Original Experimental

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Exploration of the trait-activation model of pain catastrophizing in Native Americans: results from the Oklahoma Study of Native American pain risk (OK-SNAP)

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Abstract

Objectives: Native Americans (NAs) have the highest prevalence of chronic pain of any racial/ethnic group. This issue has received little attention from the scientific community. One factor that may contribute to racial pain disparities is pain catastrophizing. Pain catastrophizing is a construct related to negative pain outcomes in persons with/without chronic pain. It has been suggested that the relationship between trait catastrophizing and pain is mediated by situation-specific (state) catastrophizing. The present study has 2 aims: (1) to investigate whether state pain catastrophizing mediates the relationship between trait catastrophizing and experimental pain (e.g., cold, ischemic, heat and electric tolerance), and (2) to investigate whether this relationship is stronger for NAs.

Methods: 145 non-Hispanic Whites (NHWs) and 137 NAs completed the study. Bootstrapped indirect effects were calculated for 4 unmoderated and 8 moderated mediation models (4 models with path *a* moderated and 4 with path *b*).

Results: Consistent with trait-activation theory, significant indirect effects indicated a tendency for trait catastrophizing to be associated with greater state catastrophizing which in turn is associated with reduced pain tolerance during tonic cold ($a \times b$=−0.158) and ischemia stimuli ($a \times b$=−0.126), but not during phasic electric and heat stimuli. Moderation was only noted for the prediction of cold tolerance (path a). Contrary to expectations, the indirect path was stronger for NHWs ($a \times b$ for NHW=−.142).

Conclusions: Together, these findings suggest that state catastrophizing mediates the relationship between trait catastrophizing and some measures of pain tolerance but this indirect effect was non-significant for NAs.

Keywords: catastrophizing; clinical health psychology; coping; diversity; pain.

Introduction

Native Americans (NA) have the highest rates of chronic pain of any U.S. racial/ethnic group but have received little scientific attention [1–3]. Pain catastrophizing is a cognitive-emotional process characterized by pain rumination, magnification and helplessness, and is among the strongest psychosocial predictors of pain and pain-related sequelae [4–9]. Recent results from the Oklahoma Study of Native American Pain Risk (OK-SNAP) found that NAs reported greater state pain catastrophizing in response to painful stimuli than non-Hispanic Whites (NHWs), but no difference in dispositional catastrophizing [10–12]. Hence, it is important understand the role of pain catastrophizing in hyperalgesia among NAs (i.e., reduced pain tolerance - a risk factor for chronic pain) [13].

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The Cultural Cognitive-Affective Processing System (C-CAPS [14, 15]) is a theory developed to understand the high interindividual variability in the link between traits and behaviors. The system suggests that situations differentially activate a dynamic array of interacting mediating units (e.g., beliefs, values) that together elicit a behavioral response. Culture – socially shared beliefs, customs, values – can mold mediating units and produce similar behavioral responses in a group of people while still allowing for intraindividual variability.

In line with C-CAPS, it has been proposed that pain catastrophizing is a trait-like disposition that must be activated for it to produce its hyperalgesic effects [9, 16]. This hypothesis draws a distinction between dispositional (trait) pain catastrophizing that assesses a person's catastrophic thoughts/emotions across past painful situations, vs. situation-specific (state) pain catastrophizing that refers to thoughts/emotions that happen during, and in response to, a painful event. To support this, state catastrophizing is a stronger predictor of experimental pain outcomes than trait-like catastrophizing [17, 18].

To date, little work has been done to test a trait-activation model of pain catastrophizing [9, 14] in which trait pain catastrophizing acts as a predisposition that promotes state catastrophizing in the moment to enhance pain. To our knowledge, all of studies assessing both dispositional and situational pain catastrophizing and experimental pain have focused on bivariate relationships between the three variables and not a model explaining their relationship [17, 19].

Moreover, there may be important individual differences in the relationship between trait and state catastrophizing and/or the relationship between state catastrophizing and pain processing that might moderate pain catastrophizing's hyperalgesic effects. Moderators of the trait-state relationship (path a) would mean that people differ in the extent of trait-activation, whereas moderators of the state catastrophizing-pain relationship (path b) would mean that people differ in the extent that state catastrophizing promotes hyperalgesia (see Figure 1). Given the strong relationship between pain catastrophizing and pain exacerbation, it is imperative to fully understand its conceptual underpinning and its differential activation across groups that are overrepresented with pain conditions (e.g., NAs).

Thus, the present ancillary analyses from OK-SNAP has 2 aims: (1) to investigate whether state pain catastrophizing mediates the relationship between trait-like catastrophizing and pain tolerance (i.e., trait-activation model of catastrophizing), and (2) to investigate whether this mediated relationship is moderated by race/ethnicity (i.e., moderated mediation). To achieve these aims, pain catastrophizing (state and trait) was recorded, and pain tolerance was measured from four stimulus modalities (cold, ischemic, heat, and electric). Since we previously demonstrated the NAs report higher levels of state catastrophizing [10], we hypothesize that NAs would show a stronger positive state-trait pain catastrophizing association, and a stronger negative state catastrophizing-pain tolerance relationship than NHWs.

Materials and methods

Participants

The parent study, OK-SNAP, had two primary aims: (1) to identify pain processing differences between NAs and NHWs that contribute to the NA pain disparity, and (2) to identify individual differences variables

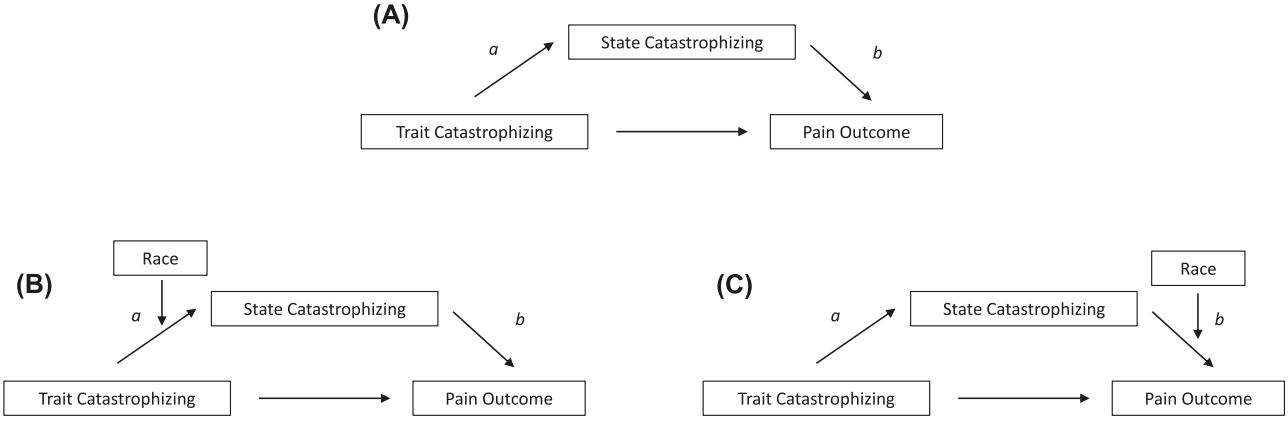

Figure 1: Mediation models. (A) Mediation model with no moderators (PROCESS model 4). (B) Mediation model with race as a moderator on the *a* path (PROCESS model 7). (C) Mediation model with race as a moderator on the *b path* (PROCESS model 14).

(e.g., catastrophizing) that contribute to the NA pain disparity (see Supplemental Table 1 for OK-SNAP manuscripts). NA and NHW healthy, pain-free participants were recruited so that pain amplifying processes could be determined before the onset of chronic pain when access to treatment or disparities in chronic pain severity might confound the identification of risk factors. Participants were recruited using newspapers ads (tribal and non-tribal), fliers, personal communications, and online strategies (e.g., Facebook, Craigslist, and email announcements).

Exclusion criteria were: (1) <18 years old, (2) history of cardiovascular, neuroendocrine, musculoskeletal, neurological disorders, and/or chronic pain, (3) BMI≥35, (4) use of central acting medications (e.g., anti-depressants, anxiolytic, analgesic, stimulant, and anti-hypertensive medication), (5) current psychotic symptoms (assessed by Psychosis Screening Questionnaire [20]), (6) problems with substance use, and/or (7) an inability to read/speak English. Data collection occurred between March 2014 and October 2018.

To qualify as part of the Native American group, individuals had to present either a Certificate of Degree of Indian Blood (CDIB) card or a tribal membership card. To respect the confidentiality of tribes, tribal affiliation is not reported, but most NAs represent tribal nations predominately from the southern plains and eastern Oklahoma tribes. The study was approved by Institutional Review Boards of The University of Tulsa (IRB#: 1016796/REC#: 13-67R1), Cherokee Nation (IRB#: Oklahoma Study of Native American Pain Risk), and Indian Health Service Oklahoma City Area Office (IRB#: P-15-07-OK OK-SNAP). Participants were given an overview of all procedures and informed they could withdraw at any time. All participants provided verbal and written informed consent prior to enrollment and inclusion/exclusion determination. Participants received a $100 honorarium for the completion of each testing day (or $10/h of non-completed days).

General overview of procedures/testing

OK-SNAP data were gathered over 2 days. For a full overview of procedures, the interested reader is referred to our prior paper [21]. All four pain tasks for the current study were assessed on the same day. Electric and heat tolerance were assessed earlier in the day (order counterbalanced), whereas cold pressor tolerance and ischemia tolerance were measured later in the day (order counterbalanced). Breaks were provided between tasks to avoid sensitization and fatigue. Trait pain catastrophizing was assessed at the beginning of the first day of testing. State catastrophizing was assessed immediately after each tolerance task.

Apparatus

Questionnaires were administered by a computer with dual monitor capacity and A/D board (PCI-6071E; National Instruments, Austin, TX). Custom built LabVIEW software (National Instruments) was used to control timing of the experimental protocol and all off-line data reduction. One computer monitor was used by the experimenter to monitor signals and experimental timing, whereas the second monitor was used by the participant to complete questionnaires and to make ratings of stimuli. Testing was completed in a sound attenuated and electrically shielded testing chamber, and participants were monitored from an adjacent control room via a video camera connected to a flat panel monitor. Participants wore sound attenuating headphones that allowed them to hear the experimenter.

Electric tolerance

To assess electric tolerance, a bipolar electrode (Nicolet; 30 mm inter-electrode distance) was placed over the retromalleolar surface of the left ankle and filled with conductive gel (Grass Technologies, West Warwick, RI; EC60). Prior to any sensor placement the skin was cleaned using isopropyl alcohol and exfoliated using an exfoliation cream (Nuprep; Weaver and Company, Aurora, CO) in order to reduce skin impedance below 5 kΩ. Stimulations were delivered by a Digitimer isolated, constant current stimulator (DS7A; Hertfordshire, England). Each stimulus was a train of five 1 ms rectangular wave pulses with a 3 ms inter-pulse interval (250 Hz); however, the train was always experienced as a single stimulation. Electric pain tolerance was assessed using a single ascending staircase of stimulations that started at 0-mA and increased in 2-mA steps until the participant rated a stimulus as maximum tolerable pain on an electronic visual analog scale (VAS) that ranged from “no pain” to “maximum tolerable pain” [22]. The maximum stimulation intensity was set at 50-mA to ensure safety.

Heat pain tolerance

Heat stimuli were generated using a Medoc (Haifa, Israel) Pathway device with a Contact Heat Evoked Potential Stimulator (CHEPS) thermode. The thermode was attached to the volar forearm of participant’s non-dominant hand using a Velcro strap. Heat pain tolerance was each assessed 4 times after an initial practice trial. Each trial started from a baseline of 32 °C and warmed at a rate of 0.5 °C/s until the participant made a button press. Participants were told to push the button when the heat became intolerable. To avoid sensitization, the thermode was moved slightly between trials. Heat pain tolerance was defined as the average of the four trials [23]. The maximum intensity of the heat stimulus was set to 51 °C.

Cold pressor tolerance

To assess cold tolerance, participants were asked to submerge their hand and forearm into a circulating water bath (Thermo Fischer Scientific, Pittsburgh, PA) held at 6 ± 0.1 °C [23–25]. Participants were instructed to keep their fingers spread apart and to place their hand on the bottom of the water tank and keep it there for as long as they could tolerate it. During the task, a computer timed the duration of the hand/arm immersion. Participants made continuous pain ratings of the cold using on the electronic VAS described above. Cold pressor tolerance was defined as the time from when the participant placed their hand in the water until the participant rated the maximum tolerable pain on the VAS. The maximum cold water exposure was set to 5 min, but the participant was not informed of the limit.

Ischemia pain tolerance

To assess ischemia tolerance, a standard forearm tourniquet test was employed [26]. First, participants used their left hand to conduct hand exercises with a dynamometer (Lafayette Hand Dynamometer, Lafayette Instrument Company, IN) at 50% grip strength for

2 min (1x/sec). Immediately after the last exercise, the left arm was raised for 15 s to allow the blood to drain from the forearm and then a blood pressure cuff was inflated to 220 mm/Hg around the left biceps to occlude blood flow to the forearm. During occlusion, participants made continuous pain ratings using the VAS described above. Ischemic tolerance was defined as the time from arm occlusion until the participant made a rating of maximum tolerable pain. The maximum exposure to ischemic pain was set to 20 min, but the participant was not made aware of this limit.

Pain catastrophizing scale

Traditional (Trait) Pain Catastrophizing: The Pain Catastrophizing (PCS) is a reliable and valid 13-item scale that assesses catastrophic thoughts associated with pain [27]. In the current sample (see [28] for details), the PCS has been shown to have construct validity in NAs with an invariant factor structure across NAs and non-Hispanic whites (NHWs). Participants made responses on a 5-point scale that ranged from 0 (not at all) to 4 (all of the time). Higher scores are indicative of greater tendency for pain catastrophizing. To assess trait catastrophizing, traditional instructions were used that ask participants: "Using the scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain." Trait catastrophizing was assessed at the beginning of the study before participants were exposed to any painful stimuli. In the current study, Cronbach's alpha (internal consistency) for the trait version of the PCS was 0.934 (NHW) and 0.925 (NA).

Situation-Specific (State) Pain Catastrophizing: To assess state pain catastrophizing, the PCS was administered, but the instructions were altered to ask about pain catastrophizing specific to each pain task (e.g., "Thinking back to your experience during the [specific pain task], please indicate the degree to which you had these thoughts and feelings"). In the current study, Cronbach's alpha (internal consistency) for the state version of the PCS ranged were (NHW/NA): α=0.945/.950 (cold tolerance), α=0.938/.947 (electric tolerance), α=0.942/.936 (heat tolerance), α=0.942/.962 (ischemia tolerance). The correlations between trait and state catastrophizing ranged from r=0.37 to 0.45.

Data analysis

Prior to analyses, variable distributions were screened using boxplots, histograms, and normality statistics. Those that were skewed were log or square transformed to reduce positive and negative skew, respectively. Next, outliers were identified using Wilcox's MAD-median procedure (using the recommended 2.24 cutoff) and then winsorized by replacing the outlier with the next nearest non-outlier value [29]. Multicollinearity diagnostics of all mediation models were acceptable (Tolerances>0.4)

The present data were analyzed using PROCESS, an SPSS macro by Hayes [30, 31], in order to conduct the moderated mediation models. Importantly, PROCESS constructs bootstrapped confidence intervals for the mediation effects (i.e., indirect effects) [30], such that if the confidence interval for the indirect effect does not contain 0 then the mediation is considered statistically significant. In order to investigate aim 1, a mediation model was used (model 4 in PROCESS). To investigate aim 2, two moderated mediation models were selected. In the first one, race/ethnicity moderated path *a* (Figure 1b; model 7 in PROCESS). In the other, race/ethnicity moderated path *b* (Figure 1c: model 14 in PROCESS). Continuous predictors used to create interaction terms were centered in each model (i.e., trait catastrophizing in PROCESS model 7 and state catastrophizing in PROCESS model 14). Race/ethnicity was coded as NHW=0 and NA=1. The state catastrophizing used in each PROCESS model was specific to the painful stimulus (e.g., state catastrophizing during electric tolerance was used as a mediator in the model predicting electric tolerance).

Results

The final sample

282 individual participants were included in the study. 29 participants quit before completing tolerance measures, and 16 only completed the experimental testing day in which tolerances were not recorded. The final sample for the mediation analyses consisted of 237 individuals. Table 1 notes sample characteristics by race/ethnicity. The results suggest there were more women in the NA group, and NAs had higher BMIs compared to the NHW group. Hence, Sex [Male=0; Female=1], and BMI were added as covariates in all mediation models. The results from correlations (see Table 2) suggests that trait catastrophizing was not significantly correlated with any pain tolerance outcome (average r=0.02) whereas state catastrophizing was significantly negatively correlated to cold (r=−0.34) and ischemic tolerances (r=−0.23).

Evidence for a trait-activation model of pain catastrophizing (aim 1)

To test aim 1, the present study conducted four mediation models in which state catastrophizing was used as a mediator for the relationship between trait catastrophizing and each of the four pain tolerance variables (electric, heat, cold, ischemia). Results are presented in Table 3. In 2 out of the 4 mediation models (cold and ischemia tolerance) the indirect effects are significant. Interestingly, the direct effect (c') was positive in both the models but was only significant in the cold tolerance model.

Evidence for a moderated effect by race/ethnicity (aim 2)

To test aim 2, 2 sets of 4 mediated moderation models were conducted. In set 1, race/ethnicity moderated path *a* (X->M; Figure 1b). In set 2, race/ethnicity moderated path *b* (M->Y; Figure 1c).

Table 1: Participant characteristics by racial/ethnic group.

Continuous variable	Missing data	NHW (n=145)		NA (n=137)		*t*
	n	*M*	SD	*M*	SD	
Age, years	0	28.50	13.48	31.28	13.26	2.79
Body Mass index, kg/m2	7	**24.25**	**3.80**	**26.05**	**4.60**	**1.80**
Categorical variable		**n**	**%**	**n**	**%**	X^2
Sex (female)	0	**68**	**46**	**87**	**63**	**7.85**
Education	2					4.74
<7th grade		1	0.4	1	0.4	
<High school		2	0.7	7	2.5	
High school grad		20	7.1	23	8.2	
Partial college		75	26.8	59	21.1	
College grad		36	69.2	37	13.2	
Graduate/professional school		10	3.6	9	3.2	
Marital status	2					8.72
Single		108	38.6	82	29.3	
Married		23	8.2	29	10.4	
Separated/divorced		11	3.9	14	5.0	
Cohabitating		2	0.7	9	3.2	
Widowed		1	0.4	1	0.4	
Employment	5					3.47
>40 h/week		30	10.8	39	14.1	
<40 h/week		65	23.5	50	18.1	
Retired		5	1.8	3	1.1	
Unemployed		42	15.2	43	15.5	
Income	9					6.76
<$9,999		55	20.1	36	13.2	
$10,000–$14,999		16	5.9	15	5.5	
$15,000–$24,999		16	5.9	19	7.0	
$25,000–$34,999		12	4.4	16	5.9	
$35,000–$49,999		13	4.8	20	7.3	
$50,000–$74,999		9	3.3	10	3.7	
$75,000–$99,999		8	2.9	6	2.2	
$100,000–$149,999		8	2.9	7	2.6	
$150,000–$199,999		2	0.7	2	0.7	
>$200,000		2	0.7	1	0.4	

Some variables had missing data, therefore not all counts sum to the total n. NA = Native American; NHW = non-Hispanic White; Bolded values are significant at $\alpha = 0.05$.

Table 2: Intercorrelations of study variables.

	Variable	1	2	3	4	5	6	7	8	9	11	12
1	Cold tolerance (Log[s+1])											
2	Ischemia tolerance (Log[s+1])	0.42[a]										
3	Heat tolerance (C°)	0.44[a]	0.47[a]									
4	Electric tolerance (mA)	0.44[a]	0.37[a]	0.40[a]								
5	State Catas cold tolerance (Log[PCS+1])	−0.34[a]	−0.15[a]	−0.07	−0.09							
6	State Catas ischemia tolerance (Log[PCS+1])	0.001	−0.23[a]	−0.03	−0.01	0.68[a]						
7	State Catas heat tolerance (Log[PCS+1])	0.03	−0.10	0.06	0.05	0.65[a]	0.71[a]					
8	State Catas electric tolerance (Log[PCS+1])	−0.06	−0.23[a]	−0.13	−0.10	0.66[a]	0.73[a]	0.70[a]				
9	Trait Catas day 1 (Log[PCS+1])	0.03	−0.04	0.04	0.04	0.37[a]	0.41[a]	0.45[a]	0.44[a]			
10	Race/Ethnicity (NHW = 0, NA = 1)	−0.14[a]	−0.14[a]	−0.13	−0.02	0.13[a]	0.03	0.11	0.08	0.03		
11	Sex (Male = 0, Female = 1)	−0.15[a]	−0.17[a]	−0.34[a]	−0.06	−0.07	−0.09	−0.14[a]	0.01	−0.13[a]	0.17[a]	
12	BMI (kg/m^2)	−0.09	−0.15[a]	0.03	0.05	0.10	−0.04	0.02	0.01	0.06	0.21[a]	−0.04

[a]$p<0.05$. s = seconds. Catas = Catastrophizing.

Table 3: Indirect tests for the trait-activation model of pain catastrophizing.

Dependent variable	Path tested	Effect	SE	95% CI	
				Lower	Upper
Electric tolerance, mA	C'	3.741	2.634	−1.450	8.932
	Indirect ($a \times b$)	−2.188	1.271	−4.851	0.178
Heat tolerance (C°)	C'	−0.044	0.337	−0.710	0.621
	Indirect ($a \times b$)	0.066	0.150	−0.221	0.364
Cold tolerance (Log[s+1])	**C'**	**0.175**	**0.071**	**0.036**	**0.315**
	Indirect ($a \times b$)	**−0.158**	**0.039**	**−0.241**	**−0.088**
Ischemia tolerance (Log[s+1])	C'	0.077	0.081	−0.081	0.236
	Indirect ($a \times b$)	**−0.126**	**0.038**	**−0.206**	**−0.050**

C' = direct effect; $a \times b$ = indirect effect; SE = Standard Error. s = seconds; CI = confidence Interval for indirect effects were bootstrapped at 10,000 samples. Bolded effects are significantly different from 0. Sex and BMI were included as covariates.

Moderation of Trait-Activation of Pain Catastrophizing (path *a*)

One model out of the 4 (cold tolerance) had a significant moderated mediation (see Table 4). Results indicated that NHWs had a significant indirect effect ($a \times b$=−0.142), but NAs did not ($a \times b$=0.003). This suggests that the trait-activation hypothesis for pain catastrophizing was not significant in the NA sample regarding cold pain tolerances.

Moderation of the Pain Enhancing Effects of State Catastrophizing

One model (heat tolerance) out of the four had significant moderated mediation (Table 5); however, neither of the simple indirect effects for NAs or NHWs was significant. This suggests that the indirect effects in NA and NHW are significantly different from each other, but they are not significantly different than 0. Thus, NAs and NHWs did not differ in the way that state catastrophizing impacted pain tolerance.

Table 4: Results of moderated mediation for the relationship between trait and state pain catastrophizing (moderated a-paths).

Dependent variable	Tests of moderation and indirect paths	Effect	SE	95% CI	
				Lower	Upper
Electric tolerance, mA	Test of moderated mediation	0.623	0.710	−0.530	2.279
	Conditional indirect effects				
	$a \times b$ for NHWs	−2.097	1.247	−4.640	0.248
	$a \times b$ for NAs	−1.474	1.111	−4.080	0.182
Heat tolerance (C°)	Test of moderated mediation	−0.021	0.067	−0.170	0.114
	Conditional indirect effects				
	$a \times b$ for NHWs	0.063	0.144	−0.212	0.354
	$a \times$ b for NAs	0.042	0.113	−0.160	0.313
Cold tolerance (Log[s+1])	**Test of moderated mediation**	**0.146**	**0.059**	**0.041**	**0.271**
	Conditional indirect effects				
	$a \times b$ for NHWs	**−0.142**	**0.037**	**−0.223**	**−0.077**
	$a \times b$ for NAs	0.003	0.059	−0.115	0.119
Ischemia tolerance (Log[s+1])	Test of moderated mediation	0.040	0.045	−0.045	0.135
	Conditional indirect effects				
	$a \times b$ for NHWs	−0.123	0.037	−0.206	−0.058
	$a \times b$ for NAs	−0.083	0.056	−0.205	0.017

Bolded values are significantly different from 0. A significant Test of Moderated Mediation indicates that there was a significant difference between the indirect path for non-Hispanic Whites (NHWs) and the indirect path for Native Americans (NAs); C' = direct effect; SE = Standard Error. s = seconds; CI = confidence Interval for indirect effects were bootstrapped at 10,000 samples. NA = Native American; NHW = non-Hispanic Whites; Sex and BMI were included as covariates.

Table 5: Results of moderated mediation for the relationship between state pain catastrophizing and pain tolerance (moderated b-paths).

Dependent variable	Tests of moderation and indirect paths	Effect	SE	95% CI	
				Lower	Upper
Electric tolerance, mA	Test of moderated mediation	−0.612	2.348	−5.377	3.9087
	Conditional indirect effects				
	$a \times b$ for NHW	−2.140	1.317	−4.862	0.338
	$a \times b$ for NA	−2.752	2.915	−8.804	2.767
Heat tolerance (C°)	**Test of moderated mediation**	**−0.628**	**0.282**	**−1.221**	**−0.114**
	Conditional indirect effects				
	$a \times b$ for NHW	0.077	0.149	−0.210	0.380
	$a \times b$ for NA	−0.551	0.314	−1.20	0.037
Cold tolerance (Log[s+1])	Test of moderated mediation	−0.035	0.050	−0.142	0.061
	Conditional indirect effects				
	$a \times b$ for NHW	−0.150	0.037	−0.230	−0.086
	$a \times b$ for NA	−0.185	0.062	−0.320	−0.076
Ischemia tolerance (Log[s+1])	Test of moderated mediation	−0.069	0.059	−0.191	0.045
	Conditional indirect effects				
	$a \times b$ for NHW	−0.122	0.037	−0.202	−0.056
	$a \times b$ for NA	−0.192	0.073	−0.345	−0.058

Bolded values are significantly different from 0. A significant Test of Moderated Mediation indicates that there was a significant difference between the indirect path for non-Hispanic Whites (NHWs) and the indirect path for Native Americans (NAs). C' = direct effect; SE = Standard Error; s = seconds; CI = confidence Interval for indirect effects were bootstrapped at 10,000 samples; NA = Native American; NHW = non-Hispanic Whites; Sex and BMI were included as covariates.

Discussion

The present study had 2 aims: (1) to investigate if state pain catastrophizing mediated the relationship between trait catastrophizing and pain outcomes (trait-activation hypothesis), and (2) to see if this mediated relationship was moderated by race.

In relation to aim 1, results indicated that state catastrophizing mediated the relationship between trait catastrophizing and cold and ischemic pain tolerances. These results partially support a trait-activation model in which trait pain catastrophizing promotes state pain catastrophizing which then in turn reduces pain tolerance [9]. As such, trait pain catastrophizing is activated during a painful event and produces hyperalgesia by causing the individual to engage in state catastrophizing.

The present study is the first to formally test the trait-activation model of pain catastrophizing. Historically, pain catastrophizing has been conceptualized as a dispositional variable that remains relatively stable over time [32]. However, recent studies have noted that pain catastrophizing, when measured in response to a painful task, can vary from one context to another [9]. These results extend past literature that suggest that measures of trait-like personality features only predispose individuals to behaviors. However, traits must be activated by situational cues which trigger myriad of psychological mediating factors that influence behavior [14].

Previous studies suggest state catastrophizing is a predictor of pain in both healthy and clinical populations [16, 18, 33, 34]. When studies compare the effect of dispositional and state catastrophizing, state catastrophizing is typically a better predictor of experimental and clinical pain [16, 19, 35]. In the present study, zero-order correlations indicated that state catastrophizing predicted pain tolerance (cold, ischemia) whereas trait catastrophizing did not. This is in line with previous studies suggesting a small (and sometimes non-significant) relationship between trait catastrophizing and experimental pain. Indeed, some studies did not find a relationship between trait catastrophizing and pressure (muscle and bone), thermal, and visceral (thermal and electric) experimental pain [36]. On the other hand, *in vivo* manipulation of state catastrophizing has been fruitful in detecting state catastrophizing-related pain changes. Notably, two studies provide compelling evidence linking state pain catastrophizing with hyperalgesia. One study experimentally decreased state pain catastrophizing with the use of positive coping statements/mental imagery in the presence of a painful threat which resulted in lower pain [37]. Another study, increased and decreased state pain catastrophizing with hypnotic suggestions, resulting in increased and decreased pain, respectively [38]. This underscores the importance of assessing both trait and state catastrophizing to fully understand the relationship between catastrophizing and pain. This could be achieved through the implementation of diary studies. For example,

one study suggested the relationship between daily pain and trait catastrophizing is amplified by state catastrophizing [39].

Interestingly, indirect effects were not significant for the heat and electric tolerance models. It is possible that prolonged, tonic, noxious stimulation is needed to elicit state catastrophizing's hyperalgesic effects. Indeed, it has been suggested that the hyperalgesic effects from state pain catastrophizing increase over the duration of the stimuli [36]. As such, tonic pain stimuli would provide enough time for pain catastrophizing to exert its hyperalgesic effects on pain. This effect was evident in a multimodal (i.e., heat, cold, pressure), multi-tissue (i.e., somatic, visceral) experimental study that found that only cold pressor pain was related to trait pain catastrophizing [36]. Although the study did not measure state catastrophizing, it can be inferred that psychological states during painful events modify pain (e.g., state) [40]. Thus, many studies that investigate state catastrophizing's influence on experimental pain use tonic noxious stimuli such as a cold pressor [17, 19, 35].

In relation to aim 2, our results indicate that in response to certain experimental pain procedures, race/ethnicity moderates the indirect effects. When path *a* was moderated, the simple indirect effect of trait pain catastrophizing on cold tolerance was significant for NHWs but not for NAs. This suggests that the path by which trait catastrophizing influences pain is not constant across people or stimuli. Indeed, the concept that activated traits do not release the same mediating factors (which ultimately influence behavior) is central to C-CAPS [14]. Mediating factors are individualized beliefs, memories, values, and a host of other psychological variables, all of which are influenced by culture. As such, trait pain catastrophizing may release different mediating factors in NAs than in NHWs after being activated by the painfully cold water. Indeed, prior studies in NAs have suggested that the increased state catastrophizing in NAs is driven by prior adverse experiences and psychological distress [11]. As such, it is possible that adverse experiences and psychological distress may influence the relationship between trait and state pain catastrophizing.

An interesting finding is that the direct effects (c' path) predicting cold tolerance scores were positive. These results indicate that when the relationship between state catastrophizing and tolerance is controlled, there is a positive relationship between trait catastrophizing and cold tolerance (hypoalgesia). This contrasts literature indicating that trait pain catastrophizing is also hyperalgesic [9]. However, the results may represent an artificial scenario in which the hyperalgesic relationship between state catastrophizing and pain tolerance is controlled for, which may erase trait pain catastrophizing's negative influence on pain.

Potential implications for reducing pain disparities

Pain catastrophizing mediates improvement in psychological pain interventions including Cognitive Behavioral Therapy (CBT) [41], and Acceptance and Commitment Therapy (ACT) [42, 43]. The results from the present study support the treatment target and suggest that psychological interventions may benefit from increased focus on the reduction of pain catastrophizing particularly in response to tonic pain. Further research is needed on the most effective way to reduce state catastrophizing. Nevertheless, we can hypothesize that this may be accomplished with live coaching while participants are exposed to uncomfortable sensations much akin to exposure-based psychological interventions, which have a history of successfully treating fear-based psychological disorders [44–48]. However, since trait and state pain catastrophizing are weakly correlated, this would suggest that reducing trait catastrophizing may lead to clinical improvements by lowering state catastrophizing (and more so in NHWs); however, this must be empirically tested.

Further, the results from this study suggest the existence of differential relationships between state and trait catastrophizing by race/ethnicity in some instances. Since trait catastrophizing acts through state catastrophizing to influence pain, this would suggest that the most effective treatment targets for chronic pain interventions may differ by race/ethnicity. However, this must be further tested, and currently appears impractical as it is unlikely that a reduction in trait catastrophizing can occur without modifying state catastrophizing.

Strengths and limitations

To our knowledge, this is the first study to formally assess the trait-activation model of pain catastrophizing. Further, we assessed whether components of the model can be moderated by race/ethnicity. The present study had several other strengths. First, we had a large sample that allowed us to investigate individual, as well as group, differences. Second, this study used several pain modalities to

investigate any possible differences between pain characteristics (e.g., tonic vs. phasic stimuli). Third, we investigated race/ethnic differences. NA status was corroborated with CDIB card and tribal membership cards. Fourth, we used state-of-the-art data analysis techniques to bolster statistical inferences.

Despite these strengths, a few limitations should be noted. First, we recruited only healthy, pain-free individuals so results may not generalize to participants with chronic pain or other health problems. Future studies could recruit participants with chronic pain to help overcome this limitation. Second, NAs in OK-SNAP were primarily from urban areas in northeastern Oklahoma; there may be important differences associated with NAs from other regions that could alter responses to the pain catastrophizing questionnaires. Indeed, tribes are not monolithic, and each may have their own unique cultural practices and beliefs about pain. Third, although the use of modern statistical analysis (e.g., bootstrapped moderated mediation models) is a strength, these models did not correct for family-wise error which is a possible limitation.

Summary

The present study is the first to give support to the idea that state catastrophizing mediates the relationship between trait catastrophizing and tonic pain outcomes. Further, this relationship, in some instances, is moderated by race/ethnicity which gives insight into treatment targets and a mechanistic theory of pain catastrophizing and pain.

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