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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.*

Catastrophizing is a potent psychological modulator of pain across several chronic pain populations.[31] 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[24]) and enhanced pain-related brain response,[27] and targeted therapeutic

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reduction in catastrophizing results in decreased clinical pain severity.[28]

Patients with sickle cell disease (SCD) catastrophize more than patients with other chronic pain conditions, perhaps because of the lifelong and life-threatening nature of SCD, and this occurs more during periods of crisis relative to noncrisis.[16] However, this elevated level of catastrophizing is not associated with heightened crisis or non-crisis pain intensity, distress, or pain-related interference when symptoms of depression are controlled.[7] Therefore, others have concluded that inferences about catastrophizing and pain drawn from other populations should not be translated to sickle cell pain.[7]

Recent work shows substantial variability in the relationship between pain and catastrophizing.[3] One possible explanation for the lack of statistical association between pain and catastrophizing in SCD may be the specificity and proximity of measures of catastrophizing to specific pain experiences. Patients with SCD regularly experience a variety of different pains including severe episodic pain during periods of vaso-occlusive crisis (sudden onsets of acute pain typically lasting 4–7 days), neuropathic pain that includes hyperalgesia and allodynia, and other types of chronic pain with or without an identifiable pathology.[9] However, traditional assessments of catastrophizing measure dispositional responses to pain in general, and do not allow for differentiation of different types of pain.[20] Others have argued that catastrophizing in response to specific stimuli may more accurately predict corresponding stimulus-related pain.[24] Recent studies have shown that measurement of situational catastrophizing (ie, catastrophizing related to evoked laboratory pain) is a better predictor of laboratory-induced pain than dispositional catastrophizing.[3] However, unlike dispositional catastrophizing, situational catastrophizing has an inconsistent relationship with clinical pain. Studies have shown that situational catastrophizing in response to evoked pain is associated with laboratory pain in healthy adults[6] and post-elective surgical pain in healthy male adults,[13] but not with later clinical pain in fibromyalgia.[4] Furthermore, situational catastrophizing can be poorly correlated with general dispositional catastrophizing, suggesting that an individual's engagement of catastrophic thinking toward pain may differ across contexts.[3,20,24]

In this report we present data on a further refinement of the measurement of pain catastrophizing, introducing a disease-specific approach to measuring catastrophizing and comparing the relationship between disease-specific, general non-disease-related, and situational pain catastrophizing in patients with SCD. On the basis of previous evidence, we hypothesized that general non-SCD catastrophizing would not be associated with clinical pain in SCD. Rather, we hypothesized that specifically anchored measures of catastrophizing would be associated with corresponding pain dimensions. We hypothesized that situational catastrophizing would be associated with laboratory pain intensity, and SCD-specific catastrophizing would be associated with clinical pain. Additionally, we examined changes in situational catastrophizing elicited during laboratory pain testing to explore the potential effects of acute changes in catastrophizing on pain sensitivity in a chronic pain population. We hypothesized on the basis of previous studies among healthy control participants, that patients with SCD would show increased psychophysical pain sensitivity associated with increases in situational catastrophizing.

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 elsewhere[2,5,22,23]). 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

Characteristic	Value
N	81
Age	38.57 (11.88)
Female	70.37
Highest education	
High school or less	18.52
Some college	43.21
Bachelor's degree	27.16
Graduate degree	11.11
Depression	14.59 (10.83)
Neuroticism	4.88 (.78)

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

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