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The role of self-evaluated pain sensitivity as a mediator of objectively measured pain tolerance in Native Americans: findings from the Oklahoma Study of Native American Pain Risk (OK-SNAP)

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Abstract Native Americans (NAs) are at increased risk for chronic pain. One mechanism contributing to this pain disparity could be personal pain beliefs, which may influence actual pain sensitivity. Thus, we examined whether self-evaluated pain sensitivity (SEPS) mediates the relationship between ethnicity [NAs vs. non-Hispanic Whites (NHWs)] and objectively-measured pain tolerance, and whether catastrophic thinking and pain-related anxiety influence these pain beliefs. 232 healthy, pain-free NAs and NHWs completed questionnaires measuring SEPS, catastrophizing, and anxiety. Objective pain tolerance was also assessed. Results suggested: (1) NAs reported higher levels of SEPS, catastrophizing, and anxiety, (2) catastrophizing may have enhanced anxiety and both catastrophizing and anxiety were associated with higher SEPS, and (3) anxiety and SEPS were associated with lower pain tolerance. A significant bootstrapped mediation analysis suggested NAs experienced higher pain-related anxiety, which may have promoted higher SEPS, that in turn reduced pain tolerance. Longitudinal research is needed to confirm this.

Keywords Native American · Chronic pain · Self-evaluated pain sensitivity · Catastrophizing · Pain-related anxiety

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Introduction

Native Americans are at significantly greater risk to develop chronic pain conditions than the rest of the U.S. population (Jimenez et al., 2011; Palit et al., 2013; Pleis et al., 2010). In fact, preliminary analyses of longitudinal data from our lab's Oklahoma Study of Native American Pain Risk (OK-SNAP) suggested that healthy, pain-free Native Americans develop chronic pain at three times the rate of healthy, pain-free non-Hispanic Whites (Ross et al., 2019). Unfortunately, most research regarding this disparity is epidemiological and reports on prevalence rates only. This body of literature has shown that Native Americans experience higher rates of arthritis (Mauldin et al., 2004), chronic headache (Rhee, 2000), back pain (Ross et al., 2019), and neck and shoulder pain (Ross et al., 2019). While multiple explanations have been posited at the societal level (Meghani et al., 2012; Shavers et al., 2010), little is known regarding potential psychological and physiological mechanisms underlying this disparity. Investigating the contributing factors to higher chronic pain rates in Native Americans is crucial to inform intervention efforts.

Experimental pain paradigms can shed insight into future development of chronic pain, as greater sensitivity to experimental pain stimuli is associated with heightened risk for chronic pain (Nielsen et al., 2009; Vaegter & Graven-Nielsen, 2016). Though few studies exist that examine risk for chronic pain in Native Americans, several studies have documented racial/ethnic differences in both experimental pain sensitivity and chronic pain risk across other minority groups. For instance, Green et al. (2003) and Hardt et al. (2008) reported that African Americans are at higher risk to develop chronic pain and were more likely to have worse clinical outcomes due to pain than non-Hispanic Whites. Campbell et al. (2005) reported that African Americans

display lower heat, cold, and ischemic pain tolerances than non-Hispanic Whites. Campbell et al. (2008a) also reported that African Americans tend to have a lower nociceptive flexion reflex (NFR) threshold than non-Hispanic Whites. Given that the NFR is a physiological correlate of pain processing within the spinal cord (Sandrini et al, 2005), this indicates that African Americans may require less noxious input to the spinal cord to achieve a withdrawal reflex, which likely reflects facilitation of spinal cord pain signaling (Campbell et al., 2008b). Campbell et al. (2008b) found that African Americans experienced less pain relief on an experimental measure of pain inhibition processes (i.e., conditioned pain modulation). Rowell et al. (2011) observed lower cold pain threshold and tolerance in individuals of Asian descent, compared to non-Hispanic Whites. Finally, Kim et al. (2017), in a meta-analysis of racial/ethnic differences in experimental pain sensitivity, reported that African Americans, Hispanics, and Asians demonstrated lower pain tolerances, greater pain sensitivity, higher pain ratings, and greater facilitation of pain signaling (i.e., temporal summation of pain).

OK-SNAP was designed to examine whether Native Americans experienced similar risks for chronic pain. The primary findings from OK-SNAP indicated that, compared to non-Hispanic Whites, Native Americans displayed lower cold pain tolerance and greater levels of catastrophic cognitions and anxiety in response to painful stimuli (Rhudy Huber et al., 2020; Rhudy, Lannon, et al., 2020a, b).

The observed differences in pain-related anxiety and catastrophizing are notable, because both can contribute to heightened pain sensations and chronic pain (Bishop et al., 2001; Gracely et al., 2004; Granot & Ferber, 2005; McCracken et al., 1998; Picavet et al., 2002; Rhudy Huber et al., 2020; Somers et al., 2009). For instance, McCracken et al. (1998) reported that pain-related anxiety is the most salient predictor of general physical discomfort in chronic pain patients, and a prospective study by Picavet et al. (2002) indicated that pain catastrophizing at baseline predicts development of chronic back pain at a six-month follow-up. Additionally, Gracely et al. (2004) reported that pain catastrophizing is associated with brain activity in areas responsible for attention, emotion, and expectation. In other words, pain catastrophizing can influence pain perception by increasing the likelihood that individuals will attend to, emotionally respond to, and expect pain (Gracely et al., 2004). Given that anxiety and catastrophizing appear to amplify pain, these cognitive-affective processes may be one way that chronic pain risk is increased in Native Americans.

The above results indicate that cognitive-affective processes may be indirect promoters of chronic pain risk in Native Americans. Consistent with this, we have found that the higher level of pain-related anxiety experienced by Native Americans is associated with lower pain tolerance (Rhudy, Lannon et al., 2020a, b), and higher levels of pain-related anxiety and pain catastrophizing are associated with impaired inhibition of spinal nociceptive signaling (as measured by conditioned pain modulation of the nociceptive flexion reflex) (Rhudy Lannon et al., 2020a, b; Toledo et al., 2020).

Given the potential importance of cognitive-affective processes in chronic pain risk, it is worth considering individuals' beliefs about their own pain sensitivity (i.e., self-evaluated pain sensitivity). Self-evaluated pain sensitivity, as discussed by Ruscheweyh et al. (2009) is an individual's own evaluation of their experienced pain level in response to everyday painful events (e.g., biting your tongue). The Pain Sensitivity Questionnaire (PSQ) was developed by Ruscheweyh et al. (2009) to mimic patients' reporting of subjective responses to clinical pain conditions and has been validated in multiple languages (Azimi et al., 2016; Kim et al., 2017; Quan et al., 2018; Sellers et al., 2013; Valeberg et al., 2017). The PSQ has been validated for use in individuals with chronic pain conditions (Ruscheweyh et al., 2012), and it has been shown to predict postoperative pain (Rehberg et al., 2017; Azimi & Benzel, 2016) and differentiate between chronic pain patients and healthy controls (Coronado, Mackie, Simon & George, 2014). Importantly, the PSQ has been used to identify racial/ethnic differences in self-evaluated pain sensitivity (Bell et al., 2018; Kim et al., 2017; Ostrom et al., 2017; Rowell et al., 2011). These studies indicate that racial/ethnic minorities generally rate themselves as being more pain sensitive (i.e., higher PSQ scores), which is consistent with their greater sensitivity to experimental pain (Bell et al., 2018; Kim et al., 2017; Ostrom et al., 2017; Rowell et al., 2011).

Prior research has found that objectively measured pain sensitivity can be influenced by a multitude of factors, including prior painful experiences, directing attention toward the painful stimulus, environmental surroundings, coping skills, emotional reactions to pain, stimulus modality, preexisting medical conditions or psychopathology, and expectations surrounding painful experiences (Goffaux et al., 2011; Hansen & Streltzer, 2005; Hechler et al., 2016; Petzke et al., 2003; Rutchick & Slepian, 2013). As discussed above, results from OK-SNAP indicated that pain-related anxiety and situation-specific catastrophizing affect objectively measured pain sensitivity in Native Americans (Rhudy Huber et al., 2020; Toledo et al, 2020). Given this, pain-related anxiety and catastrophic cognitions about pain may also influence self-evaluated pain sensitivity. In other words, individuals may construct beliefs about their pain sensitivity based on their cognitive-affective state (see Fig. 1 for a conceptual model). Indeed, research has shown that memory for pain is influenced by cognitive-affective processes (Gedney & Logan, 2004, 2006; Gedney et al., 2003; Noel et al., 2015; Shimo

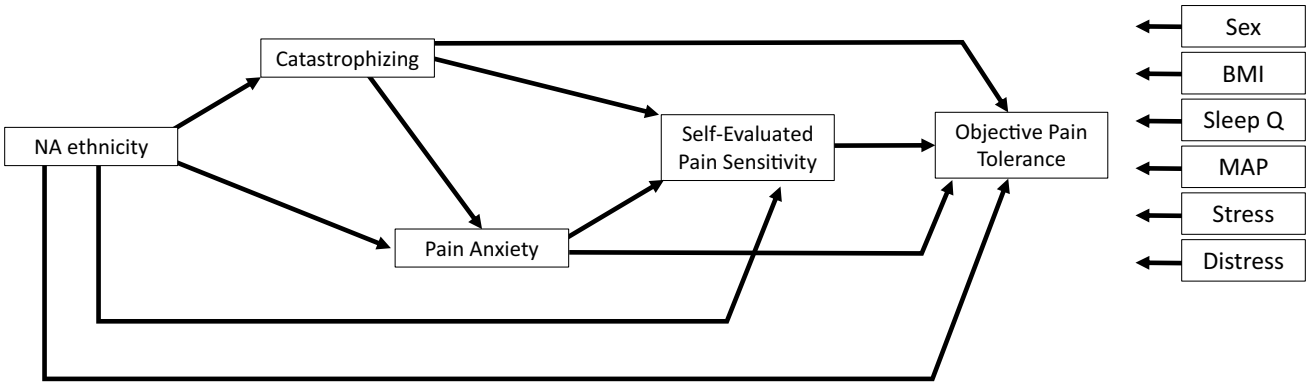

Fig. 1 The hypothetical model linking Native American (NA) ethnicity to objectively measured pain tolerance. We propose that situational pain catastrophizing promotes pain-related anxiety and that both pain catastrophizing and pain-related anxiety lead to higher reports of pain sensitivity. Higher self-evaluated pain sensitivity is hypothesized to be associated with lower objectively measured pain tolerance

et al., 2011). For example, the memory of experienced pain intensity is more strongly influenced by the individual's affective state prior to the painful experience than the actual experienced intensity of the pain itself (Gedney & Logan, 2006; Gedney et al., 2003). These findings are corroborated by brain imaging research that suggests that remembering a painful event activates brain areas commonly associated with both pain and negative emotions (e.g., insula, hippocampus, posterior cingulate cortex; Shimo et al., 2011). In other words, negative affect and cognitions (i.e., pain-related anxiety, catastrophizing) may influence the formation of pain-related memories and therefore, self-evaluated pain sensitivity.

Given these linkages, racial/ethnic differences in self-evaluated pain beliefs may be associated with racial/ethnic differences in pain sensitivity assessed from objective measures (e.g., pain tolerance assessed from quantitative sensory testing). To date, no study has examined whether Native Americans differ from non-Hispanic Whites in self-evaluated pain sensitivity, and if such a discrepancy exists, whether individual differences in this belief are associated with hyperalgesia (lower pain tolerance) in Native Americans, as objectively assessed by laboratory measures of pain tolerance. Therefore, the present study had three primary aims: (1) to determine whether a group difference in self-evaluated pain sensitivity is present between Native Americans and non-Hispanic Whites, (2) to determine whether self-evaluated pain sensitivity is associated with group differences in objective measures of pain tolerance [i.e., to determine whether self-evaluated pain sensitivity provides an indirect (mediated) pathway between race/ethnicity and objective measures of pain tolerance], and (3) to determine the contributions of catastrophizing and pain-related anxiety to self-evaluated pain sensitivity, and therefore, to pain tolerance outcomes.

Methods

Participants

Participants (n = 302) in the Oklahoma Study of Native American Pain Risk (OK-SNAP) were community-dwelling adults recruited via outreach efforts (e.g., flyers, Facebook advertisements, etc.). Native American individuals with a Certificate of Degree of Indian Blood (CDIB) card or tribal membership card and non-Native Americans were eligible for participation. Native American participants represented tribal nations predominately from the southern plains and eastern Oklahoma tribes. Although no one was excluded from the study based on race/ethnicity, non-Native minority participants were excluded from the current analyses (n = 22). Another five participants were excluded for missing data. Demographic characteristics of participants are reported in Table 1.

The purpose of OK-SNAP was to explore potential physiological and psychological risk factors for the development of chronic pain in Native Americans, given that Native Americans develop chronic pain at three times the rate of non-Hispanic Whites (Ross et al., 2019). The OK-SNAP study was prospective in design, meaning only healthy, pain-free individuals were included to determine whether tests of pain processing could identify ethnic differences in chronic pain risk before it developed. As such, participants were excluded for neurological disorders (e.g., neuropathy, history of stroke), cardiovascular disorders (e.g., history of heart attack, arrhythmia, high blood pressure), chronic pain conditions, body mass index over 35, pregnancy, under 18 years of age, substance dependence, and the use of antihypertensives, statins, antidepressants, anxiolytics, or stimulants.

Table 1 Group differences between non-Hispanic Whites and Native Americans on study variables (N = 232)

Study variable	Non-Hispanic White			Native American			*t*-test	*p*-value
	N	*M*	*SD*	*N*	*M*	*SD*		
BMI (kg/m^2)	117	24.33	3.94	115	25.76	4.68	− 2.507	.01
Heat tolerance (°C)	117	45.77	2.02	115	45.57	1.62	0.836	.40
Cold tolerance (log10[sec + 1])	117	1.79	0.42	115	1.66	0.31	2.654	.01
Ischemia tolerance (log10[sec + 1])	117	2.22	0.41	115	2.12	0.45	1.807	.07
Electric tolerance (mA)	117	30.56	13.41	115	31.65	12.72	− 0.634	.53
Mean arterial pressure (mmHg)	117	82.75	8.12	115	88.37	9.58	− 4.815	< .001
Situational catastrophizing (PCS; 0–52)	117	8.56	6.90	115	10.39	7.12	− 1.991	.048
Pain-related anxiety (0–100)	117	34.65	19.84	115	42.66	20.41	− 3.033	< .001
Perceived stress (PSS; 0–40)	117	13.46	5.97	115	14.55	6.29	− 1.349	.18
Psych distress (GSI; 0–4)	117	0.11	0.08	115	0.13	0.09	− 1.73	.09
Sleep quality (PSQI; 0–3)	117	0.96	0.64	115	1.26	0.85	− 3.08	< .001
Self-eval pain Sens (PSQ; 0–10)	117	3.36	1.07	115	3.79	1.25	− 2.831	.01

BMI = body mass index. PCS = Pain Catastrophizing Scale. PSS = Perceived Stress Scale. GSI = Global Severity Index from the SCL-90R. PSQI = Pittsburgh Sleep Quality Index. PSQ = Pain Sensitivity Questionnaire

Study procedure

Data collection occurred between 2014 and 2018. Study procedures were approved by the Institutional Review Boards of the University of Tulsa, Cherokee Nation, and Indian Health Service Oklahoma City Area Office. Both verbal and written consent were obtained prior to initiation of study procedures, and participants were compensated $200 for completion of both days of the study (or $10/hour for every hour completed). Testing was completed over two days, in sessions lasting 4–6 hours. Most stimuli and questionnaires were presented by a computer while an experimenter observed from another room via a live video/audio feed.

The assessment of cold, heat, electric, and ischemia tolerance was pseudo-randomized within one of the testing days with breaks in between tasks to reduce carry over effects. Multiple questionnaires were presented throughout both days to collect various demographic and psychological characteristics relating to pain perception and modulation. A complete account of study procedures can be found elsewhere (Rhudy Lannon et al., 2020a, 2020b).

Questionnaires

Questionnaires were administered to assess self-evaluated pain sensitivity, pain catastrophizing, and pain-related anxiety, as well as several variables used to describe the sample (and determine whether there were racial/ethnic group differences prior to testing). Moreover, some variables were measured to control for possible confounds.

Pain Sensitivity Questionnaire

The Pain Sensitivity Questionnaire (PSQ; Ruscheweyh et al., 2009) was administered during the second day of testing. The PSQ is a brief, 17-item questionnaire on which participants rate their pain experiences on a Likert scale of 0 (no pain) to 10 (pain as bad as it could be) in response to imaginary scenarios (e.g., "Imagine you bump your elbow on the edge of a table"; Ruscheweyh et al., 2009). The PSQ has internal consistency of $\alpha = 0.92$ and test–retest reliability of $r = 0.83$ (Ruscheweyh et al., 2009). Evidence for the validity of this measure is reported in Ruscheweyh et al. (2009) and Sellers et al. (2013). Self-evaluated pain sensitivity was defined as the mean of participants' ratings of the 17 items. Higher scores represent greater self-evaluated pain sensitivity; therefore, higher scores should be associated with lower pain tolerance.

Pain Catastrophizing Scale

The Pain Catastrophizing Scale (PCS; Sullivan et al., 1995) is a 13-item questionnaire in which participants rate the frequency of their pain-related cognitions on a Likert scale of 0–4 (0 = not at all, 4 = all the time). Statements include items such as, "I worry all the time about whether the pain will end," or, "I keep thinking about how badly I want the pain to stop" (Sullivan et al., 1995). The PCS has internal consistency of $\alpha = 0.87$ (Sullivan et al., 1995); evidence for the validity of this measure is reported in Osman et al. (1997). Ratings across items were added to create a total score, with higher scores indicating a greater degree of catastrophizing in response to painful stimuli. Participants completed

the PCS at the beginning of the first testing day prior to any pain tasks to assess trait catastrophizing (i.e., how an individual generally reacts to painful events), but also completed it immediately following all pain tolerance measures to assess situation-specific catastrophizing (Rhudy Lannon et al., 2020a, b; Sullivan et al., 1995). When administered following pain tasks, the instructions of the PCS were modified to ask participants to report on their thoughts during the pain task itself and answer the questions in reference to that task. Trait pain catastrophizing was used in analyses to ensure groups did not significantly differ. Situation-specific pain catastrophizing (averaged across pain tasks) was used as a measure of how participants responded in the moment to pain. Given that studies have found that situation-specific catastrophizing is a better predictor of pain outcomes than trait pain catastrophizing (Campbell et al., 2010; Grosen et al., 2016; Quartana et al., 2009), it was used in primary analyses. For the present study, the situation-specific catastrophizing scores were averaged across pain tasks to generate a global pain catastrophizing score, with higher scores reflecting a higher level of catastrophic cognitions.

Pain-related anxiety

Following each pain task in OK-SNAP, participants were presented with a visual analog scale (VAS) with the anchors "not at all anxious" to "extremely anxious" (Rhudy Huber et al., 2020; Robinson et al., 2010) and the following instructions: "Using this scale, rate how anxious the [insert pain stimulus here] made you feel." Participant responses were electronically converted to scores ranging from 0 to 100 and averaged to create a global measure of pain-related anxiety. Although different stimuli evoked different mean levels of pain-related anxiety, we found that the separate items loaded onto a single component in a principal component analysis, and Cronbach's alpha was high ($\alpha = 0.91$) (Rhudy Huber et al., 2020). Thus, the items were averaged across tasks to generate a global pain-related anxiety score, with higher scores representing higher pain-related anxiety.

Control variables

In the parent study, we found that NAs and NHWs differed on the following variables: body mass index (BMI), self-reported sleep quality, resting blood pressure, perceived stress, and psychological distress. Additionally, there is considerable evidence supporting sex differences in pain tolerance (Bartley & Fillingim, 2013). Given these issues, we controlled for these variables in the primary PROCESS mediation model. BMI was calculated by measuring participants' height and weight on a medical weight scale. The perceived sleep quality item from the Pittsburgh Sleep Quality Index (PSQI) was used to assess differences in sleep (Buysse et al., 1989). The item ranges from 0 (very good) to 3 (very bad), thus higher scores represent poorer sleep. Mean arterial blood pressure (MAP) was assessed from a medical grade instrument (Dinamap; Tampa, FL) 3 times at the beginning of each testing session (3 min inter-test interval) while the participants sat comfortably and quietly in a recliner with their arm resting on the arm of the chair. The average of each variable from the first testing day was used to control for differences in blood pressure. The Perceived Stress Scale (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 (Cohen et al., 1983). The Symptom Checklist-90-Revised (SCL-90-R) was used to assess general psychological distress (Derogatis, 1994). The scale consists of 90 items that assess various psychological symptoms (e.g., somatization, obsessive–compulsive, depression, phobic anxiety, paranoia). The Global Severity Index (GSI) of the SCL-90-R was used to assess overall psychological distress. Total GSI scores range from 0 to 4 with higher scores indicating greater distress.

Objective measures of pain tolerance

Cold pain tolerance

Cold pain tolerance was assessed using a circulating water bath at 6 °C (Thermo Fisher Scientific, Pittsburgh, PA) with the water level set to 6 inches deep to keep procedures standardized and maintain a similar level of cold exposure across participants. Participants were instructed to submerge their hand up to their wrist, with fingers outspread in the water (Campbell et al., 2005; Meagher et al., 2001; Rhudy et al., 2006). Participants continuously rated their sensation on a computer-presented visual analog scale (VAS) ranging from 0 to 100 (0 being "no pain" and 100 being "maximum tolerable pain"). Cold pain tolerance was defined as time (in sec) to reach a rating of 100. Participants were not allowed to keep their hand in the water longer than five minutes, though participants were not informed of this limitation prior to the task.

Heat pain tolerance

Heat pain tolerance was assessed using a Medoc Pathway device (Haifa, Israel) with a Contact Heat Evoked Potential Stimulator (CHEPS) thermode that was attached to participants' volar forearm. The thermode heated up at a rate of 0.5 °C/second from a baseline of 32 °C until participants rated the stimulus as intolerable by pushing a button, which terminated the stimulus. This procedure started with a practice trial to familiarize participants with the task and then pain tolerance was assessed from the average of the last four

trials (Campbell et al., 2005). The thermode was moved between trials to avoid sensitization. For heat pain tolerance, the maximum intensity of the heat stimulus was set to 51 °C.

Ischemia pain tolerance

To first create energy and oxygen demand in the forearm muscle, participants conducted hand exercises once per second for two minutes with a dynamometer (Lafayette Hand Dynamometer; Lafayette Instrument Company, Lafayette, IN) at 50% grip strength. Immediately after completing the hand exercises, participants raised their arm for 15 s to allow blood to drain away from the hand and forearm. A blood pressure cuff was then placed on the biceps muscle and inflated to 220 mmHg to prevent blood flow back to the forearm. Participants were instructed to keep their arm still throughout the task and continuously rate their pain on the same electronic VAS used during cold pain tolerance. The time taken to reach a pain rating of 100 was used to indicate tolerance. Participants were not allowed to keep the cuff around their arm for longer than 25 min, though participants were not informed of this limitation prior to the task.

Electric pain tolerance

Electric pain tolerance was assessed by placing a bipolar electrode (Nicolet; 30 mm inter-electrode distance) filled with conductive gel (EC60; Grass Technologies, West Warwick, RI) over the retromalleolar surface of the left ankle after the skin was cleaned with an alcohol swab and abraded with an exfoliating gel (NuPrep; Weaver and Company, Aurora, CO). Stimulations were delivered by an isolated, constant current stimulator (Digitimer DS7A; Hertfordshire, United Kingdom). Each stimulus constituted a train of five 1-ms rectangular wave pulses with a 3-ms interpulse interval (250 Hz); however, the train was experienced as a single stimulus. The maximum stimulation intensity for electric pain tolerance was set to 50 mA.

In order to determine electric pain tolerance, a single ascending staircase of stimulations was delivered, beginning at 0 mA and increasing in intensity in 2 mA steps. Participants rated their pain after each stimulus on the same electronic VAS described above. The stimulation intensity increased until participants reached a rating of 100, which was also used to determine when tolerance was reached.

Data analysis

The Statistical Package for the Social Sciences v25 (SPSS; IBM Corp) was used for all analyses, unless otherwise stated. Ethnic differences on continuous outcomes were assessed using independent samples t-tests, whereas categorical outcomes were assessed using chi-squared tests. Significance was set at $p < 0.05$ (2-tailed) for these analyses.

A principal component analyses (PCA) was conducted on the four pain tolerance outcomes to reduce into a single outcome variable for the mediation model. The correlations among tolerance outcomes (range of rs = 0.365–0.432), Kaiser–Meyer–Olkin measure of sampling adequacy (KMO = 0.758) and the Bartlett's test of sphericity ($p < 0.001$) all suggested that the correlation matrix was factorable. The scree plot suggested a clear single component should be extracted that explained 55% of the variance in the 4 outcomes (loadings were heat = 0.752, cold = 0.742, ischemia = 0.739, electric = 0.725).

The primary analysis was conducted using the PROCESS macro for SPSS (Hayes, 2017). This was a custom analysis that examined a serial mediation model (Fig. 1). PROCESS allows researchers to compute bootstrapped confidence intervals for all coefficients, including: (1) the paths (unstandardized regression slopes) linking the IV (NA ethnicity) to mediators, (2) the paths linking the mediators to the DV (pain tolerance), and (3) the indirect (mediated) pathways. In all cases, 5000 bootstrapped samples were generated, and the 95% confidence interval was created from these observed samples. The path coefficients are the average of these 5000 bootstrapped samples. The indirect paths are a result of the product (via multiplication) of all unstandardized coefficients that make up the indirect (mediated) path linking one variable to another. Rather than using traditional tests of significance (i.e., *p*-values), significance for coefficients in these models is determined from the bootstrapped 95% confidence interval. If the interval contains zero, then that coefficient is not statistically different from zero (i.e., the null hypothesis is not rejected).

Prior to analyses, SPSS Explore was used to assess the distributional characteristics of all variables. In the event of skewness, it was corrected using a log10 (for positive skew) or square root (for negative skew) transformations. Cold tolerance, ischemia tolerance, and psychological distress were log10 transformed. Next, outliers were identified using Wilcox's MAD-median procedure and then winsorized to the next nearest non-outlier value (Wilcox, 2016). The following variables were winsorized: blood pressure (MAP), perceived stress (PSS), psychological distress (GSI), situational catastrophizing (PCS), self-evaluated pain sensitivity (PSQ), heat tolerance, cold tolerance, and ischemia tolerance.

Results

Final sample

Of the 302 eligible participants in the full OK-SNAP sample, 232 completed the PSQ. T-tests and chi-squared analyses

were conducted to determine if there were group differences on study variables. There were no group differences on biological sex ($p = 0.09$), ethnicity ($p = 0.49$), BMI ($p = 0.91$), heat tolerance ($p = 0.61$), cold tolerance ($p = 0.79$), ischemia tolerance ($p = 0.38$), electric tolerance ($p = 0.42$), MAP ($p = 0.79$), perceived stress ($p = 0.37$), or psychological distress ($p = 0.29$). None of those that failed to complete the PSQ filled out the PSQI, so differences in sleep quality could not be addressed. However, there were significant differences on situational pain catastrophizing ($p = 0.04$) and pain-related anxiety ($p = 0.02$). Non-completers reported greater pain catastrophizing (M = 12.02, SD = 9.26 vs. M = 9.47, SD = 7.05) and pain-related anxiety (M = 46.97, SD = 24.92 vs. M = 38.62, SD = 20.47). Thus, results may not generalize to those that tend to engage in more of these psychological processes.

After excluding persons without the PSQ, there were only a few other missing values (BMI = 3, heat tolerance = 3, cold tolerance = 2, ischemia tolerance = 1, electric tolerance = 1, MAP = 2, perceived stress = 1, psychological distress = 1). Table 1 reports the N's for all study variables and thus illustrates the amount of missingness for each variable. Most missingness was due to participants not attending the second day of testing in the parent study. To avoid listwise deletion due to this missingness, the expectation maximization algorithm in LISREL v8.8 (Scientific Software International; Lincolnwood, IL) was used to impute these missing values (Jöreskog & Sörbom, 2006). Thus, the full sample of 232 was available for PROCESS analysis, which improved statistical power.

Sample characteristics

Table 1 presents ethnic group differences on study variables. As shown, NAs had higher BMIs, MAP, situational pain catastrophizing, pain-related anxiety, and self-evaluated pain sensitivity (PSQ), but lower cold tolerances and sleep quality.

Mediated paths linking Native American ethnicity to objectively measured pain tolerance

Intercorrelations among study variables are presented in Table 2. Figure 2 presents the bootstrapped estimates and 95% confidence intervals for model paths. As shown, there were significant paths linking NA ethnicity to situational

Table 2 *Intercorrelations among study variables following imputation of missing data (N = 232)*

Variable	Native	SScatas	Pain-Anx	Pain-Sens	HeatTol	Cold-Tol	Isch-Tol	Elec-Tol	BMI	SleepQ	MAP	Stress	Psych
NA Ethnicity	1.000												
Pain Catas	.130*	1.000											
Pain Anx	.196*	.742*	1.000										
PSQ	.184*	.394*	.407*	1.000									
Heat Tol	− .055	− .119	− .213*	− .185*	1.000								
Cold Tol	− .172*	− .210*	− .243*	− .315*	.399*	1.000							
Isch Tol	− .118	− .235*	− .307*	− .196*	.432*	.389*	1.000						
Elec Tol	.042	− .138*	− .192*	− .179*	.385*	.407*	.365*	1.000					
BMI	.163*	.099	.154*	.121	.046	− .124	− .160*	.094	1.000				
Sleep Q	.200*	.131*	.119	.180*	.042	.044	.012	.130*	− .005	1.000			
MAP	.303*	.094	.102	.134*	.219*	− .017	.045	.043	.362*	.112	1.000		
Stress	.089	.321*	.229*	.139*	− .084	.092	.004	.087	− .037	.337*	− .021	1.000	
Psych	.113	.366*	.254*	.149*	.001	.077	.030	.075	− .040	.424*	.060	.743*	1.000
Female Sex	.035	− .033	− .012	− .010	− .373*	− .135*	− .161*	− .084	− .129	− .055	− .229*	− .032	− .050

Ethnicity was coded 0 = non-Hispanic Whites, 1 = Native American (NA). Sex was coded 0 = male, 1 = female. Catas = catastrophizing, Anx = anxiety. PSQ = Pain Sensitivity Questionnaire. Tol = tolerance. Isch = ischemia. Elec = electric. BMI = body mass index. Sleep Q = sleep quality from PSQI. MAP = mean arterial pressure. Psych = psychological distress from Global Severity Index of SCL-90R

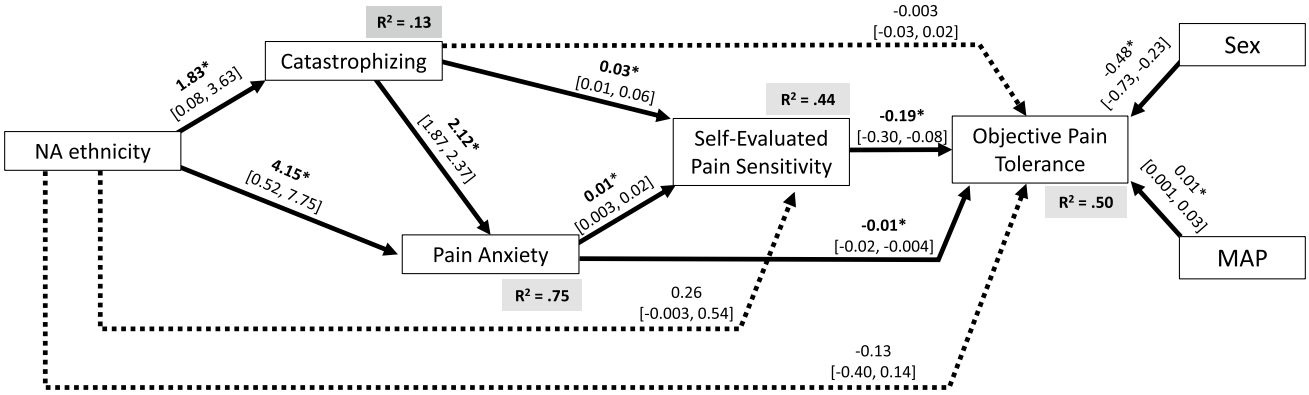

Fig. 2 Results of the PROCESS analyses. Bootstrapped unstandardized path coefficients and their bootstrapped 95% confidence intervals (in brackets) are reported next to model paths. Significant paths are represented by solid arrows, whereas non-significant paths are dashed arrows. R^2 values next to each variable's box indicates the total variance explained. NA = Native American. BMI = body mass index. Sleep Q = sleep quality. MAP = mean arterial blood pressure. Sex was coded 0 = male, 1 = female

pain catastrophizing and pain-related anxiety. This replicates our previous findings that NAs report higher levels of situational pain catastrophizing and pain-related anxiety during painful tasks. Further, there were significant paths linking situational pain catastrophizing with pain-related anxiety and self-evaluated pain sensitivity. These paths indicated that greater pain catastrophizing was associated with greater pain-related anxiety and higher self-reported pain sensitivity. There were also significant paths linking pain-related anxiety to self-evaluated pain sensitivity and objectively measured pain tolerance. Higher pain-related anxiety was associated with greater self-evaluated pain sensitivity and lower pain tolerance. Of the control variables, only biological sex and MAP were significantly related to pain tolerance. Women had lower pain tolerance, and higher blood pressure was associated with higher pain tolerance.

Table 3 presents the results of the bootstrapped indirect (mediated) effects. There were three indirect paths that had a confidence interval that did not contain zero, thus suggesting they were statistically significant. The first path was: NA–> Pain Anx–> Pain Tol. The second path was: NA–> Catas–> Pain Anx–> Pain Tol. The third path was: NA–> Pain Anx–> SE Pain Sens–> Pain Tol. These results suggest that pain-related anxiety is critical for mediating the relationship between NA ethnicity and reduced pain tolerance, but that some of that relationship is due to situational pain catastrophizing promoting pain-related anxiety. And finally, self-evaluated pain sensitivity mediated the relationship between NA ethnicity and pain tolerance as a result of increased pain-related anxiety.

Discussion

Consistent with hypotheses, we found a group difference between Native Americans and non-Hispanic Whites on a

Table 3 Results of PROCESS bootstrapped indirect pathways.

Indirect Pathway	Effect	Boot SE	Bootstrapped 95% CI	
			Lower	Upper
NA –> Catas –> Pain Tol	– 0.007	0.026	– 0.063	0.045
NA –> Pain Anx –> Pain Tol	**– 0.053**	0.031	– 0.126	– 0.004
NA –> SE Pain Sens –> Pain Tol	– 0.050	0.033	– 0.128	0.000
NA –> Catas –> Pain Anx –> Pain Tol	**– 0.050**	0.033	– 0.127	– 0.001
NA –> Catas –> SE Pain Sens –> Pain Tol	– 0.012	0.009	– 0.035	0.000
NA –> Pain Anx –> SE Pain Sens –> Pain Tol	**– 0.011**	0.007	– 0.029	– 0.001
NA –> Catas –> Pain Anx –> SE Pain Sens –> Pain Tol	– 0.010	0.008	– 0.030	0.000

Bolded indirect pathways are statistically significant according to the bootstrapped 95% confidence interval

NA = Native American ethnicity (0 = non-Hispanic White, 1 = Native American). Catas = situational pain catastrophizing. Pain Tol = objectively measured pain tolerance. Pain Anx = pain-related anxiety. SE Pain Sens = self-evaluated pain sensitivity (assessed from the Pain Sensitivity Questionnaire, higher scores mean more sensitive to pain). CI = confidence interval. Effect = indirect effect. If the bootstrapped 95% confidence interval does not contain zero, this suggests a significant indirect effect linking NA ethnicity to objective pain tolerance via the indirect path.

measure of self-evaluated pain sensitivity, such that Native Americans reported that they were more sensitive to every-day painful events than non-Hispanic Whites. Additionally, this difference was associated with lower pain tolerance for Native Americans on experimental pain tests. Both catastrophizing and pain-related anxiety were associated with reports of lower self-evaluated pain sensitivity. In other words, cognitive and emotional reactions to pain were possible promoters of elevated self-evaluated pain sensitivity in Native Americans (Lumley et al., 2011; Quartana et al., 2009; Sullivan et al., 2001). Our mediation analyses indicated that, while both catastrophizing and pain-related anxiety may contribute to lower pain tolerance in Native Americans, catastrophizing does so indirectly by increasing pain-related anxiety. Moreover, the effect of pain-related anxiety on objective pain tolerance was partially mediated by self-reported pain sensitivity. Implications of these findings are discussed below.

The finding that Native Americans perceived themselves as more sensitive to painful stimuli under every-day circumstances aligns with multiple studies documenting greater sensitivity to pain in ethnic minorities (Bell et al., 2018; Callister, 2003; Kim et al., 2017; Ostrom et al., 2017; Rahim-Williams et al., 2012). However, the results of this paper are unique for two reasons. First, to the authors' knowledge, no prior study has examined self-evaluated pain sensitivity among Native Americans. Second, this study determined that beliefs about one's pain sensitivity are associated with reactions to painful stimuli under experimental conditions, which we observed in our mediation model. In other words, perceiving oneself as more sensitive to painful events may decrease the ability to tolerate pain. However, a causal relationship cannot be inferred without a longitudinal study. It is also plausible that people are accurate reporters of their pain sensitivity; thus, their objective pain tolerance aligns with their self-evaluated pain sensitivity.

This study examined two variables that potentially contribute to the development of pain-related beliefs, i.e., pain-related anxiety and situation-specific pain catastrophizing. The results of our mediation model may suggest Native Americans' self-referential pain-related beliefs are constructed with negative emotions and cognitions as building blocks. Further, our model may indicate that pain catastrophizing influences self-evaluated pain sensitivity while also serving to increase pain-related anxiety, which may then lead to reports of higher pain sensitivity, and therefore lower objectively measured pain tolerance. Lumley et al. (2011) clarify that pain-related anxiety is the result of anticipating pain and can be accompanied by hypervigilance and increased pain sensitivity. Pain catastrophizing can serve to direct attentional resources to pain sensations, as well as generate feelings of helplessness regarding ability to control pain sensations (Lumley et al., 2011). Both of these processes are associated with maladaptive coping and poor adjustment to acute and chronic pain (Lumley et al., 2011). In other words, pain-related anxiety and catastrophizing may shape the formation of Native Americans' beliefs about their pain sensitivity, which may then negatively impact their ability to tolerate and cope with pain. However, it is also possible that alternative processes are at play in the formation of individuals' beliefs about their own sensitivity to pain, such as past experiences and family modeling. As such, Native Americans who believe they are more sensitive to painful stimuli may also be more likely to experience pain-related anxiety or catastrophic thoughts about pain. These variables may also reciprocally interact with each other (e.g., catastrophizing may heighten pain-related anxiety, which may increase sensitivity, which may produce more catastrophic cognitions, etc.). Therefore, no concrete conclusions regarding the direction of the relationship among self-evaluated pain sensitivity, pain-related anxiety, and pain catastrophizing can be drawn without a longitudinal study.

Importantly, the findings of this study are unique in that situation-specific catastrophizing and pain-related anxiety have not been considered in the same model when predicting Native American pain tolerance outcomes, though we have formerly demonstrated that Native Americans experience greater pain-related anxiety than non-Hispanic White participants (Rhudy Huber et al., 2020). It appears that, when considered together, catastrophizing does not directly impact Native Americans' pain tolerance. Rather, catastrophizing may serve to heighten pain-related anxiety, which then provides a mediated effect from ethnicity to pain tolerance. In other words, pain catastrophizing may impact pain tolerance by enhancing pain-related anxiety, which then appears to reduce pain tolerance in Native Americans. Additionally, though potentially influenced by both catastrophic cognitions and negative emotions, self-evaluated pain sensitivity provides a mediated pathway from ethnicity to pain tolerance only via pain-related anxiety. In other words, our model may suggest that Native Americans experience greater pain-related anxiety (possibly enhanced by situation-specific pain catastrophizing), which may produce higher evaluations of subjective pain sensitivity, which then may result in lower pain tolerance.

There may be more pieces to the puzzle not considered in this study. Zborowski (1952) noted that broad-scale cultural attitudes and norms cannot be discounted when examining ethnic differences in pain sensitivity. Indeed, Rahim-Williams et al. (2007) reported that a strong sense of belonging within a particular ethnic group predicts greater pain sensitivity to experimental pain stimuli. Anderson and Losin (2017), in their neurocultural pain model, demonstrated how various cultural factors and experiences (e.g., discrimination, historical trauma, prejudice, genetic variation, coping norms, spirituality, social norms) have additive effects on

downstream emotional reactions to pain and verbal descriptions of pain. Given that these neurocultural factors are relevant for Native Americans, some of these other variables should be considered in future studies (Rhudy Huber et al., 2020; Rhudy Lannon et al., 2020a, b).

Taken together, these findings provide insight into Native Americans' heightened risk for chronic pain. We have demonstrated that self-evaluated pain sensitivity may be influenced by emotional and cognitive appraisals of pain, which may then affect ability to tolerate pain. Pain-related anxiety and pain catastrophizing, then, may provide targets for clinical interventions aimed at improving pain tolerance and enhancing general coping strategies for those with chronic pain conditions. Additionally, our model may suggest that targeting pain catastrophizing first may be indicated, given that catastrophizing seems to be an upstream promotor of pain-related anxiety.

Strengths and limitations

OK-SNAP is the largest and most comprehensive study of Native American pain processing to date. Additionally, the study employed multiple stimulus modalities in order to assess pain tolerance (cold, heat, ischemia, and electric). The design of the study was such that experimenter effects were minimized; most stimuli and questionnaires were presented by a computer, while an experimenter observed from another room via a live video/audio feed. And finally, we used state-of-the-art bootstrapped sampling to evaluate the mediated model. However, there are a few limitations that should be reviewed.

First, this study included only healthy, pain-free participants. Therefore, our results may not generalize to individuals with chronic pain diagnoses or other health conditions. Second, our Native American participants were recruited from northeastern Oklahoma; therefore, it is uncertain whether our results will generalize to Native Americans from other regions. Third, Thorn and Williams (1989) state that the phrasing of instructions for pain tolerance tasks can affect both participant pain ratings and latency to end the task. Therefore, it is possible that the instructions given to participants prior to pain tolerance tasks affected their performance in some way (i.e., tolerating the stimulus for longer than they might under everyday circumstances outside the laboratory). However, care was taken to deliver instructions in a standardized manner to minimize experimenter effects, as mentioned above. Finally, this study was cross-sectional in nature and was unable to identify a causal relationship or the direction of the observed effects. We based the order of our effects on theoretically sound logic. Nonetheless, our model or its interpretation could be spurious, in that it assumes cognitive-affective variables precede self-evaluated pain sensitivity, and that self-evaluated pain sensitivity preceded objectively measured pain sensitivity. We have attempted to reflect this important limitation throughout by avoiding language that would imply causality.

Conclusion

This study found that Native Americans and non-Hispanic Whites differ in self-evaluated pain sensitivity, such that Native Americans tend to view themselves as more sensitive to pain. This subjective evaluation of pain sensitivity mediated the relationship between race/ethnicity and pain tolerance measures. In other words, Native Americans tended to exhibit lower tolerance to experimental pain stimuli than non-Hispanic Whites, a difference which may have been influenced by greater self-evaluated pain sensitivity in Native Americans. Interestingly, racial/ethnic differences in self-evaluated pain sensitivity may have been driven primarily by differences in pain-related anxiety and situation-specific pain catastrophizing. These results were consistent with prior documentation of greater pain-related anxiety and pain catastrophizing in Native Americans (Rhudy Huber et al., 2020; Rhudy Lannon et al., 2020a, b). Specifically, pain catastrophizing may have served to increase pain-related anxiety, which then appeared to promote higher self-evaluated pain sensitivity and lower pain tolerance. However, future research should confirm the proposed temporal relationship between self-evaluated pain sensitivity and pain tolerance. Answering these questions will aid in ascertaining the validity of self-evaluated pain sensitivity as a predictor of chronic pain risk in Native Americans.

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Declarations

Conflict of interest Erin N. Ross, Tyler A. Toledo, Felicitas Huber, Parker A. Kell, Natalie Hellman, Joanna O. Shadlow, Jamie L. Rhudy declares they do not have any conflict of interest.

Ethical approval Study procedures were approved by the Institutional Review Boards of the University of Tulsa, Cherokee Nation, and Indian Health Service Oklahoma City Area Office.

Human and animal consent All procedures followed were in accordance with ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000.

Informed consent Both verbal and written consent were obtained prior to initiation of study procedures.

References

Anderson, S. R., & Losin, E. A. R. (2017). A sociocultural neuroscience approach to pain. *Culture and Brain, 5*, 14–35

Azimi, P., Azhari, S., Shahzadi, S., Nayeb Aghaei, H., Mohammadi, H. R., & Montazeri, A. (2016). Outcome measure of pain in patients with lumbar disc herniation: Validation study of the Iranian version of Pain Sensitivity Questionnaire. *Asian Spine Journal, 10*, 480–487

Bartley, E. J., & Fillingim, R. B. (2013). Sex differences in pain: A brief review of clinical and experimental findings. *BJA: British Journal of Anaesthesia, 111*, 52–58

Bell, B. A., Ruscheweyh, R., Kelley, B. J., Ness, T. J., Vetter, T. R., & Sellers, A. B. (2018). Ethnic differences identified by Pain Sensitivity Questionnaire correlate with clinical pain responses. *Regional Anesthesia & Pain Medicine, 43*, 200–204.

Bishop, K. L., Holm, J. E., Borowiak, D. M., & Wilson, B. A. (2001). Perceptions of pain in women with headache: A laboratory investigation of the influence of pain-related anxiety and fear. *Headache: The Journal of Head and Face Pain, 41*, 494–499

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193–213

Callister, L. C. (2003). Cultural influences on pain perceptions and behaviors. *Home Health Care Management & Practice, 15*, 207–211

Campbell, C. M., Edwards, R. R., & Fillingim, R. B. (2005). Ethnic differences in responses to multiple experimental pain stimuli. *Pain, 113*, 20–26

Campbell, C. M., France, C. R., Robinson, M. E., Logan, H. L., Geffken, G. R., & Fillingim, R. B. (2008a). Ethnic differences in the nociceptive flexion reflex (NFR). *Pain, 134*, 91–96

Campbell, C. M., France, C. R., Robinson, M. E., Logan, H. L., Geffken, G. R., & Fillingim, R. B. (2008b). Ethnic differences in diffuse noxious inhibitory controls (DNIC). *The Journal of Pain : Official Journal of the American Pain Society, 9*, 759–766

Campbell, C. M., Kronfli, T., Buenaver, L. F., Smith, M. T., Berna, C., Haythornthwaite, J. A., & Edwards, R. R. (2010). Situational vs. dispositional measurement of catastrophizing: Associations with pain responses in multiple samples. *The Journal of Pain: Official Journal of the American Pain Society, 11*, 443–453

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*, 385–396

Coronado, R., Mackie, L., Simon, C., & George, S. (2014). (180) Pain sensitivity questionnaire differentiates individuals with shoulder pain who demonstrate widespread sensitivity with quantitative sensory testing. *The Journal of Pain, 15*(4), S21.

Derogatis, L. R. (1994). *SCL-90-R: Administration, scoring and procedures manual* (3rd ed.). NCS Pearson.

Gedney, J. J., & Logan, H. (2004). Memory for stress-associated acute pain. *The Journal of Pain, 5*, 83–91

Gedney, J. J., & Logan, H. (2006). Pain related recall predicts future pain report. *Pain, 121*, 69–76

Gedney, J. J., Logan, H., & Baron, R. S. (2003). Predictors of short-term and long-term memory of sensory and affective dimensions of pain. *The Journal of Pain, 4*, 47–55

Goffaux, P., Michaud, K., Gaudreau, J., Chalaye, P., Rainville, P., & Marchand, S. (2011). Sex differences in perceived pain are affected by an anxious brain. *Pain, 152*, 2065–2073

Gracely, R. H., Geisser, M. E., Giesecke, T., Grant, M. A. B., Petzke, F., Williams, D. A., & Clauw, D. J. (2004). Pain catastrophizing and neural responses to pain among persons with fibromyalgia. *Brain, 127*, 835–843

Granot, M., & Ferber, S. G. (2005). The roles of pain catastrophizing and anxiety in the prediction of postoperative pain intensity: A prospective study. *The Clinical Journal of Pain, 21*, 439–445

Green, C. R., Baker, T. A., Smith, E. M., & Sato, Y. (2003). The effect of race in older adults presenting for chronic pain management: A comparative study of black and white Americans. *The Journal of Pain, 4*, 82–90

Grosen, K., Drewes, A. M., Pilegaard, H. K., Pfeiffer-Jensen, M., Brock, B., & Vase, L. (2016). Situational but not dispositional pain catastrophizing correlates with early postoperative pain in pain-free patients before surgery. *The Journal of Pain, 17*, 549–560

Hansen, G. R., & Streltzer, J. (2005). The psychology of pain. *Emergency Medicine Clinics, 23*(2), 339–348.

Hardt, J., Jacobsen, C., Goldberg, J., Nickel, R., & Buchwald, D. (2008). Prevalence of chronic pain in a representative sample in the United States. *Pain Medicine, 9*, 803–812

Hayes, A. F. (2017). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. Guilford Publications.

Hechler, T., Endres, D., & Thorwart, A. (2016). Why harmless sensations might hurt in individuals with chronic pain: About heightened prediction and perception of pain in the mind. *Frontiers in Psychology*. https://doi.org/10.3389/fpsyg.2016.01638

Jimenez, N., Garroutte, E., Kundu, A., Morales, L., & Buchwald, D. (2011). A review of the experience, epidemiology, and management of pain among American Indian, Alaska Native, and Aboriginal Canadian peoples. *The Journal of Pain : Official Journal of the American Pain Society, 12*, 511–522

Jöreskog, K. G., & Sörbom, D. (2006). LISREL 8.80. *Chicago: Scientific Software International*.

Kim, H. J., Yang, G. S., Greenspan, J. D., Downton, K. D., Griffith, K. A., Renn, C. L., Johantgen, M., & Dorsey, S. G. (2017). Racial and ethnic differences in experimental pain sensitivity: Systematic review and meta-analysis. *Pain, 158*, 194–211

Lumley, M. A., Cohen, J. L., Borszcz, G. S., Cano, A., Radcliffe, A. M., Porter, L. S., Schubiner, H., & Keefe, F. J. (2011). Pain and emotion: A biopsychosocial review of recent research. *Journal of Clinical Psychology, 67*, 942–968

Mauldin, J., Cameron, H. D., Jeanotte, D., Solomon, G., & Jarvis, J. N. (2004). Chronic arthritis in children and adolescents in two Indian health service user populations. *BMC Musculoskeletal Disorders, 5*, 30

McCracken, L. M., Faber, S. D., & Janeck, A. S. (1998). Pain-related anxiety predicts non-specific physical complaints in persons with chronic pain. *Behaviour Research and Therapy, 36*, 621–630

Meagher, M. W., Arnau, R. C., & Rhudy, J. L. (2001). Pain and emotion: Effects of affective picture modulation. *Psychosomatic Medicine, 63*, 79–90

Meghani, S. H., Byun, E., & Gallagher, R. M. (2012). Time to take stock: A meta-analysis and systematic review of analgesic

treatment disparities for pain in the United States. *Pain Medicine, 13*, 150–174

Nielsen, C. S., Staud, R., & Price, D. D. (2009). Individual differences in pain sensitivity: Measurement, causation, and consequences. *The Journal of Pain, 10*, 231–237

Noel, M., Palermo, T. M., Chambers, C. T., Taddio, A., & Hermann, C. (2015). Remembering the pain of childhood: Applying a developmental perspective to the study of pain memories. *Pain, 156*, 31–34

Osman, A., Barrios, F. X., Kopper, B. A., Hauptmann, W., Jones, J., & O'Neill, E. (1997). Factor structure, reliability, and validity of the Pain Catastrophizing Scale. *Journal of Behavioral Medicine, 20*, 589–605

Ostrom, C., Bair, E., Maixner, W., Dubner, R., Fillingim, R. B., Ohrbach, R., Slade, G. D., & Greenspan, J. D. (2017). Demographic predictors of pain sensitivity: Results from the OPPERA Study. *The Journal of Pain : Official Journal of the American Pain Society, 18*, 295–307

Palit, S., Kerr, K. L., Kuhn, B. L., Terry, E. L., DelVentura, J. L., Bartley, E. J., Shadlow, J. O., & Rhudy, J. L. (2013). Exploring pain processing differences in Native Americans. *Health Psychology, 32*, 1127–1136

Petzke, F., Clauw, D. J., Ambrose, K., Khine, A., & Gracely, R. H. (2003). Increased pain sensitivity in fibromyalgia: Effects of stimulus type and mode of presentation. *Pain, 105*, 403–413.

Picavet, H. S. J., Vlaeyen, J. W. S., & Schouten, J. S. A. G. (2002). Pain catastrophizing and kinesiophobia: Predictors of chronic low back pain. *American Journal of Epidemiology, 156*, 1028–1034

Pleis, J. R., Ward, B. W., & Lucas, J. W. (2010). Summary health statistics for U.S. adults: National Health Interview Survey, 2009. *Vital and Health Statistics. Series 10 Data from the National Health Survey, 249*, 1–207

Quan, X., Fong, D. Y. T., Leung, A. Y. M., Liao, Q., Ruscheweyh, R., & Chau, P. H. (2018). Validation of the Mandarin Chinese version of the Pain Sensitivity Questionnaire. *Pain Practice, 18*, 180–193

Quartana, P. J., Campbell, C. M., & Edwards, R. R. (2009). Pain catastrophizing: A critical review. *Expert Review of Neurotherapeutics, 9*, 745–758

Rahim-Williams, B., Riley, J. L., Williams, A. K. K., & Fillingim, R. B. (2012). A quantitative review of ethnic group differences in experimental pain response: Do biology, psychology, and culture matter? *Pain Medicine, 13*, 522–540

Rahim-Williams, F. B., Riley, J. L., Herrera, D., Campbell, C. M., Hastie, B. A., & Fillingim, R. B. (2007). Ethnic identity predicts experimental pain sensitivity in African Americans and Hispanics. *Pain, 129*, 177–184

Rehberg, B., Mathivon, S., Combescure, C., Mercier, Y., & Savoldelli, G. L. (2017). Prediction of acute postoperative pain following breast cancer surgery using the pain sensitivity questionnaire. *The Clinical Journal of Pain, 33*(1), 57–66.

Rhee, H. (2000). Prevalence and predictors of headaches in US adolescents. *Headache: The Journal of Head and Face Pain, 40*, 528–538

Rhudy, J. L., Dubbert, P. M., Parker, J. D., Burke, R. S., & Williams, A. E. (2006). Affective modulation of pain in substance-dependent veterans. *Pain Medicine, 7*, 483–500

Rhudy, J. L., Huber, F., Kuhn, B. L., Lannon, E. W., Palit, S., Payne, M. F., Hellman, N., Sturycz, C. A., Güereca, Y. M., Toledo, T. A., Demuth, M. J., Hahn, B. J., & Shadlow, J. O. (2020). Pain-related anxiety promotes pronociceptive processes in Native Americans: Bootstrapped mediation analyses from the Oklahoma Study of Native American Pain Risk. *Pain Reports, 5*, e808

Rhudy, J. L., Lannon, E. W., Kuhn, B. L., Palit, S., Payne, M. F., Sturycz, C. A., Hellman, N., Güereca, Y. M., Toledo, T. A., Huber, F., Demuth, M. J., Hahn, B. J., Chaney, J. M., & Shadlow, J. O. (2020b). Assessing peripheral fibers, pain sensitivity, central sensitization, and descending inhibition in Native Americans: Main findings from the Oklahoma Study of Native American Pain Risk. *Pain, 161*, 388–404

Robinson, M. E., Bialosky, J. E., Bishop, M. D., Price, D. D., & George, S. Z. (2010). Supra-threshold scaling, temporal summation, and after-sensation: Relationships to each other and anxiety/fear. *Journal of Pain Research, 3*, 25–32

Ross, E., Huber, F., Kuhn, B., Lannon, E., Sturycz, C., Payne, M., Hellman, N., Toledo, T., Güereca, Y., Palit, S., Demuth, M., Shadlow, J., & Rhudy, J. (2019). (232) Assessing chronic pain onset in Native Americans: Follow-up results from the Oklahoma Study of Native American Pain Risk (OK-SNAP. *The Journal of Pain, 20*, S33

Rowell, L. N., Mechlin, B., Ji, E., Addamo, M., & Girdler, S. S. (2011). Asians differ from non-Hispanic Whites in experimental pain sensitivity. *European Journal of Pain (london, England), 15*, 764–771

Ruscheweyh, R., Marziniak, M., Stumpenhorst, F., Reinholz, J., & Knecht, S. (2009). Pain sensitivity can be assessed by self-rating: Development and validation of the Pain Sensitivity Questionnaire. *Pain, 146*, 65–74

Rutchick, A. M., & Slepian, M. L. (2013). Handling ibuprofen increases pain tolerance and decreases perceived pain intensity in a cold pressor test. *PLoS ONE, 8*, e56175

Sandrini, G., Serrao, M., Rossi, P., Romaniello, A., Cruccu, G., & Willer, J. C. (2005). The lower limb flexion reflex in humans. *Progress in Neurobiology, 77*, 353–395

Sellers, A. B., Ruscheweyh, R., Kelley, B. J., Ness, T. J., & Vetter, T. R. (2013). Validation of the english language pain sensitivity questionnaire. *Regional Anesthesia and Pain Medicine, 38*, 508–514

Shavers, V., Bakos, A., & Sheppard, V. (2010). Race, ethnicity, and pain among the U.S. adult population. *Journal of Health Care for the Poor and Underserved, 21*, 177–220.

Shimo, K., Ueno, T., Younger, J., Nishihara, M., Inoue, S., Ikemoto, T., Taniguchi, S., & Ushida, T. (2011). Visualization of painful experiences believed to trigger the activation of affective and emotional brain regions in subjects with low back pain. *PLoS ONE, 6*, e26681

Somers, T. J., Keefe, F. J., Pells, J. J., Dixon, K. E., Waters, S. J., Riordan, P. A., Blumenthal, J. A., McKee, D. C., LaCaille, L., Tucker, J. M., Schmitt, D., Caldwell, D. S., Kraus, V. B., Sims, E. L., Shelby, R. A., & Rice, J. R. (2009). Pain catastrophizing and pain-related fear in osteoarthritis patients: Relationships to pain and disability. *Journal of Pain and Symptom Management, 37*, 863–872

Sullivan, M. J., Bishop, S. R., & Pivik, J. (1995). The pain catastrophizing scale: Development and validation. *Psychological Assessment, 7*, 524

Sullivan, M. J. L., Thorn, B., Haythornthwaite, J. A., Keefe, F., Martin, M., Bradley, L. A., & Lefebvre, J. C. (2001). Theoretical perspectives on the relation between catastrophizing and pain. *The Clinical Journal of Pain, 17*, 52–64

Thorn, B. E., & Williams, G. A. (1989). Goal specification alters perceived pain intensity and tolerance latency. *Cognitive Therapy and Research, 13*, 171–183

Toledo, T. A., Kuhn, B. L., Payne, M. F., Lannon, E. W., Palit, S., Sturycz, C. A., Hellman, N., Güereca, Y. M., Demuth, M. J., Huber, F., Shadlow, J. O., & Rhudy, J. L. (2020). The effect of pain catastrophizing on endogenous inhibition of pain and spinal nociception in Native Americans: Results from the Oklahoma Study of Native American Pain Risk. *Annals of Behavioral Medicine, 54*, 575–594

Vaegter, H. B., & Graven-Nielsen, T. (2016). Pain modulatory phenotypes differentiate subgroups with different clinical and experimental pain sensitivity. *Pain, 157*, 1480–1488

Valeberg, B. T., Pedersen, L. M., Girotto, V., Christensen, V. L., & Stubhaug, A. (2017). Validation of the Norwegian Pain Sensitivity Questionnaire. *Journal of Pain Research, 10*, 1137–1142

Wilcox, R. R. (2016). *Understanding and applying basic statistical methods using R*. Wiley.

Zborowski, M. (1952). Cultural components in responses to pain. *Journal of Social Issues, 8*, 16–30

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