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The Association Between Adverse Life Events, Psychological Stress, and Pain-Promoting Affect and Cognitions in Native Americans: Results from the Oklahoma Study of Native American Pain Risk

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Abstract
Native Americans (NAs) experience higher rates of chronic pain. To examine the mechanisms for this pain inequity, we have previously shown that NAs report higher levels of pain-related anxiety and pain catastrophizing, which are in turn related to pronociceptive (pain-promoting) processes. But, it is currently unclear why NAs would report greater pain-related anxiety and catastrophizing. Given that NAs are also more likely to experience adverse life events (ALEs) and associated psychological distress, it was hypothesized that higher anxiety/catastrophizing in NAs would be partially explained by higher rates of ALEs and psychological distress. Structural equation modeling was used to analyze these pathways (NA ethnicity ➔ ALEs ➔ psychological distress ➔ pain anxiety/catastrophizing) in 305 healthy, pain-free adults ($N = 155$ NAs, $N = 150$ non-Hispanic Whites [NHWs]). Pain-related anxiety and situational pain catastrophizing were assessed in response to a variety of painful tasks. The Life Events Checklist was used to assess cumulative exposure to ALEs that directly happened to each participant. A latent psychological distress variable was modeled from self-reported perceived stress and psychological symptoms. Results found that NAs experienced more ALEs and greater psychological distress which was associated with higher rates of pain-related anxiety and pain catastrophizing. Notably, NAs did not report greater psychological distress when controlling for ALE exposure. This suggests that a higher risk of chronic pain in NAs may be due, in part, to psychological distress, pain-related anxiety, and pain catastrophizing that are promoted by exposure to ALEs. These results highlight several targets for intervention to decrease NA pain risk.

Keywords Racial/ethnic differences · American Indians · Pain affect · Pain-related cognitions · Chronic pain risk factors

Introduction

Chronic pain is typically defined as pain that persists or recurs for more than 3 to 6 months [1]. Prevalence studies indicate that Native Americans (NAs) have higher rates of chronic pain than the general US population [2–4], yet the biological, psychological, and sociocultural mechanisms underlying this increased risk are understudied and remain elusive.

The Role of Pain-Related Anxiety and Pain Catastrophizing in Native American Pain Risk

A variety of studies suggest that psychological factors may promote or maintain chronic pain, and that some may have stronger effects for NAs than non-Hispanic Whites (NHWs) [5–7]. To identify the factors that promote pain in NAs, our lab conducted the *Oklahoma Study of Native American Pain Risk* (*OK-SNAP*). We found that NAs reported higher pain-related anxiety and pain catastrophizing in response to multiple painful tasks [6]. Pain catastrophizing refers to an individual's tendency to ruminate, magnify, and feel helpless in the face of pain, and includes thoughts such as "I can't stop thinking about how much it hurts," "I worry that something serious

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may happen," and "It's terrible and I think it's never going to get any better" [8]. Pain-related anxiety includes anxious or fearful responses to pain and is frequently conceptualized as part of pain-related fear, which also includes fear of activities that may elicit pain and fear of movement or (re)injury [9]. Our findings of greater pain-related anxiety and catastrophizing for NAs are consistent with literature reporting greater pain-related cognitive-affective processes in other minorities [5, 7], and suggest that pain-related anxiety and catastrophizing may promote chronic pain for NAs.

Indeed, both pain catastrophizing and pain-related anxiety are strongly associated with chronic pain conditions and their negative sequelae (e.g., impairment/disability). For instance, pain catastrophizing is linked to pain severity in chronic pain populations, as well as pain-related disability, illness behaviors, depression, and negative mood [10]. Additionally, treatment studies suggest that reductions in catastrophizing mediate improvement in pain levels and pain-related disability [10]. Furthermore, presurgical catastrophizing predicts both acute postoperative pain severity and the progression of acute to chronic postsurgical pain [11]. Similarly, pain-related fear predicts postsurgical pain [12]. Even in healthy, pain-free individuals, greater pain-related anxiety/fear is associated with delayed recovery (i.e., increased interference/disability) in response to acute injury [9]. In individuals with chronic low back pain, pain-related fear may broadly contribute to greater disability through inactivity [9]. Additionally, some of the strongest predictors of pain intensity in fibromyalgia are negative emotions that are associated with pain-related anxiety [13, 14].

Effects of Pain-Related Anxiety and Pain Catastrophizing on Experimental Pain

Experimental studies on pain-related catastrophizing and anxiety in healthy samples show results similar to clinical pain studies. For instance, catastrophizing assessed after a cold pressor task was associated with higher pain ratings and lower pain tolerance during the task [15]. Catastrophizing was also positively correlated with pain scores during heat and muscle pain induction [16]. Additionally, healthy participants undergoing an intervention to reduce catastrophizing showed lower pain and temporal summation of pain ratings, and analyses revealed that these reductions were indeed mediated by a reduction in catastrophizing [17]. Similarly, induced anxiety about a cold pressor task increased pain experienced [18] and anxiety induced by threat of shock increased pain reactivity and decreased pain thresholds [19]. Thus, clinical findings on the positive association between catastrophizing/anxiety and pain appear to apply to healthy participants experiencing experimental pain as well.

The Pronociceptive Effects of Pain-Related Anxiety and Pain Catastrophizing

Several studies have examined how pain catastrophizing and pain-related anxiety may promote pronociceptive (pain-signal promoting) processes to increase chronic pain risk. Following exposure to a noxious (potentially tissue damaging) stimulus, peripheral fibers transmit the nociceptive signal to the spinal cord where it is transmitted to brainstem, subcortical, and cortical areas that work together to integrate sensory and affective information, ultimately leading to the experience of pain [20, 21]. In addition to this ascending pain system, the experience of pain is also influenced by a brain-to-spinal cord (descending) system that can modulate the incoming nociceptive signals [22–24]. This system can either facilitate (increase) or inhibit (decrease) subsequent nociceptive signaling at the spinal cord, thereby turning pain levels up or down, respectively [25]. Prior analyses of OK-SNAP data suggest that pain catastrophizing and pain-related anxiety mediate the relationship between NA ethnicity and impaired descending inhibition of spinal nociception [26, 27] and hyperalgesia [27]. Having established these pronociceptive sequelae of pain-related anxiety and pain catastrophizing in NAs (impaired descending inhibition, decreased pain tolerance), the next step was to determine the factors that predict higher rates of pain-related anxiety and catastrophizing in this population.

What Promotes Higher Pain-Related Anxiety and Pain Catastrophizing in Native Americans?

It is currently unknown why NAs would experience greater pain-related anxiety and pain catastrophizing. Importantly, identifying the mechanisms that increase these pain-promoting factors could help identify targets for interventions. It is well known that NAs have experienced systemic and historical adversity (e.g., genocide, forced relocation, forced separation of children to attend boarding schools, racism, discrimination), and evidence suggests they continue to experience adverse life events (ALEs), including physical and sexual assault, at higher rates than other cultural groups in the USA [28–31]. Additionally, there is a large body of evidence showing comorbidity between exposure to ALEs and chronic pain states [32, 33]. Thus, ALE exposure may promote chronic pain risk in NAs.

Findings from OK-SNAP, as well as research from other labs, have shown that exposure to ALEs can lead to greater pain amplification [34] and sensitization of spinal neurons [35–37]. Thus, ALEs may directly impact pronociceptive processes, but they may also have an indirect effect through psychological consequences. Indeed, much is known about the negative psychological outcomes of ALEs such as depression, anxiety, and posttraumatic stress disorder (PTSD) [38]. Not all individuals that experience adversity go on to develop chronic

pain. Thus, the level of psychological stress experienced subsequent to ALEs may affect who ultimately develops chronic pain, with higher levels of stress leading to greater pain risk. Given this, ALEs and psychological distress/stress may promote pain-related negative affect and cognition (i.e., pain catastrophizing and pain-related anxiety), which enhance chronic pain risk. Thus, psychological stress/distress may be an important factor in determining who is vulnerable and more likely to experience pain-related anxiety and catastrophizing (and ultimately go on to develop chronic pain).

Given these relationships, the current study (an ancillary analysis of OK-SNAP) examined whether ALEs and psychological distress are associated with higher rates of pain-related anxiety and pain catastrophizing in NAs. As our measures of psychological distress did not ask participants to report on distress directly related to ALEs, our psychological distress variable may capture ALE-related and ALE-unrelated psychological distress. Thus, the SEM model included a direct pathway from NA ethnicity to psychological distress, as well as an indirect pathway through ALEs. It was hypothesized that (1) ALEs would be associated with greater pain catastrophizing and pain-related anxiety, and (2) these relationships would be at least partially mediated by psychological distress in NAs.

Methods

Brief Overview of Procedures

A full description of the OK-SNAP parent study is reported elsewhere [39]. Figure 1 gives an overview of study procedures. In brief, testing was conducted over 2 days, with each day lasting 4–6 h. Informed consent, review of inclusion/exclusion criteria, and the Life Events Checklist (LEC-4; a measure of ALEs) were conducted on the first day. On one of the testing days, pain threshold and tolerance were assessed from multiple stimulus modalities (e.g., electric, heat, cold, ischemic, mechanical pressure). The other day of testing involved multiple electrophysiological and dynamic measures of pain: nociceptive flexion reflex (NFR) threshold, temporal summation of NFR, response to brief heat pulses, conditioned pain modulation, and emotional modulation of pain and NFR. Situational pain catastrophizing and pain-related anxiety were assessed immediately after pain tasks. Order of testing day was randomized but blocked by ethnicity and sex. Moreover, tests within each day were partly randomized to avoid order effects. Breaks were provided between tasks to minimize carryover effects. Measures of psychological distress (perceived stress, psychological symptoms) were assessed during one of these breaks on the first day of testing.

Participants

Healthy, pain-free participants were recruited in OK-SNAP to help ensure that any observed differences in pain processing were not due to disparities in pain condition/etiology, disease severity, and/or pain treatment [40–42]. Recruitment efforts included tribal and non-tribal newspaper ads, fliers, personal communications with NA groups, email announcements, online platforms (e.g., Facebook), and word of mouth. Those who appeared eligible following a phone screen were invited to attend an initial laboratory visit, which began with a thorough screening for inclusion and exclusion criteria. Data collection occurred between March 2014 and October 2018. All participants were given an overview of procedures and told they could withdraw at any time before providing verbal and written informed consent. The study was approved by IRBs of The University of Tulsa, Cherokee Nation, and the Indian Health Service Oklahoma City Area Office. Participants received a $100 honorarium for the completion of each testing day.

Exclusion criteria included the following: (1) < 18 years old, (2) history of self-reported cardiovascular, neuroendocrine, musculoskeletal, neurological disorders, (3) chronic pain or current acute pain, (4) BMI ≥ 35 (due to difficulties recording electromyogram for NFR), (5) current/recent use of anti-depressants, anxiolytic, analgesic, stimulant, or anti-hypertensive medication, (6) current psychotic symptoms (assessed by Psychosis Screening Questionnaire [43]) or substance use problems, and/or (7) an inability to read and speak English. Of the 329 eligible participants, 247 completed both testing days, 41 completed one day, and 39 completed part of one day. Two participants' data were lost due to a computer malfunction. Twenty-two participants were non-NA minorities and were excluded from analyses. Thus, 155 NA (64 males) and 150 NHW (76 males) were included in the current study (Table 1).

Power analyses for the primary aims of the parent (OK-SNAP) study were conducted with G*Power (version 3.1.9.2) and indicated that 120 per group ($N = 240$ total) would result in power of 0.80 for most outcomes. The sample size estimates needed for structural equation modeling are often based on N-to-parameter ratios, with ratios varying between 10:1 [44] and 20:1 [45]. There were 29 estimated parameters in the model and 305 participants; thus, the ratio for the current study was 10.5 which should be adequate.

Primary Variables

Native American Status Race/ethnicity was first assessed by self-report, and NA status was subsequently verified from Certificate of Degree of Indian Blood or tribal membership cards. NA participants represented tribal nations predominately from the southern plains and eastern Oklahoma tribes. To

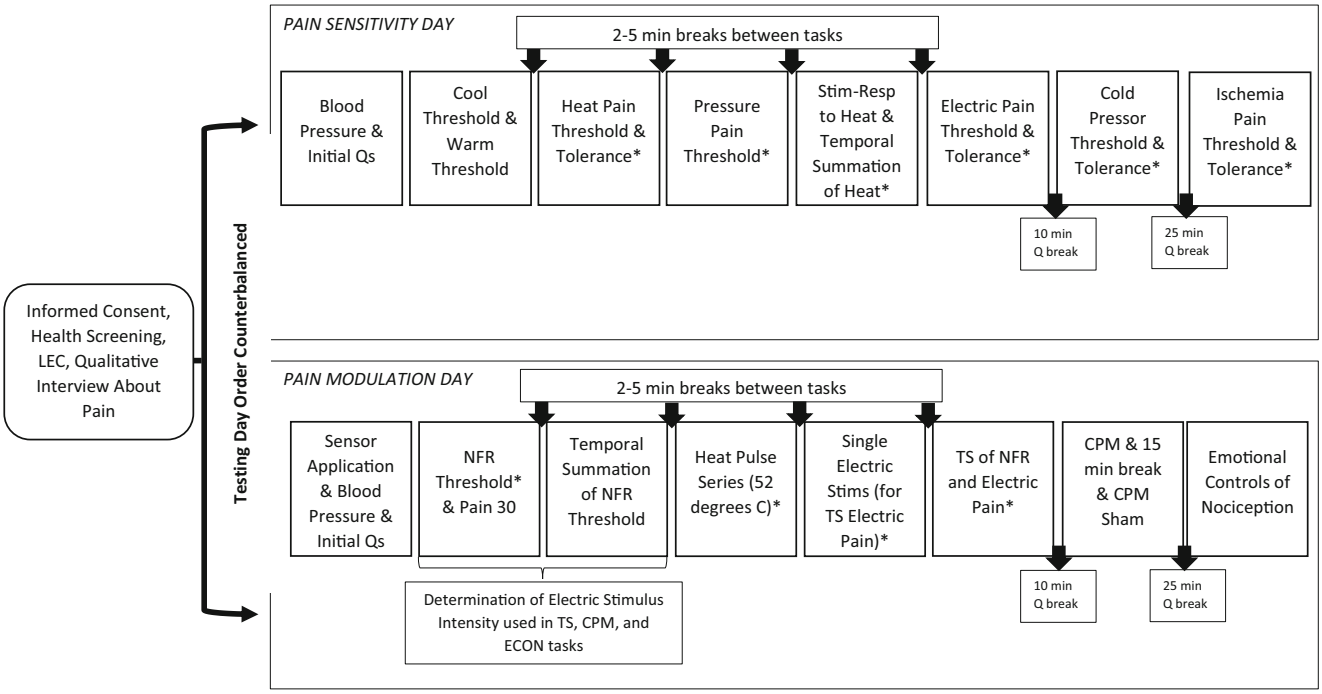

Fig. 1 Study procedures. Testing was conducted over a 2-day period (approximately 1 week apart) with testing day order counterbalanced. To minimize carryover effects, mandatory breaks were included. The asterisk indicates that pain-related anxiety and situational pain catastrophizing were measured immediately after this pain task. Stim-Resp stimulus response, NFR nociceptive flexion reflex, TS temporal summation, CPM conditioned pain modulation, ECON emotional controls of nociception

respect tribal confidentiality, tribal affiliations are not reported. For this variable, NHWs were coded 0 and NAs were coded 1.

Adverse Life Events The Life Events Checklist (LEC-4) was used to assess ALEs [46]. The LEC is comprised of 17 items assessing various stressful events: physical assault, sexual assault, other unwanted sexual contact, combat exposure, captivity, natural disaster, fire/explosion, sudden violent death, sudden unexpected death of someone close, serious harm/death you caused to someone else, transportation accident, serious accident at work/home/etc., exposure to toxic substance, life-threatening illness, severe human suffering, and other stressful events. The participant is asked to indicate the proximity to the event by endorsing if the event "happened to me," "witnessed it," or "learned about it." To be consistent with our other studies showing ALEs are related to pain-promoting processes [35, 47], items in which the participant responded with "happened to me" were summed to yield a total ALEs score that ranged between 0 and 17. For descriptive purposes, the frequencies of experienced ALEs for each ethnic group are reported in Table 2. Transportation accidents, sudden unexpected deaths, physical assaults, and natural disasters were most frequently experienced for both NAs and NHWs.

Psychological Stress/Distress A latent variable for psychological stress/distress was constructed from the Perceived Stress Scale (PSS) and the Global Severity Index (GSI) of the

Table 1 Means and standard deviation of study variables by the racial/ethnic group

	NHW Mean	(n=150) SD	NA Mean	(n=155) SD	t test	p value	Cohen's d
Situational pain catas (PCS; 0–52)	8.744	7.068	11.188	7.881	2.853	0.005	0.326
Pain-related anxiety (VAS; 0–100)	36.500	20.840	44.046	21.646	3.100	0.002	0.355
Adverse life events (LEC; 0–17)	1.733	1.496	2.116	1.554	2.191	0.029	0.251
Perceived stress (PSS; 0–40)	13.087	5.930	14.497	5.972	2.069	0.039	0.237
Psychological symptoms (Log GSI)	0.108	0.082	0.134	0.090	2.591	0.010	0.296
General health (SF-36; 0–100)	80.467	13.449	78.458	13.379	1.307	0.192	0.150

PCS Pain Catastrophizing Scale, *VAS* visual analog scale, *NHW* non-Hispanic White, *NA* Native American, *LEC* Life Events Checklist, *PSS* Perceived Stress Scale, *GSI* Global Severity Index of the SCL-90

Table 2 Frequency of adverse life events experienced by ethnicity

	NHW		NA	
	N	%	*N*	%
Natural disaster	35	11.6	39	13
Fire or explosion	12	4	25	8.4
Transportation accident	69	22.8	79	26.1
Serious accidents work/home/recreation	18	6.1	15	5.1
Exposure to toxic substance	4	1.4	6	2
Physical assault	33	11.1	47	15.8
Assault with a weapon	11	3.7	17	5.7
Sexual assault	9	3	15	5.1
Other unwanted sexual exp.	16	5.4	29	9.8
Combat or exposure to war zone	5	1.7	3	1
Captivity	4	1.3	1	.3
Life-threatening illness or injury	1	.3	5	1.7
Severe human suffering	0	0	2	.7
Sudden, violent death	2	.7	5	1.7
Sudden, unexpected death	43	14.4	58	19.4
Serious injury/harm/death you caused	3	1.0	7	2.4
Other	27	9.1	31	10.5

NA Native American, *NHW* non-Hispanic White

Symptom Checklist-90-Revised (SCL-90-R). The PSS is a 10-item measure that assesses psychological stress within the past month. Scores range from 0 to 40, with higher scores indicating more perceived stress [48]. The GSI of the SCL-90-R is assessed from 90 items querying different psychological symptoms [49]. GSI scores range from 0 to 4 with higher scores indicating more problems.

Situational Pain Catastrophizing Situational (state) pain catastrophizing represents catastrophic thoughts that happened during a specific painful event. Situational pain catastrophizing was assessed using the Pain Catastrophizing Scale (PCS) [8], which we have previously shown to have a similar factor structure in NAs and NHWs [50]. Similar to previous research [26], the instructions on the PCS were modified, asking participants to think back on their thoughts during each painful task and to rate the degree to which they catastrophized during the task (i.e., situational pain catastrophizing) [10]. Total scores for the situational PCS ranged from 0 to 52, with higher scores indicating greater catastrophic thoughts. As noted in Fig. 1, situational pain catastrophizing was assessed following 10 pain tasks and the total score was calculated in response to each task. Although the different pain tasks evoked different mean levels of situational pain catastrophizing [6], the 10 situational pain catastrophizing total scores had a high Cronbach's alpha in both ethnic groups (NA: $\alpha = .92$; NHW: $\alpha = .93$) suggesting they could be averaged to create a global situational pain catastrophizing score.

Pain-Related Anxiety Pain-related anxiety was measured from a VAS with the anchors "not at all anxious" and "extremely anxious" [51, 52]. The computer converted the participants' response to scores that ranged from 0 to 100. We have shown elsewhere that pain-related anxiety measured in this way shows similar predictive validity in NHWs and NAs [27], because race/ethnicity did not moderate the relationships between the pain-related anxiety VAS and experimental pain outcomes. As noted in Fig. 1, the pain-related anxiety VAS was assessed following 10 pain tasks [39] with the instructions: "Using this scale, rate how anxious the [*insert pain task here*] made you feel." Although the different pain tasks evoked different mean levels of pain-related anxiety [6], the 10 pain-related anxiety scores had a high Cronbach's alpha in both ethnic groups (NA: $\alpha = .90$; NHW: $\alpha = .92$) suggesting they could be averaged to create a global pain-related anxiety score.

Control Variables In the parent study, we found that general health perception (subscale of the Medical Outcomes Study 36-item Short Form Health Survey (SF-36)) [53] was marginally lower in NAs, relative to NHWs. Given the potential for health to affect psychological stress, pain catastrophizing, and pain-related anxiety, this variable was entered as control variable in the analyses. Furthermore, there are well-established sex differences in pain [54], pain coping (including pain catastrophizing and anxiety) [55], and psychological distress [56, 57]; therefore, participant sex was also entered as a control variable.

Data Analysis

All structural equation models were conducted with LISREL 8.8 using full information maximum likelihood (FIML) [58]. Psychological distress (GSI) was log10 transformed to reduce positive skew. Outliers were identified according to established procedures (median absolute deviation) and then winsorized to the next nearest non-outlier value [59]. Outliers were identified and winsorized for the following variables: general health (SF-36), perceived stress (PSS), psychological distress (GSI), and situational pain catastrophizing (PCS). After normality and outliers were corrected, missing observations on the corrected variables were estimated with multiple imputations using LISREL. Model fit was assessed from the RMSEA (root mean square error of approximation). As noted by Kline [45], values ≤ 0.05 are considered "close fit," values between .05 and .08 are considered a "reasonable approximate fit," and values $\geq .10$ are considered "poor fit." Moreover, the "Test of Close Fit" provided by LISREL was also reported. The Test of Close Fit assesses the null hypothesis (H_0) that RMSEA < 0.05; thus, if H_0 is rejected, the model is not close fitting. The chi-square goodness of fit is also reported but is sensitive to sample size. Independent samples *t* tests were

conducted in SPSS to examine racial/ethnic differences on study variables for descriptive purposes. Significance was set at $p < 0.05$ (2-tailed).

Results

Background Characteristics

In the sample of 305 participants, 155 were NA (55% female) and 150 were NHW (45% female; $\chi^2 = 2.70$, $p = 0.100$). Table 1 presents means and standard deviations (SD) for study variables by racial/ethnic group. Overall means and SDs, as well as Pearson's correlations for study variables are presented in Table 3. 1.76% of values were imputed.

Consistent with what we have reported previously [6, 35, 39], Table 1 shows that NAs reported higher levels of situational pain catastrophizing, pain-related anxiety, adverse life events, perceived stress, and psychological symptoms (all p's < 0.05). Although sex was not a focus of the manuscript, sex differences have been noted in other studies of pain processing [60, 61], but as Table 3 shows, we did not find that in the current study.

Mediators of the Relationship Between Ethnicity and Pain-Promoting Psychological Variables

Results for analysis are presented in Table 4 and the structural model is depicted in Fig. 2. As shown, the model demonstrated a close fit to these data, with RMSEA = 0.029 (90% CI: 0.00, 0.0799; test of close fit $p = 0.691$) and a non-significant chi-square ($\chi^2 = 8.80$, df = 8, $p = 0.27$).

As shown in Fig. 2, NAs experienced more ALEs which, in turn, was associated with greater psychological distress. Subsequently, psychological distress was associated with greater levels of situational pain catastrophizing and pain-related anxiety. Notably, exposure to ALEs did not have a direct relationship with situational pain catastrophizing nor with pain-related anxiety. The relationships between ALEs and these pain-promoting psychological factors were mediated via psychological distress. This was confirmed by significant indirect effects between ALEs and situational catastrophizing (indirect effect = 0.378, SE = 0.134, $p = 0.005$) and between ALEs and pain-related anxiety (indirect effect = 0.586, SE = 0.258, $p = 0.023$).

Of importance, there were significant indirect pathways linking NA ethnicity to situational pain catastrophizing (indirect effect = 0.847, SE = 0.400, $p = 0.034$) and pain-related anxiety (indirect effect = 1.463, SE = 0.740, $p = 0.048$). When these indirect paths were accounted for, the direct relationship between NA ethnicity and situational pain catastrophizing was no longer significant, but there was still a significant direct relationship with pain-related anxiety. Taken together, these analyses provide support for the hypothesis that exposures to adverse life events and psychological stress/distress have downstream effects on pain-promoting psychological factors (catastrophizing and anxiety).

Discussion

This study examined the relationships between adverse life events, psychological distress, and pain-promoting cognition and affect in a sample of healthy, pain-free NAs and NHWs. Consistent with our hypotheses, ALEs were associated with increased psychological distress, which in turn was associated with increases in both situational pain catastrophizing and pain-related anxiety. However, there was no direct effect of ALEs on either of these outcomes. Thus, the ALEs-psychological distress pathway provided an indirect linkage between NAs and pain-related anxiety and pain catastrophizing.

Table 3 Means, standard deviations, and intercorrelations among study variables

	M	SD	S-Catas	PainAnx	Race	ALEs	Stress	Psych	Sex	GenHlth
Situational pain catas (PCS; 0–52)	9.986	7.579	1							
Pain-related anxiety (VAS; 0–100)	40.334	21.552	.733*	1						
Race (0=NHW, 1=NA)	0.508	0.501	.161*	.175*	1					
Adverse life events (LEC; 0–17)	1.928	1.535	.115*	0.109	.125*	1				
Perceived stress (PSS; 0–40)	13.803	5.984	.351*	.225*	.118*	.117*	1			
Psychological symptoms (log GSI; 0–1)	0.121	0.087	.386*	.258*	.147*	.236*	.708*	1		
Biological sex (0=male, 1=female)	0.541	0.499	−0.014	0.028	0.094	−0.022	−0.019	−0.022	1	
General health (SF-36; 0–100)	79.446	13.429	−0.182*	−.193*	−0.075	−0.107	−.337*	−.346*	0.022	1

PCS Pain Catastrophizing Scale, *VAS* visual analog scale, *NHW* non-Hispanic White, *NA* Native American, *LEC* Life Events Checklist, *PSS* Perceived Stress Scale, *GSI* Global Severity Index of the SCL-90

The asterisk indicates $p < 0.05$ (2-tailed)

Table 4 Unstandardized coefficients for the structural equation model predicting pain-promoting psychological factors ($N = 305$)

	Estimate	SE	*Z* test	R^2
Measurement model				
Native American status ➜ race (NHW = 0, NA = 1)	1.00			1.00
Adverse life events ➜ LEC	1.00			1.00
Psychological distress ➜ PSS	1.00			.62
Psychological distress ➜ GSI	*0.0164*	0.00157	10.453	0.800
Situational pain catastrophizing ➜ PCS	1.00			1.00
Pain-related anxiety ➜ VAS of PainAnx	1.00			1.00
Biological sex ➜ sex	1.00			1.00
Health perception ➜ SF-36 GH	1.00			1.00
Error in race	—			
Error in ALEs	—			
Error in PSS	*13.813*	2.186	6.320	
Error in GSI	*0.00152*	0.000517	2.949	
Error in PCS	—			
Error in VAS PainAnx	—			
Error in sex	—			
Error in SF-36 GH	—			
Structural model				
Predicting adverse life experiences				0.0158
NA status ➜ adverse life experiences	*0.387*	0.176	2.204	
Predicting psychological distress				0.198
NA status ➜ psychological distress	1.039	0.562	1.848	
ALES ➜ psychological distress	*0.577*	0.185	3.123	
Sex ➜ psychological distress	−0.258	0.554	−0.467	
Health perception ➜ psychological distress	*−0.130*	0.0221	−5.896	
Predicting situational pain catastrophizing				0.195
NA status ➜ situational pain catastrophizing	1.415	0.827	1.710	
ALES ➜ situational pain catastrophizing	0.0514	0.273	0.189	
Psychological distress ➜ situational pain catastrophizing	*0.655*	0.110	5.963	
Sex ➜ situational pain catastrophizing	−0.196	0.814	−0.241	
Health perception ➜ situational pain catastrophizing	−0.00510	0.0333	−0.153	
Predicting pain-related anxiety				0.105
NA status ➜ pain-related anxiety	*5.375*	2.448	2.196	
ALES ➜ pain-related anxiety	0.470	0.806	0.583	
Psychological distress ➜ pain-related anxiety	*1.015*	0.316	3.213	
Sex ➜ pain-related anxiety	1.011	2.409	0.420	
Health perception ➜ pain-related anxiety	−0.143	0.0980	−1.462	
Residual variance				
Residual for adverse life events	*2.327*	0.189	12.288	
Residual for psychological distress	*18.203*	2.669	6.821	
Residual for situational pain catastrophizing	*46.567*	3.989	11.673	
Residual for pain-related anxiety	*420.058*	34.819	12.064	

Z tests ≥ 1.96 or ≤ − 1.96 are statistically significant at $p < 0.05$. Italicized estimates are statistically significant. *NHW* non-Hispanic White, *NA* Native American, *LEC* Life Events Checklist, *PSS* Perceived Stress Scale, *GSI* Global Severity Index of the SCL-90, *PCS* Pain Catastrophizing Scale, *VAS* visual analog scale, *SF-36 GH* General Health subscale of the SF-36

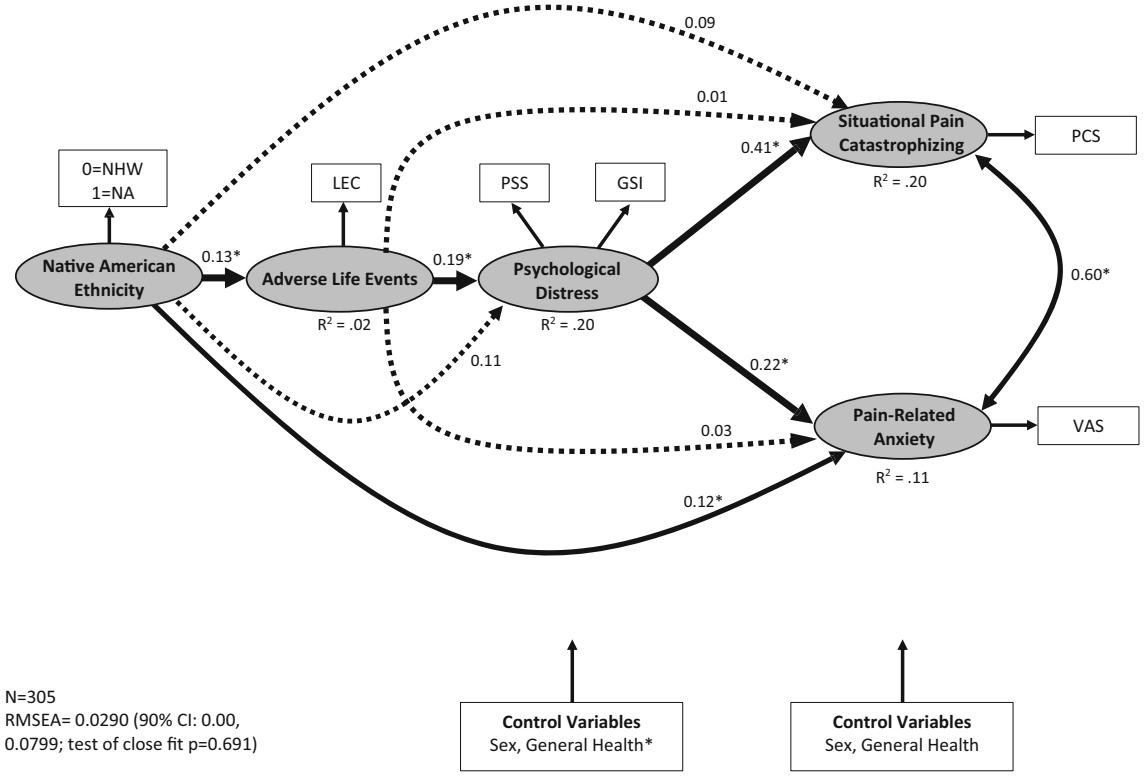

Fig. 2 Structural model linking Native Americans (NA) to pain-promoting psychological factors (situational pain catastrophizing, pain-related anxiety). We have demonstrated in previous studies that NAs experience greater situational pain catastrophizing and pain-related anxiety and that these psychological factors are associated with pronociceptive processes (e.g., reduced descending inhibition of nociception, reduced pain tolerance). Results of this structural model indicate that NAs' greater exposure to adverse life events (ALEs) promotes psychological distress that in turn increases pain catastrophizing and pain-related anxiety, but that NAs also experience more pain-related anxiety (even after controlling for adversity and psychological distress.) Standardized estimates are reported next to each structural path and tested paths that are non-significant are represented by dashed lines. Control variables (biological sex, general health perceptions) were included as predictors of psychological distress, situational pain catastrophizing, and pain-related anxiety; however, only the (negative) relationship between general health perceptions and psychological distress was significant. NHW non-Hispanic Whites, LEC Life Events Checklist, PSS Perceived Stress Scale, GSI Global Severity Index of the SCL-90; $^{*}p < 0.05$

Pathways of Native American Pain Risk

The higher levels of pain-related anxiety and pain catastrophizing that NAs experience may put them at increased risk for chronic pain [6]. Pain catastrophizing is a strong predictor of postoperative pain severity, the progression of postoperative to chronic pain, and negative sequelae of chronic pain disorders (e.g., heightened pain perception, greater levels of disability/depression) [10, 11, 62]. Similarly, pain-related anxiety/fear leads to greater pain intensity, disability, and impairment in chronic pain populations [9, 13, 63]. Thus, there are strong associations between pain-related anxiety/catastrophizing and chronic pain conditions.

Previous research has outlined ways pain catastrophizing and anxiety may confer increased chronic pain risk. Pain catastrophizing is associated with temporal summation of pain (TS-pain) [51], even in healthy, pain-free participants. TS-pain is the psychophysical correlate of increased hyperexcitability of spinal neurons, which amplifies incoming nociception; thus, catastrophizing may amplify nociceptive signaling even in pain-free individuals. Similarly, pain-related anxiety has been linked to hyperalgesia in pain-free individuals [19]. Thus, higher levels of pain-related anxiety and pain catastrophizing in NAs are believed to increase their chronic pain risk through pronociceptive processes, such as amplification of incoming nociceptive signaling.

Indeed, other analyses of OK-SNAP data suggest that pain-related anxiety and pain catastrophizing promote pronociceptive processes in NAs. For example, a paper by Toledo and colleagues demonstrated that pain catastrophizing was a mediator between NA ethnicity and impaired inhibition of spinal nociception [26]. This suggests catastrophizing promotes pain risk by allowing greater ascending signals to reach the brain. Additionally, Rhudy and colleagues found that pain-related anxiety was a mediator between NA ethnicity and impaired descending inhibition of spinal nociception, as well as reduced pain tolerance (i.e., hyperalgesia) [27]. Thus, higher levels of pain-related anxiety in NAs led to fewer nociceptive signals being inhibited at the spinal level which resulted in amplified pain perception.

Another pathway to NA pain risk may be through greater exposure to ALEs. Numerous studies have found relationships between ALEs exposure and chronic pain. For example, childhood physical and sexual abuse is associated with greater pain in adulthood [64], as well as a higher prevalence of fibromyalgia [32, 65], chronic pelvic pain [66], and arthritis/rheumatism [67]. Although prospective studies are uncommon, one found that ALEs experienced before age 7 (specifically, physical trauma due to accidents, being institutionalized, maternal separation, and financial family difficulties) predicted chronic pain at age 45 [68]. Studies of pain-free individuals also suggest a linkage between ALEs and chronic pain risk. For example, our lab and others have shown that greater exposure to ALEs is associated with amplification of nociceptive signaling at the spinal level [35, 37] and hyperalgesia [35].

The present study extended this literature to show that exposure to ALEs and psychological distress leads to increased pain-related anxiety and pain catastrophizing (NA ➜ ALEs ➜ psychological distress ➜ catastrophizing/pain-related anxiety). Thus, when taken together with our prior findings from OK-SNAP, NAs may be at greater risk for chronic pain via direct (exposure to ALEs ➜ pronociceptive processes) and indirect (exposure to ALEs ➜ psychological distress ➜ maladaptive cognition and affect ➜ pronociceptive processes) pathways. This is consistent with studies showing ALEs led to several negative psychological and physiological sequelae such as higher risk of affective disorders and hyperactivity of the stress response systems [38, 69].

Notably, the coefficient linking psychological distress to pain catastrophizing was larger than that of distress to pain-related anxiety (distress ➜ situational catastrophizing = .41 vs. distress ➜ pain-related anxiety = .22). This could reflect that situational catastrophizing (i.e., maladaptive thinking patterns) is a more proximal outcome of psychological distress than pain-related anxiety [70]. Although our study cannot confirm this due to its cross-sectional nature, this notion would be consistent with the fear avoidance model that describes catastrophic thoughts as precursor to pain-related anxiety/fear [70]. Future studies need to examine this issue further.

NAs experienced more ALEs and greater psychological distress (stress/psychological symptoms) in our sample. However, NAs did not experience greater psychological distress when controlling for ALE exposure. ALE exposure may therefore be a specific source of psychological distress for NAs. Indeed, ALEs have previously been associated with psychological distress, reduced well-being, and impaired daily activities [71]. Thus, greater psychological distress experienced by NAs may be at least partially explained by increased experiences of adversity. However, it is worth noting that several important NA experiences of historical adversity (e.g., genocide, forced relocation, forced boarding schools) were not assessed by the current study. Thus, future studies are needed to determine the role that historical adversity plays in NA distress and pain risk and whether the effects are similar to the ALEs measured in the current study. Indeed, research suggests that culturally relevant stressors (loss of culture, family ties, land, language) can be a significant source of stress for NAs [72].

Interestingly, NAs reported experiencing more pain-related anxiety, even after controlling for the ALEs-distress relationship. That said, the standardized coefficients linking ethnicity and pain catastrophizing and ethnicity and pain-related anxiety were similar ($\beta = .09$ vs. $\beta = .12$), even though the coefficient for pain catastrophizing was non-significant. Nonetheless, this could imply that, for NAs, anxiety may be partially promoted by other factors.

Clinical Implications

The current study adds to the literature on factors contributing to pain risk in NAs, thereby expanding on potential points for intervention. Given that NAs experience greater rates of adverse life events, chronic pain risk may be decreased by treatment of ALE-related distress (i.e., a reduction in distress is expected to stifle the relationship between ALEs and catastrophizing/anxiety). For instance, individuals with a positive history of ALEs may be offered therapies such as cognitive processing therapy or prolonged exposure therapy which reduce posttraumatic stress symptoms [73]. A cultural adaption of trauma-focused cognitive-behavioral therapy for American Indian and Alaska Natives may be especially suitable for NAs that have been exposed to adversity [74]. Other interventions (e.g., cognitive-behavioral therapies) may target heightened levels of anxiety/catastrophizing that result. Indeed, adolescents who experience ALEs show less psychological distress if they engaged in cognitive reappraisal strategies [75]. Lastly, treatments that target pain catastrophizing and pain-related anxiety may buffer against their pronociceptive effects and offer additional points for intervention (e.g., CBT; Acceptance and Commitment Therapy; [76]).

Potential Limitations

One limitation of this study is the use of the LEC to collect information about ALEs. The LEC does not assess the timing of adversity, whether adverse events occurred once or repeatedly, or the event's severity. Nor does it assess all possible adversity types (e.g., childhood neglect). As a result, we cannot examine the contribution of these factors on our outcomes.

Another limitation is that only healthy, pain-free individuals were recruited for the study so that we could study chronic pain risk factors before disease status confounded the relationships. As a result, this limits the generalizability of our findings to other populations, including those with chronic

pain. Indeed, it is yet to be determined whether NAs and NHWs differ in their levels of pain-related anxiety or catastrophizing in clinical samples. Moreover, by studying only healthy individuals, this may have excluded those at highest risk of developing chronic pain. Nonetheless, preliminary results of our longitudinal surveys (conducted every 6 months) suggest that ~16% of individuals in our sample are developing chronic pain, and NAs develop pain at higher rates than NHWs [77].

Additionally, our NA sample represents tribes mostly from northeastern Oklahoma; however, NAs reside in over 30 different states and there are 573 federally recognized tribes [78]. Therefore, these results may not generalize to NAs from other areas since cultural beliefs and health behaviors may differ by region [79]. We also assessed pain-related anxiety with a single visual analog scale that was averaged across tasks instead of a multi-item measure. While this allowed for rapid measurement in response to each painful event, other anxiety measures may yield different results. However, anxiety has been measured similarly and successfully in prior studies [51, 52]. And finally, these data were collected cross-sectionally and correlational analyses were used. Although the directions of the paths are consistent with theory, causality cannot be inferred.

Summary

This study found that adversity and psychological distress were associated with greater pain-related anxiety and catastrophizing in NAs. These findings are relevant given that pain-related anxiety and catastrophizing have been previously shown to promote pronociceptive processes. Therefore, these findings suggest several targets for intervention to reduce chronic pain risk in NAs.

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Code Availability Not applicable

Authors' Contributions F.A.H. collected data and co-wrote the manuscript for publication. J.L.R. and J.O.S. served as the primary investigators, designed the study, and helped write the manuscript. Moreover, J.L.R. analyzed data for publication. P.K. helped write the manuscript. B.L.K., E.W.L., S.P., M.F.P., N.H., C.A.S., Y.M.G., T.A.T., M.J.D., and B.H. collected data and provided revisions to the manuscript. All authors read and approved the final manuscript.

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Data Availability Means, standard deviations, and intercorrelations necessary to replicate the analyses are provided in the manuscript. Deidentified raw data are available upon request.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethics Approval The study was approved by IRBs of The University of Tulsa, Cherokee Nation, and the Indian Health Service Oklahoma City Area Office.

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