance of catastrophizing in understanding pain in SCD, and suggest that context-specific anchors may be beneficial in predicting different aspects of the pain experience (eg, chronic pain, pain sensitization).

Perspective: Patients with SCD report greater catastrophizing about sickle cell-specific pain relative to other pains. Disease-specific and non-disease-related pain catastrophizing were associated with clinical pain, and situational catastrophizing predictive of subsequent laboratory pain. Evaluation of context-specific catastrophizing may more accurately predict different aspects of the pain experience.

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Key words: Sickle cell disease, pain catastrophizing, quantitative sensory testing, chronic pain, central sensitization.

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Catastrophizing is a potent psychological modulator of pain across several chronic pain populations. Pain catastrophizing involves exaggerated negative affective and cognitive appraisals of pain, such as rumination, helplessness, and magnification of pain. Current evidence suggests a direct link between pain catastrophizing and physiological pain facilitation processes, such that increased catastrophizing is associated with centrally mediated pain enhancement (ie, decreased conditioned pain modulation (CPM) and increased temporal summation) and enhanced pain-related brain response, and targeted therapeutic
Catastrophizing and Pain in Sickle Cell Disease

Methods

Participants

Eighty-one volunteers (57 female, 78 African American/black, 3 multiracial) with SCD participated in this study (Table 1) as part of an ongoing larger study on pain in SCD (additional data on these participants has been published elsewhere2,5,22,23). Patients were recruited from the Sickle Cell Center for Adults at Johns Hopkins Hospital as well as through advertisements. Interested volunteers were included if they were 18 years of age or older, had a formal diagnosis of SCD (hemoglobinopathy genotype [Hb SS, Hb SC, Hb S/β-thalassemia]), and were receiving a stable dose (if any) of nonsteroidal anti-inflammatory drugs, acetaminophen, or opioids 1 month before pain testing. Exclusion criteria included current alcohol or substance abuse/dependence and significant psychological impairment that would preclude completion of study measures (eg, dementia, cognitive impairment, unstable psychiatric illness). Participants were free of any major medical conditions other than SCD and none reported having other chronic pain.

Procedure

Pain testing sessions were scheduled on days when patients were experiencing SCD pain at the level of 5 or lower on a 0 to 10 pain rating scale and when they had not had a vaso-occlusive crisis in the past 3 weeks. First, informed written consent was obtained from each participant. After the consent process, participants completed the surveys described in the Survey Measures section, and a psychophysical pain testing battery lasting approximately 1 hour. Participants were allowed to stop or refuse any procedure at any time. This study was approved by the Johns Hopkins University School of Medicine institutional review board.

Table 1. Demographic Characteristics

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<th>Characteristic</th>
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<td>Depression</td>
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</tr>
<tr>
<td>Neuroticism</td>
<td>4.88 (.78)</td>
</tr>
</tbody>
</table>

NOTE. Data (except for sample size, N) are reported as mean (SD) or percent.
Survey Measures

Catastrophizing

To allow for direct comparison of the 3 catastrophizing measures, scores are reported and analyzed as average, rather than summed, scores. To allow for comparison with previous research on dispositional catastrophizing, summed scores are also reported in Table 2.

Non-SCD catastrophizing. Non-SCD catastrophizing was assessed using the Pain Catastrophizing Scale (PCS)\(^3\) that measures trait-like exaggerated negative cognitive and affective responses to pain. The PCS consists of 13 items rated on a 5-point scale (0 = not at all to 4 = all the time) with higher scores indicating greater pain catastrophizing. In the current study, we asked patients to base their answers on painful experiences other than sickle cell pain (“Please answer based on how you feel regarding painful situations other than sickle cell pain”). Total score was calculated as the average of all responses. Similar to previous reports,\(^3,30\) internal reliability of this measure was found in our sample (\(\alpha = .92\)).

SCD-specific catastrophizing. Motivated by patients’ self-disclosure of differential responses to pain in general and sickle cell pain, we modified the PCS such that patients were directed to respond to the 13 items on the basis of their sickle cell pain specifically (“Please answer based on how you feel about your sickle cell pain”). All other aspects of the instructions and questionnaire were identical to the PCS. Total score was calculated as the average of all responses. Similar to previous reports,\(^3,30\) internal reliability of this measure was found in our sample (\(\alpha = .93\)).

Situational catastrophizing. The situational catastrophizing scale\(^4\) was assessed at 4 points during the pain testing session: after heat pain threshold (HPTh) and pressure pain threshold (PPTh), thermal temporal summation (TTS), mechanical temporal summation (MTS), and CPM with hot water. The situational catastrophizing scale consists of 6 items modified from the PCS (eg, “I worried about when it would end”) and was scored on the same 5-point scale as the PCS. The situational catastrophizing scale assesses responses directly after administration of noxious stimulation and directs participants to reference these procedures while answering (“please indicate the degree to which you had these thoughts and feelings during this pain-testing session”). A total score was calculated as the average of responses after each pain testing procedure for use in the primary analyses. Secondary analyses of the relationship between situational catastrophizing at the end of one procedure on pain sensitivity during subsequent procedures were run using individual procedure-specific situational catastrophizing scores. Internal reliability (\(\alpha\)) was high within the scale across modalities (\(\alpha\): HPTh/PPTh = .84, TTS = .93, MTS = .94, CPM = .94).

Clinical Pain Severity and Pain-Related Interference

Clinical pain severity was calculated as the average of 4 self-reported pain ratings (ie, current pain and worst, least, and average pain over the past week) using an 11-point scale (0 = no pain to 10 = pain as bad as it could be; \(\alpha = .87\)).

Clinical pain interference was assessed using the 10-item extended\(^18,32\) Brief Pain Inventory (BPI)\(^8\) pain interference subscale. This extended subscale assesses functional interference caused by pain during the past week in the areas of mood, sleep, relationships with others, and various daily activities and is also scored on an 11-point scale (0 = does not interfere to 10 = completely interferes; \(\alpha = .97\)).

Depression

Depressive symptomatology was assessed as a potential confounding covariate of catastrophizing, and was measured using the Center for Epidemiological Studies Depression Scale (CES-D),\(^25\) which assesses frequency of 20 feelings and experiences during the past week on a 4-point scale (0 = rarely/less than 1 day to 3 = most of the time/5–7 days). Total score was calculated as the sum of responses (\(\alpha = .78\)). Although this screening measure is not intended as a diagnostic tool, a score of \(\geq 16\) corresponds to “clinically significant” depressive symptomatology.

Neuroticism

Because of its known association with pain catastrophizing in other populations,\(^7,30\) neuroticism was also assessed as a potential confounding variable and was measured using the neuroticism subscale of the Big Five Inventory (BFI).\(^19\) The BFI assesses characteristic behaviors and emotions associated with 5 personality traits (including extraversion, conscientiousness, openness to experience, agreeableness, and neuroticism) by asking participants to rate the extent to which they believe each item is self-characteristic on a 5-point scale (1 = disagree strongly to 5 = agree strongly). The neuroticism subscale is calculated as the average of the 8 neuroticism items on the BFI (\(\alpha = .77\)).

Demographic Characteristics

Participants also provided demographic information (including age, sex, race, and education) and completed a health history form.
Psychophysical Pain Testing

The pain testing procedures conducted in our study have been used extensively by our laboratory and others across chronic pain populations and have recently been shown to be safe and effective in testing pain sensitivity among patients with SCD.5,10

Pain Ratings

Pain ratings during each of the pain testing procedures were assessed using a numerical rating scale ranging from 0 (no pain) to 100 (worst pain imaginable).

Thermal Stimuli

Heat stimuli were delivered to participants’ dominant ventral forearm using a contact heat-evoked potential stimulator (CHEPS; Medoc Ltd, Ramat Yishai, Israel) system, a peltier element-based stimulator with a 9 cm² rapidly heating/cooling probe.

HPTh/Tolerance

HPTh and tolerance (HPTo) are reported in degrees Celsius and were calculated as the average of two corresponding trials administered using an ascending method of limits paradigm. On each trial, the contact thermode gradually increased in temperature, from a baseline of 30°C at a .5°C/second rate of increase, until the subject indicated via button press that the stimulus first felt painful (HPTh) or when the stimulus became intolerable (HPTo). Between trials, the thermode was moved up the arm slightly to avoid overlapping stimulation sites.

PPTh

PPTh was measured using an electronic algometer (SBmedic, Solna, Sweden) with a 1-cm² probe covered with a 1-mm polypropylene material.17 Pressure was applied to the muscle belly and increased steadily at a rate of 50 kPa/s until the subject verbally indicated the pressure first felt painful (PPTh). Thresholds were assessed twice at each of 4 body sites, bilaterally (trapezius muscle, interphalangeal joint of the thumb, the wrist, and the middle of the quadriceps insertion point), for a total of 16 PPTh assessments. A minimum 1-minute interval was maintained between applications at the same site. An average PPTh was calculated for each site.

TTS

Ten repetitive thermal stimuli were applied rapidly in a series of identical pulses. Pain ratings were obtained for each pulse. The thermode remained in a fixed position during administration of each sequence of 10 heat pulses (.5 seconds each, with a 2.5-second interpulse interval). A practice trial with pulses at participants’ warmth detection threshold was conducted to familiarize participants with the procedure. Experimental trials were conducted at tailored temperatures (HPTh and HPTh +2°C), and at a standard temperature of 45°C. The thermode was moved slightly between trials to avoid overlapping stimulation sites. TTS was calculated as the difference between the maximum and first pain rating for each temperature. TTS after-sensations were assessed 15 seconds after the final stimulus at each temperature. An average TTS after-sensation score was calculated across trials.

MTS

MTS was calculated as the difference between pain ratings in response to a single punctuate stimulus compared with a sequence of 10 identical punctuate stimuli. Weighted pinprick stimulators with a flat contact area of .2 mm diameter were used to deliver stimuli at a 1 per second rate to the middle phalange of the middle finger. A practice trial was conducted with a stimulator that produced 32 mN force. Experimental trials were conducted at 128 mN and 256 mN.

CPM and Hot Water Procedures

CPM was assessed using pressure applied to the trapezius as the test stimulus, and hot water bath as the conditioning stimulus. First, PPTh was again assessed (separate from PPTh mentioned previously) twice at the nondominant trapezius. The dominant hand was then submerged in a hot water bath for 20 seconds, at which time PPTh was reassessed (immediately before hand removal). If participants removed their hands before 20 seconds, PPTh was assessed immediately upon withdrawal. CPM was calculated as the difference between the PPThs during and before water submersion. This procedure was repeated a second time, and final scores reflect an average of both trials.

The hot water temperature used for CPM was determined early in the pain testing session as the temperature at which patients rate their pain in the range of 60 to 70 out of 100 after 20 seconds of hand submersion.

Hot water after-sensations were assessed at 30 seconds and 1 minute after hand withdrawal. Final scores were calculated as an average of after-sensations at both time points.

Order of Testing and Catastrophizing Measurements

HPTh/HPTo and PPThs were randomized, but always conducted at the beginning of the psychophysical pain testing session, followed by either MTS or TTS (also randomized), with CPM/hot water procedures occurring last. Situational catastrophizing was measured after thresholds (once after the thermal and pressure threshold testing), once after each of the temporal summation series (which were organized into the proper temporal order for cross-lag panel analyses purposes), and once after the CPM/hot water procedures.

Data Reduction

To reduce the number of comparisons examined, laboratory pain was quantified using 2 a priori defined composite summary scores: a central sensitization index and a quantitative sensory testing (QST) index. Category determination was based on previous work1,5,14,34 and was confirmed using factor analysis.
Z-scores were first created for each variable and reverse scored where appropriate (by multiplying by $-1$) such that higher scores correspond to greater pain sensitivity.

**Central Sensitization Index**

The central sensitization index was calculated as the average of the following 7 individually z-scored values: MTS (128 mN and 256 mN), TTS (HPTH, HPTH +2°C, and 45°C), and the after-sensations to TTS and hot water. Reliability analysis indicated internal consistency of this index ($\alpha = .74$).

**QST Index**

The QST index was calculated as the average of the remaining psychophysical pain measures not included in the central sensitization index (ie, heat and pressure thresholds, heat pain tolerance, hot water temperature, hot water pain rating and withdrawal time, and CPM). Reliability analysis indicated internal consistency of this index ($\alpha = .75$).

**Data Analyses**

All data analyses were conducted using SPSS (version 21; IBM Corp, Armonk, NY).

**Correlation and Multivariate Regression Analyses**

First, the relationship between each of the 3 catastrophizing measures and age, sex, education, depression, and neuroticism were assessed. Second, intercorrelations among the 3 catastrophizing variables were measured. Third, the univariate relationships between all 3 catastrophizing measures and clinical and psychophysical pain responses were evaluated. Fourth, we investigated whether significant catastrophizing–pain relationships survived correction for covariates using multivariate hierarchical regression models. Demographic characteristics (eg, age, sex, education) were entered in the first step, depression and/or neuroticism (if correlated) in the following step(s), and the correlated catastrophizing measure in the final step of the model. We chose the conservative approach of including correlated covariates for any catastrophizing measure in all models so as to not differentially parse the variance across models.

**Cross-Lagged Panel Analyses**

Finally, we further interrogated the situational catastrophizing data to examine whether or not situational catastrophizing during one psychophysical pain testing procedure was associated with increased pain response to a subsequent procedure. We used a cross-lagged panel analysis design in which standardized residualized change scores were calculated as an index of change between psychophysical tests. Situational catastrophizing data were collected first after heat and pressure threshold (T1), next after either TTS or MTS (T2), which were presented in randomized order, then the remaining temporal summation procedure (T3), and finally after CPM (T4). Average psychophysical pain sensitivity was calculated for each of these time points (eg, T1 pain was calculated as the average standardized pain threshold for HPTH as well as PPTH at all sites). These 4 time points were the only ones assessed in the cross-lagged panel analysis. Hierarchical regression was used to determine whether changes in situational catastrophizing (eg, between T1 and T2) predicted subsequent changes in psychophysical pain sensitivity (eg, between T2 and T3) controlling for autocorrelations (eg, correlations with changes in situational catastrophizing at different time points) and synchronous correlations (eg, correlations with change in pain during the same time period). The reverse relationship examining whether changes in pain sensitivity precede changes in catastrophizing was also examined. This resulted in 4 models predicting the change in pain or situational catastrophizing between T2/T3 and T3/T4.

**Missing Data**

Participants were not excluded because of partially missing data and most participants ($n = 73$; 90%) completed every component of all procedures. Missing psychophysical data are due to either participant choice to discontinue a certain procedure, or the participant rating the maximum (100) before completion of a procedure. Composite scores were calculated using all available data for each participant. Missing values were left missing and not imputed. The central sensitization score could not be calculated for 1 participant ($n = 80$), however, the QST index was calculated for all participants ($n = 81$). Missing survey data values were due to participants leaving the questionnaire blank. All participants ($n = 81$) completed the non-SCD catastrophizing questionnaire, 78 completed the SCD-specific questionnaire, and 80 completed situational questionnaires.

**Results**

Non-SCD catastrophizing was negatively associated with patient education ($R = -.26, P = .02$), positively associated with depression ($R = .27, P < .02$), and a sex difference was found such that male patients reported greater non-SCD catastrophizing ($mean = 1.34, SD = .89$) compared with female patients ($mean = .88, SD = .71$, $t_{79} = 2.43, P < .02$). Situational catastrophizing was significantly ($R = .25, P = .03$), and SCD-specific catastrophizing marginally ($R = .20, P = .09$), associated with depression, but neither was related to demographic variables. None of the catastrophizing measures were associated with patient age or neuroticism. Thus, sex, education, and depression were used as covariates in subsequent multivariate models.

Participant responses to the 3 catastrophizing measures differed, suggesting these measures are at least partially independent (Table 2). Patients reported greater SCD-specific catastrophizing ($mean = 2.05, SD = 1.03$) compared with non-SCD catastrophizing ($mean = 1.00, SD = .78, t_{77} = 8.70, P < .001$) and situational catastrophizing ($mean = .90, SD = .81, t_{76} = 9.14, P < .001$). SCD-specific catastrophizing significantly correlated
with non-SCD and situational catastrophizing. Situa-
tional and non-SCD catastrophizing were not signifi-
cantly different in this sample and were marginally
correlated.

Non-SCD and SCD-specific catastrophizing scores
were significantly correlated with clinical pain
severity and interference, but not the QST Index or
the Central Sensitivity Index (Table 3). Situational cata-
lossizing was significantly correlated with both
psychophysical pain indices as well as clinical pain
severity and was marginally associated with clinical
pain interference.

Hierarchical multiple regression (Table 4) revealed
that situational catastrophizing remained a significant
predictor of both psychophysical pain indices even after
controlling for education, sex, and depression. Sita-
tional catastrophizing was not associated with either
clinical pain or pain interference when demographic
characteristics and depression were covaried. SCD-
specific catastrophizing remained marginally associated
with clinical pain severity and significantly associated
with clinical pain interference, even after controlling
for the effects of education, sex, and depression. Non-
SCD catastrophizing remained significantly associated
with clinical pain severity and interference in these
models.

Results from the cross-lagged panel analyses (Table 5,
Figure 1) revealed that increases in situational cata-
lossizing during the first period (T1–T2) were posi-
tively associated with increases in pain sensitivity
during the second period (T2–T3). However, change in
situational catastrophizing during the second period
did not significantly predict change in pain sensitivity

### Table 3. Relationship (R) Between Catastrophizing and Pain

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<td>Interference</td>
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Abbreviation: NS, not significant (P > .10).

* P ≤ .05.
| P ≤ .10.
| ** P ≤ .005.

### Table 4. Hierarchical Regression Models

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</table>

Abbreviations: B, unstandardized coefficient; β, standardized beta coefficient.

NOTE. Statistics are presented in sequential fashion, such that the first step includes coefficients when only step 1 is executed. Subsequent steps show adjusted co-
efficients controlling for the predictors entered previous steps. Because all 3 catastrophizing scales were at least marginally associated with clinical pain outcomes, in-
dividual models entering a different scale in step 3 were compared. Multiple catastrophizing measures were not entered into the same model.

* P ≤ .05.
| P ≤ .10.

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Catastrophizing was significantly correlated with both
psychophysical pain indices as well as clinical pain
severity and was marginally associated with clinical
pain interference.

Hierarchical multiple regression (Table 4) revealed
that situational catastrophizing remained a significant
predictor of both psychophysical pain indices even after
controlling for education, sex, and depression. Sita-
tional catastrophizing was not associated with either
clinical pain or pain interference when demographic
characteristics and depression were covaried. SCD-
specific catastrophizing remained marginally associated
with clinical pain severity and significantly associated
with clinical pain interference, even after controlling
for the effects of education, sex, and depression. Non-
SCD catastrophizing remained significantly associated
with clinical pain severity and interference in these
models.

Results from the cross-lagged panel analyses (Table 5,
Figure 1) revealed that increases in situational cata-
lossizing during the first period (T1–T2) were posi-
tively associated with increases in pain sensitivity
during the second period (T2–T3). However, change in
situational catastrophizing during the second period
did not significantly predict change in pain sensitivity

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during the third period (T3–T4). The reverse model revealed that increased pain during the first period also showed a tendency toward predicting later change in situational catastrophizing during the second period. Changes during the second period did not predict changes during the third. These effects remained when depression was controlled for in the models.

**Discussion**

In this study we showed that catastrophizing in SCD varies across pain contexts, with patients reporting greater catastrophizing about SCD pain compared with general non-SCD pain experiences and laboratory pain. Disease-specific and general non-disease-related...
catastrophizing are associated with clinical pain outcomes, whereas situational catastrophizing is associated with sensitization to laboratory pain. Furthermore, our results suggest that situational catastrophizing and pain sensitivity may mutually facilitate each other in SCD, such that increases in catastrophizing predict later increases in pain sensitivity and early increases in pain sensitivity predict slight (marginal) increases in catastrophizing.

Although SCD patients’ level of non-SCD catastrophizing was similar to, or lower than, average PCS scores in other chronic pain populations, SCD-specific catastrophizing scores were higher than PCS averages. This suggests that previous findings that SCD patients tend to catastrophize more than other chronic pain patients may be driven by a number of disease-related factors that deserve future investigation. The nature of SCD pain as related to a lifelong and life-threatening disease may promote disease-related catastrophizing. Furthermore, the severe episodic and chronic pain experienced by SCD patients is often undertreated, which reasonably may further exacerbate SCD-specific pain ruminations, magnification, and helplessness. One consideration for future studies is that disease-specific pain may be more prevalent and salient, and therefore may lead to greater disease-specific catastrophizing. Future studies are also needed to tease apart growing evidence of greater catastrophizing in SCD patients from demonstrated racial differences in catastrophizing.

Despite differential patterns of SCD-specific and non-SCD pain catastrophizing responses, both measures were associated with clinical pain severity and interference. This may reflect the “trait” component of catastrophizing that has been conceptualized as not being disease-specific but a general response to many types of pain. The pattern of findings suggests that catastrophic cognitions in this population are not isolated to disease-related pain, and that such cognitions in response to pain unrelated to the disease may in turn be associated with worsened disease-related pain and the effect of pain on daily activities. The relationships between SCD-specific and non-SCD catastrophizing with clinical pain severity and pain interference largely remained even after controlling for known covariates of pain-related catastrophizing including sex, education, and depression (although the SCD-specific catastrophizing/pain severity correlation was marginal). Depression is common in SCD, and is associated with chronic pain and catastrophizing. Citero et al reported that the relationship between general catastrophizing and daily pain in SCD was not significant when controlling for depression. In our study, although we found a relationship between depression and our 3 catastrophizing measures, we found that the association between catastrophizing and pain is independent of the effects of depression. Important differences between this and the previous study are the instruments used to assess the primary outcomes of interest: catastrophizing and pain. Citero and colleagues used the catastrophizing subscale of the Coping Strategies Questionnaire (CSQ) and we used a slightly modified version of the PCS (referencing non-SCD pain specifically). Although the CSQ-catastrophizing subscale and the PCS are correlated, the CSQ scale includes only the helplessness dimension of pain catastrophizing, whereas the PCS added the components of pain magnification and rumination. Additionally, pain assessment differed between studies (multi-item assessment cross-sectionally vs single-item assessment using daily diaries). Both studies confirm the significance of depressive symptoms and our finding suggests that continued investigation of the role of pain catastrophizing is warranted, particularly as rumination and/or magnification may be important components for sickle cell patients.

Our findings further show that situational catastrophizing is strongly associated with indices of central sensitization and laboratory pain in SCD, but not clinical pain severity or interference after controlling for sex, education, and depression. This pattern suggests that acute catastrophic cognitions may enhance pain sensitivity. Indeed, cross-lagged panel analyses show that changes in situational catastrophizing were associated with subsequent changes in laboratory pain sensitivity, a finding our group has previously shown in healthy control subjects. Changes in catastrophizing precede heightened pain sensitivity. However, unlike our findings among healthy control subjects, these results in SCD suggest that there is a possible bidirectional relationship between situational catastrophizing and pain. Early changes in psychophysical pain sensitivity were associated with marginal increases in subsequent situational catastrophizing, suggesting that situational catastrophizing and pain sensitivity may mutually facilitate each other in SCD. Future work is needed to further explore the temporal dynamics of this relationship. If a mutually facilitatory relationship between situational catastrophizing and pain sensitization exists in SCD, catastrophizing may be especially harmful in this population, triggering a pattern of ever increasing sensitization. Furthermore, in SCD, this pattern may be particularly important for understanding the longitudinal effects of episodic crisis pain. Over years, pain-related catastrophizing may lower the individual patient’s threshold for crisis and/or increase the frequency or severity of crises.

Limitations of this study include that both measures of trait-like catastrophizing (SCD-specific and non-SCD) and clinical pain reports were cross-sectional, limiting causal inferences. We also do not have qualitative data on the specific aspects of these types of pain that patients considered in their ratings. Additionally, in creating the SCD-specific measure, we slightly altered the commonly used dispositional catastrophizing measure to be specific to only non-SCD pain. Although the development of the SCD-specific measure was motivated by spontaneous patient reports, our separation of these 2 targets (SCD pain and non-SCD pain) may encourage separation of these constructs by patients, and should be considered when using with other patient groups. It is likely that patients combine disease-specific and nondisease pain as targets when
research is needed to probe specific mechanisms of sensitization and their temporal aspects that may be influenced by pain catastrophizing.

Conclusions

Taken together, the current findings suggest 1) that sickle cell patients catastrophize most about their SCD pain rather than showing greater catastrophic thinking about pain in general, but that 2) catastrophizing across pain contexts—even about pain not related to SCD—may predict greater clinical pain severity and interference, and that 3) greater situational catastrophizing may influence and be influenced by greater sensitization to pain. Our findings suggest, somewhat contrary to early research on the topic, that catastrophizing may play an important role in the severity and exacerbation of SCD pain, as well as its effect on daily life.

References


