Does Pain Catastrophizing Moderate the Relationship Between Spinal Nociceptive Processes and Pain Sensitivity?

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Abstract: Existing evidence indicates that pain catastrophizing is associated with enhanced pain reports and lower pain threshold/tolerance levels, but is not significantly related to nociceptive flexion reflex (NFR) threshold in healthy and clinical pain samples. This suggests pain catastrophizing may modulate pain threshold at a supraspinal level without influencing descending modulation of spinal nociceptive inputs. To examine this issue further, the present study assessed NFR threshold, electrocutaneous pain threshold, and electrocutaneous pain tolerance, as well as subjective ratings of noxious stimuli in a sample of 105 healthy adults. Pain catastrophizing was assessed prior to testing using traditional instructions and after pain testing with instructions to report on cognitions during testing (situation-specific catastrophizing). As expected, NFR threshold was correlated with pain sensitivity measures, but uncorrelated with both measures of catastrophizing. Although situation-specific catastrophizing was correlated with some pain outcomes, neither catastrophizing measure (traditional or situation specific) moderated the relationship between NFR and pain sensitivity. These findings confirm and extend existing evidence that catastrophizing influences pain reports through supraspinal mechanisms (eg, memory, report bias, attention) without altering transmission of spinal nociceptive signals.

Perspective: Assessing catastrophic thoughts related to a specific painful event (situation-specific catastrophizing) provides important additional information regarding the negative cognitions that influence pain-related processes. However, neither situation-specific nor traditionally measured pain catastrophizing appear to enhance pain by engaging descending controls to influence spinal nociceptive processes.

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Key words: Coping, catastrophizing, nociceptive flexion reflex, pain perception, electrocutaneous stimulation, pain threshold, pain tolerance.

Pain catastrophizing is an important predictor of pain experience, yet the mechanisms remain poorly understood. Recent evidence suggests the manner by which pain catastrophizing is measured can influence the strength of the relationship with pain outcomes. In particular, the relationship between catastrophizing and pain is stronger when participants are asked to report on catastrophic cognitions during painful stimulation vs pain catastrophizing during daily life. The label “in vivo” catastrophizing has been used to describe retrospective reports of catastrophic thoughts during pain testing, however, we will use “situation-specific” to distinguish retrospective report from “real time” assessment of cognitions during pain.

Numerous studies indicate catastrophizing enhances pain perception/sensitivity, however, it does not appear to do so by engaging descending controls that modulate spinal nociception. Specifically, 3 studies have failed to observe an association between catastrophizing and the nociceptive flexion reflex (NFR) threshold, a measure of spinal nociceptive processes that is typically...
correlated with pain threshold but can be modulated by psychological factors.\textsuperscript{27,36,41} If catastrophizing directly engages descending controls, the threshold to elicit the NFR should be lower in high catastrophizers. Not only is this direct relationship unsupported,\textsuperscript{18,19,39} but it also appears that catastrophizing does not indirectly engage descending controls via emotional processes (eg, increasing negative affect-induced hyperalgesia).\textsuperscript{5} While 2 of these studies did assess catastrophizing after pain testing,\textsuperscript{5,39} instructions were not altered to specifically assess cognitions that occurred during testing. Thus, a formal test of the relationship between situation-specific catastrophizing and NFR is needed.

Studies of temporal summation, a process believed mediated by second-order spinal neurons,\textsuperscript{33,34} have observed a significant relationship with catastrophizing.\textsuperscript{15,20,23} While this observation would suggest catastrophizing influences spinal nociception, it cannot be firmly concluded because these studies assessed temporal summation by subjective report. By contrast, brain imaging during noxious stimulation suggests a correlation between catastrophizing and activation of supraspinal areas associated with attention, anticipation, affect, and motor responses to pain.\textsuperscript{22,42} Therefore, catastrophizing may amplify nociception supraspinally without influencing descending modulation of nociceptive inputs (ie, NFR threshold).

Thus, based on evidence of a relationship between catastrophizing and pain threshold but not NFR threshold, one could assert 2 competing hypotheses (Fig 1). The “moderator hypothesis” suggests pain threshold is significantly correlated with nociceptive threshold in low catastrophizers, but the correlation between pain threshold and nociceptive threshold is attenuated or absent in high catastrophizers because they report lower pain thresholds regardless of nociceptive input (ie, NFR \times\textsuperscript{15} Catastrophizing interaction). Alternatively, the “independent-influences hypothesis” suggests pain catastrophizing and ascending nociceptive input (as assessed by NFR) have independent effects on pain threshold (ie, both main effects are significant). Ostensibly, situation-specific catastrophizing should have a greater effect, regardless of the hypothesis supported.

The present study extends our previous research\textsuperscript{39} by formally assessing situation-specific catastrophizing and adding an assessment of electrocutaneous pain threshold/tolerance to allow a test of the competing hypotheses. These data were collected during a larger study assessing emotional processing and modulation of nociception, but we carefully embedded the current procedures to explicitly test our hypotheses. Specifically, pain catastrophizing was assessed before (traditional instructions) and after (situation-specific instructions) assessment of NFR threshold and electrocutaneous pain threshold and tolerance. The aims of the study were to 1) assess the relationship between 2 methods of pain catastrophizing measurement (traditional and situation specific) and both NFR and pain threshold, and 2) determine whether the “moderator” or “independent influence” hypothesis best describes the relationship between these variables. Sex,\textsuperscript{11,17,47} age,\textsuperscript{13} and depressive symptoms\textsuperscript{1,44-46} were controlled for in the analyses, because they are known to influence catastrophizing and/or pain. Portions of this paper were presented at the 2008 annual meeting of the American Pain Society.\textsuperscript{40}

**Methods**

**Participants**

Participants were recruited from the Tulsa community by fliers, email distribution, and newspaper advertisements. Participants were excluded for the following: < 18 yrs old; self-reported history of neurological, cardiovascular, and/or circulatory problems; use of over-the-counter analgesics in the previous 24 hours or
prescription analgesics in the previous 2 weeks; current use of antidepressant, anxiolytic, or antihypertensive medications; specific phobia of snakes or spiders (due to picture viewing in other unrelated phases of the study; see Procedure section); any diagnosis of chronic pain; recent psychological trauma as defined by the DSM-IV; or Raynaud’s disease. Participants who completed initial pain testing (NFR threshold, electrocutaneous-pain threshold and tolerance) are included in the present analyses (n = 105). An additional 19 participants were enrolled, but not included in analyses due to: equipment problems (n = 2), withdrawal before pain testing was completed (n = 12), or failure to elicit the NFR (n = 5). Of the 105 included, most were female (60%), white (73%), single (64%), and employed (77%), with an average age of 32 years (SD = 14). Additional characteristics of participants are reported in Table 1. Participants were paid $100 for completing all study procedures. All participants provided written and verbal informed consent after the study procedures were fully described to them.

Apparatus

Data acquisition, as well as stimulus and questionnaire presentation, were controlled by a PC equipped with dual monitors, A/D board (PCI-6036E; National Instruments, Austin, TX), and LabVIEW (National Instruments) software. An LCD projector was used to display questionnaires on a large screen in front of the participant (approximately 3 meters). Physiological signals and experimental timing were monitored by the experimenter from an adjacent control room by a 17” flat panel monitor. Sound attenuating headphones, as well as a video camera, allowed the experimenter to monitor and communicate with the participant. A Grass instruments stimulator (Model S88 or S48; Grass Technologies, West Warwick, RI), stimulus isolation unit (Model SIU8; Grass Technologies), constant current unit (Model CCU1; Grass Technologies), and bipolar stimulating electrode (019-401400; Nicolet Instruments Inc, Madison, WI) were used to deliver noxious electrocutaneous stimuli to the left ankle over the retromalleolar pathway of the sural nerve. Biceps femoris electromyogram (EMG) recording electrodes for measuring NFR were Ag-AgCl. The computer controlled the onset/offset of the stimulator, and the stimulus intensity (max current = 40 mA) was varied by a computer-controlled voltage regulator. A Grass Instruments Model 15LT Bipolar Amplifier with Quad AC (15A54) module collected/filtered biceps femoris EMG, which was sampled at 1000 Hz.

Pain-Related Outcomes

NFR Threshold

The NFR is a spinally-mediated protective reflex, elicited by activation of A-delta fibers, that can be quantified using electromyography (EMG). Because supraspinal regions are not necessary for its elicitation, the NFR is often used to assess spinal nociceptive processes. A bipolar
surface-stimulating electrode was affixed to the left ankle over the retromalleolar pathway of the sural nerve to elicit the NFR. Biceps femoris electromyogram (EMG) was recorded by attaching 2 active Ag-AgCl electrodes over the left biceps femoris muscle 10 cm superior to the popliteal fossa. The raw biceps femoris signal was amplified 20,000× and frequencies below 10 Hz and above 300 Hz were filtered. A common reference electrode was attached over the lateral epicondyle of the left femur. To facilitate relaxation of the leg muscles, participants were seated comfortably in a recliner, with a small pillow situated under the left ankle. The procedures for assessing NFR threshold were adapted from France et al.\textsuperscript{18} and used in previous studies.\textsuperscript{36,37} Trains of 5 1-ms rectangular wave pulses at 250 Hz (ie, 3 ms ITI) were delivered to the sural nerve with a varying intertrain interval of 8 to 12 seconds to reduce stimulus predictability. The first train started at 0 mA (current) and was increased in steps of 1.5 mA until a NFR was detected. The NFR was defined as a mean biceps femoris EMG response in the 90 to 150 msec poststimulus interval that exceeded mean EMG activity during the 60 msec prestimulus baseline interval by 1.4 SD, because of recent empirical support for this definition.\textsuperscript{38} Using the 90 to 150 msec timeframe reduces potential contamination by the non-nociceptive RII reflex, startle responses, and/or voluntary movements.\textsuperscript{12} The stimulus intensity was then decreased in .75 mA steps until a NFR was no longer observed. This up-down staircase process was repeated 2 more times, but with the use of .5 mA steps. NFR threshold was defined as the average stimulation intensity (in mA) of the last 2 peaks and troughs of the up-down staircase procedure.

Pain Ratings of Suprathreshold Stimuli

Following every electrocutaneous stimulus, participants rated their pain using a computer-presented numerical rating scale (NRS).\textsuperscript{36} The scale ranged from 0 to 100 with the following labels: 0 (no sensation), 1 (just noticeable), 25 (uncomfortable), 50 (painful), 75 (very painful), and 100 (maximum tolerable). Participants responded by moving an indicator to a position along the line that corresponded to their rating and submitted their answer by computer mouse. To assess the subjective evaluation of NFR threshold level stimuli, the pain ratings from the last 2 peaks and troughs in the NFR threshold assessment were averaged.

Pain Threshold and Tolerance

Immediately following NFR threshold assessment, subjective-pain threshold and tolerance levels were assessed. Electric stimulation of the sural nerve (using the same pulse train parameters) was increased in 1.5 mA steps (variable 8 to 12 seconds ITI) until pain tolerance (intensity corresponding to 100 rating on the NRS) or the 40 mA maximum was achieved. Pain threshold was defined as the first stimulus intensity rated ≥50 on the NRS pain rating scale (see description in previous section), and pain tolerance was defined as the stimulus intensity rated 100 (or 40 mA if the maximum was reached).

Retrospective Ratings of Pain Tolerance Testing

Immediately following the pain threshold/tolerance procedure, participants rated their overall sensory and affective pain experience for the pain-tolerance phase using the McGill Pain Questionnaire-Short Form (MPQ-SF).\textsuperscript{30} The MPQ-SF is reliable and valid, and commonly used in pain research.\textsuperscript{30} Respondents rated 11 sensory (eg, throbbing, shooting) and 4 affective (eg, sickening, fearful) pain descriptors on a scale from 0 (none) to 3 (severe). A sum of all sensory words and affective words were then used to compute sensory and affective pain rating scores.

Other Questionnaires

Demographics Form

Age and participant sex were acquired from this questionnaire used to obtain standard background information and potential exclusionary criteria (cardiovascular, neurological, chronic pain, phobia, recent trauma, and medications).

Pain Catastrophizing

Recent evidence suggests that how pain catastrophizing is measured can influence the degree of association with pain outcomes.\textsuperscript{11,14,16,24,39} Specifically, pain catastrophizing measured after pain testing (situation-specific or “in vivo” catastrophizing) is a better predictor of pain than baseline (traditional) catastrophizing. In these previous studies, situation-specific catastrophizing was measured by altering the instructions of a traditional measure of catastrophizing such that the participant is asked to reflect on thoughts during pain testing. This has been accomplished with the Catastrophizing subscale of the Coping Strategies Questionnaire (CSQ).\textsuperscript{24} but more often with the Pain Catastrophizing Scale (PCS).\textsuperscript{7,16,24,29} For the present study, traditional and situation-specific pain catastrophizing were assessed with the PCS.

The PCS is a reliable and valid 13-item scale that assesses catastrophic thinking in response to pain.\textsuperscript{36} Responses are on a 5-point scale that ranges from “not at all” to “all of the time.” For the present study, all items were summed to achieve a PCS total score. The PCS was administered prior to pain testing using the traditional instructions (“We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain.”). In addition, the PCS was administered after pain testing with altered instructions to assess situation-specific catastrophizing (“Thinking back to your experience during the electric stimulations, please indicate the degree to which you had these thoughts and feelings.”). In the present study, the internal consistency was excellent for both administrations (traditional: $\alpha = .91$; situation-specific: $\alpha = .93$).
Center for Epidemiological Studies-Depression Scale (CES-D)

The CES-D is a reliable and valid 20-item questionnaire in which participants respond on a scale that ranges from 0 (rarely or none of the time) to 3 (most or all of the time) with instructions to rate their symptoms during the past week.35 Items are summed to achieve a total score that can range from 0 to 60, with higher scores indicating greater symptomatology. Scores ≥ 16 indicate clinically significant symptoms. The CES-D was used to control for depressive symptoms in analyses, due to concern that pain catastrophizing may index depression.44

Procedure

A brief health status interview was conducted by phone prior to study participation to assess for exclusion criteria. At the testing session, participants were provided an overview of the experiment and informed consent was obtained. A demographics form was then administered to attain background information and thoroughly assess inclusion criteria. Participants were then instructed on the use of the numerical rating scale (NRS) for rating pain following each electrical stimulation. After participants were familiarized with the NRS, skin was degreased with alcohol and prepared with Nu-Prep gel to attain impedances below 5 KΩ. Next, physiological sensors were attached with self-adhering collars, and then participants completed the CES-D and the pretest PCS.

The initial phase of testing involved affective modulation of the startle reflex that involved the presentation of emotionally-charged pictures during which noise bursts were delivered. In brief, 54 pleasant, neutral, and unpleasant pictures (6-second picture duration, 12- to 22-second interpicture intervals) were presented in random order, during which 50-ms, 105-dB noise bursts were delivered during 50% of pictures. The emotion elicited by these procedures is short-lived and should not influence subsequent testing.6 Nonetheless, following startle modulation testing, a short break was provided and physiological signal integrity was checked before NFR threshold testing began. NFR threshold was assessed by sending electric pulses to the sural nerve of the left ankle to determine the level of stimulus intensity (NFR threshold) that would reliably elicit the reflex. Throughout this phase, the NRS was presented by LCD projector on a large screen mounted in front of the participant. A digital light positioned next to the scale was illuminated when the participant was to make a pain rating. Following assessment of NFR threshold, the participant’s subjective pain threshold and tolerance were measured (as described in previous sections). Immediately after the pain threshold and tolerance procedure, participants completed the MPQ-SF to assess their sensory and affective pain experience during the tolerance phase, as well as the PCS to evaluate situation-specific catastrophizing during pain testing. After another short break, these procedures were followed by a 2-hour phase unrelated to the current study that included more emotional picture viewing during which noxious shocks were delivered. At study completion, participants were debriefed and thanked for their participation. In exchange for their participation, participants received monetary compensation. All procedures were fully approved by the University of Tulsa ethics review board.

Data analysis

Before testing the competing hypotheses (moderator vs independent influences), zero-order relationships among study variables were tested using Pearson’s r correlation coefficients. Hypotheses regarding the moderating effect of pretest and situation-specific catastrophizing were tested using hierarchical regression analyses. Consistent with the recommendations of Cohen et al9, main effect variables were centered before creating interaction terms. Participant sex,11,17,47 age,13 and depressive symptoms1,44-46 were controlled for by entering them in step 1 (although all conclusions were the same without their inclusion). In step 2, the centered main effects of NFR threshold, pretest catastrophizing, and situation-specific catastrophizing were entered. And finally, the NFR Threshold × Pretest Catastrophizing and the NFR Threshold × Situation-Specific Catastrophizing interactions were entered in step 3. All regression assumptions, including multicollinearity, were met.

Hypotheses

Preliminary analyses: It was predicted that NFR threshold would be significantly related to pain threshold and pain tolerance.

Study aim 1: It was predicted that traditional (pretest) and situation-specific pain catastrophizing would not be significantly related to NFR threshold, but would significantly correlate with measures of pain perception (pain threshold, pain tolerance, ratings of suprathreshold stimuli, sensory pain ratings, affective pain ratings).

Study aim 2: It was predicted that pain catastrophizing would moderate the relationship between NFR threshold and pain sensitivity (pain threshold and pain tolerance). Given that situation-specific catastrophizing has been shown to be a better predictor of pain outcomes, it was predicted that situation-specific catastrophizing would moderate the NFR-pain sensitivity relationship to a greater degree than traditionally-measured pain catastrophizing.

Results

Table 1 presents descriptives and zero-order correlations for study variables, whereas Table 2 presents results from hierarchical regression analyses.

Preliminary Analyses: The Relationship Between NFR Threshold and Pain Sensitivity

Consistent with our hypothesis, zero-order correlations indicated that NFR threshold was strongly correlated with measures of pain sensitivity (pain threshold, pain tolerance). Additionally, NFR threshold was weakly correlated with ratings of suprathreshold stimuli, but
uncorrelated with retrospective ratings of sensory and affective pain.

The Relationship Between Pain Catastrophizing, NFR Threshold, and Pain Perception

Consistent with our hypothesis, zero-order correlations suggested pretest (traditional) and situation-specific pain catastrophizing were both unrelated to NFR threshold. Furthermore, situation-specific catastrophizing was correlated with pain threshold, ratings of the suprathreshold stimuli, sensory pain ratings, and affective pain ratings. However, despite a moderate-to-strong correlation between pretest catastrophizing and situation-specific catastrophizing, pretest catastrophizing was not significantly correlated with any pain outcome. To statistically compare whether situation-specific catastrophizing was a stronger predictor than pretest catastrophizing (comparing beta weights). However, neither variable was a significant predictor of pain tolerance.

Does Pain Catastrophizing Moderate the Relationships Between NFR Threshold and Measures of Pain Sensitivity?

Contrary to prediction, hierarchical regression analyses failed to show a significant interaction between pain catastrophizing and NFR threshold when predicting pain threshold or pain tolerance (Table 2). In the model predicting pain threshold, squared semi-partial correlations suggested the NFR x Pretest Catastrophizing interaction uniquely explained less than .1% of the variance and the NFR x Situation-Specific Catastrophizing interaction uniquely explained .4% of the variance. NFR threshold explained 36.1% of the unique variance, whereas situation-specific catastrophizing, although a significant predictor, explained only 4.2% of the unique variance. No other predictors were significant.

In the model predicting pain tolerance, squared semi-partial correlations suggested the NFR x Pretest Catastrophizing interaction explained .7% of the variance and the NFR x Situation-Specific Catastrophizing interaction explained .2% of the variance. NFR threshold explained 39.1% of the unique variance. No other predictors were significant, including pretest

Table 2. Hierarchical Regression Models Examining the Relationship Between Pain Catastrophizing, Spinal Nociceptive Processes (NFR), and Pain Sensitivity (Pain Threshold, Pain Tolerance)

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<th>$\beta$ (β)</th>
<th>Squared semi-partial correlation ($sr^2$)</th>
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Abbreviations: Situation-spec, Situation-specific; Catas, catastrophizing.

NOTE. Bolded statistics are significant at $P < .05$. All beta weights and semipartial correlations are from final regression models with all variables entered.
and situation-specific catastrophizing, both of which explained ≤ 1% of the unique variance in pain tolerance.

For exploratory purposes, we also tested whether either measure of catastrophizing moderated the relationship between NFR threshold and subjective pain ratings. These interactions were not significant for suprathreshold ratings (Ps > .05, variance explained < 3%), MPQ-SF sensory ratings (Ps > .05, variance explained < 2%), or MPQ-SF affective ratings (Ps > .05, variance explained < .2%).

**Post-Hoc Analysis: Sex, Catastrophizing, and Pain Threshold**

Table 1 indicates that there was a significant relationship between participant sex and pain threshold; however, in the hierarchical regression analysis predicting pain threshold, sex did not emerge as a significant predictor. Although previous studies have noted that catastrophizing can mediate the relationship between sex and pain, to our knowledge, no one has examined whether situation-specific catastrophizing mediates this relationship. Following the guidelines of Baron and Kenny, Table 1 shows that sex is correlated with pain threshold (P < .05); however, the correlation with situation-specific catastrophizing just missed significance (P = .065). For the purposes of exploration, mediation was tested nonetheless. When situation-specific catastrophizing and sex were entered as predictors in a multiple regression model predicting pain threshold, situation-specific catastrophizing (β = −.221, P = .026), but not sex (β = −.159, P = .107), was a significant predictor. This suggests situation-specific catastrophizing is at least a partial mediator of the sex and pain relationship. Traditional catastrophizing is not likely to have mediated the sex and pain relationship in our study, because pretest catastrophizing was uncorrelated with sex.

The lack of sex differences in NFR threshold, pain tolerance, suprathreshold ratings, and sensory ratings could be due to changes in hormonal status of women due to age (pre- vs post-menopausal). To determine whether menopausal status influenced the relationship of sex with pain outcomes, age was dichotomized (≤ 45 years vs > 45 years) and entered into separate ANOVAs along with sex to analyze each pain outcome. The interaction of age x sex was nonsignificant in all models (Ps > .05), suggesting menopausal status did not contribute to the lack of sex differences.

**Discussion**

**The Relationship Between NFR Threshold and Pain Perception**

Consistent with previous research, the current study suggests NFR threshold (a measure of spinal nociceptive processes) was associated with pain sensitivity (pain threshold, pain tolerance), and, to a lesser extent, the subjective ratings of suprathreshold stimuli. Zero-order correlations between NFR threshold and electrocutaneous pain threshold and tolerance were strong, whereas the correlation with ratings of the suprathreshold stimuli was weak to moderate. NFR threshold was not significantly correlated with retrospective ratings of sensory and affective pain elicited by the tolerance procedure. Hierarchical regression analyses that controlled for age, gender, and pain catastrophizing (traditional and situation specific) also suggested NFR threshold was significantly related to measures of pain sensitivity.

It is important to note that NFR and subjective pain can diverge under some circumstances (eg, pharmacological intervention, hypnosis). But, within the current paradigm, NFR threshold may serve as a physiological marker of individual differences in pain sensitivity. However, it would appear that additional mechanisms (eg, memory, cortico-cortical mechanisms, report bias) may contribute to the subjective report of suprathreshold stimuli and retrospective evaluation of the tolerance procedure, because the relationships between NFR threshold and these outcomes were weaker.

**Pain Catastrophizing, Spinal Nociceptive Processes, and Pain Perception**

Our findings are consistent with research suggesting situation-specific catastrophizing provides important additional information regarding the negative cognitions that influence pain-related processes. The internal consistency was high for traditional and situation-specific measures of catastrophizing (α > .90), but the shared variance was low (r² = .21). Zero-order correlations with pain outcomes implied situation-specific catastrophizing was significantly related to pain threshold and ratings of suprathreshold stimuli, but magnitudes of the correlations were even larger for relationships with retrospective sensory and affective pain ratings of the tolerance procedure. By contrast, traditional catastrophizing was not significantly correlated with any pain outcome. This suggests there is additional predictive validity for the situation-specific measure, a conclusion supported by hierarchical regression analyses that entered traditional catastrophizing and situation-specific catastrophizing in the same models to predict pain sensitivity. In those models, situation-specific catastrophizing emerged as a better predictor (ie, Beta magnitudes), although neither measure significantly predicted pain tolerance. Together, these data suggest traditionally measured catastrophizing and situation-specific catastrophizing could be distinct constructs, or at least distinct components of the catastrophizing construct. However, future research is needed to address this question.

Neither measure of catastrophizing was associated with NFR threshold, with zero-order correlations being r = 0 (situation-specific) and .07 (traditional). This represents the fourth independent study to conclude that pain catastrophizing is unrelated to NFR threshold, and the first study to clearly demonstrate that situation-specific cognitions are also unrelated. Moreover, the effect sizes for the relationship between catastrophizing and NFR threshold are consistent with prior studies (present study = .5 and 0%; prior studies = .39 and .4%). Therefore, the consistency of this finding provides
confidence in the conclusion that the effect size is a good estimate of the population effect size, and that a lack of power does not explain our null finding. Further, our observation of no association between catastrophizing (both traditional and situation-specific) and NFR threshold provides added support for the notion that catastrophizing does not influence descending modulation of spinal nociceptive signals.

Previous studies have noted that catastrophizing can mediate the relationship between participant sex and pain.25 Although not a goal of the current study, our results suggest that situation-specific catastrophizing may do a better job of mediating the sex-pain relationship than traditional catastrophizing measures. The zero-order correlation for sex and pain threshold was significant; however, the Beta weight for sex was nonsignificant in the regression model predicting pain threshold when situation-specific catastrophizing was also included as a predictor.

Pain Catastrophizing and the Relationships Between NFR and Pain Sensitivity

Contrary to our prediction, the results of the hierarchical regression analyses suggest that pain catastrophizing, regardless of whether it was traditionally measured or situation-specific, did not moderate the relationships between NFR threshold and pain sensitivity. The squared semipartial correlations from those models indicated that the interaction terms explained little variance in the pain dependent variables ($\hat{R}^2 < .8\%$). Therefore, assuming these effect sizes are reasonable estimates of the population values, the null findings are not likely to be related to low power.

As noted above, the lack of an association between either traditional or situation-specific catastrophizing and NFR threshold suggests that catastrophizing does not influence descending modulation of spinal nociceptive signals. Similarly, the absence of a moderating effect of catastrophizing on the relationship between NFR and pain thresholds suggests that catastrophizing (both traditional and situation specific) does not modulate the effect of nociceptive inputs on pain reports. Rather, both nociceptive inputs and catastrophizing appear to exert an independent influence on individual differences in pain sensitivity. These conclusions are supported by the fact that 1) NFR threshold was significantly related to pain threshold and pain tolerance in the zero-order correlations, but also in the regression analyses that controlled for pain catastrophizing, age, depression, and participant sex, and 2) situation-specific pain catastrophizing was a significant predictor of pain threshold even after controlling for depressive symptoms, age, sex, and NFR threshold. Interestingly, the effect of situation-specific pain catastrophizing was larger on retrospective reports of pain evoked during the tolerance procedure (MPQ-SF sensation and affect ratings) rather than on measures of pain sensitivity. This finding may imply that situation-specific pain catastrophizing influences memory for pain more so than altering pain sensitivity.28 Additionally, participants may have based their situation-specific pain catastrophizing ratings on their MPQ-SF subjective ratings given the close temporal proximity of the ratings. This would explain why the correlations between situation-specific catastrophizing and MPQ-SF ratings were higher than ratings of the supra-threshold stimuli during NFR testing, which were spaced farther in time. Future studies could address this issue by counterbalancing the order of catastrophizing and MPQ-SF ratings.

The current study had a number of strengths including a relatively large sample size, use of well-controlled laboratory conditions to test pain and nociceptive reactivity, and the inclusion of traditional and situation-specific pain catastrophizing measures. Moreover, our sample was diverse with a wide range of individual differences in pain catastrophizing, depressive symptoms, age, and pain outcomes (see Table 1). Such diversity suggests our nonsignificant findings are not due to range restriction in our measured variables. These methodological strengths provide reasonable confidence in our conclusions. Nevertheless, it is important that these results be replicated in other samples. In particular, it is important to determine whether similar findings can be observed in patients with chronic pain. Evidence suggests that central pain processing can become dysregulated in persons with persistent pain,31,48,53 and such changes could alter the manner in which catastrophizing influences nociceptive processing. For example, sensitization of nociceptive systems may provide a means by which catastrophizing can alter the relationship between NFR threshold and pain sensitivity in chronic pain.

Another potential limitation stems from the timing of pain-sensitivity testing. NFR threshold, pain threshold, and pain tolerance were assessed after participants viewed emotionally charged pictures (during startle modulation testing). These procedures could have altered individual responses to pain testing and the correlations between pain catastrophizing and pain outcomes. However, there are reasons to believe that startle testing did not negatively influence our conclusions. First, pictures were randomized between subjects; thus, the probability of starting or ending on a certain emotional valence (pleasant, neutral, unpleasant) was equal. Therefore, any primacy or recency effects should be washed out. Second, any general bias from viewing emotionally charged pictures should be washed out, because there were equal numbers of pleasant, neutral, and unpleasant pictures presented. And third, each picture was presented only briefly (6 seconds), and research has shown there is little carryover of emotion after picture offset when this paradigm is used.6 Additionally, our lack of counterbalancing and use of a method-of-limits procedure to assess pain threshold/tolerance may have influenced our pain sensitivity measurement. With regard to order of NFR and pain threshold/tolerance assessment, we chose not to counterbalance the order of presentations because our experience suggests that some participants inflate subjective ratings when initially exposed to electric stimuli. Thus, it is advantageous to assess NFR threshold first, because the validity of NFR threshold does not depend on subjective reports. Also, the alternative strategy of testing
tolerance first would expose participants to very intense stimulation before NFR testing, which could increase drop-out and, perhaps more importantly, influence NFR threshold. With regard to the method-of-limits procedure used to determine pain threshold and tolerance, we acknowledge that such an approach is not optimal and encourage future researchers to consider employing a multiple random-staircase method to enhance overall accuracy of the assessment.

Altogether, results suggest situation-specific catastrophizing was a better predictor of pain than traditionally measured catastrophizing, with the strongest effects being on measures of retrospective evaluation of pain testing. However, neither situation-specific nor traditionally measured pain catastrophizing enhance pain by engaging descending controls. Rather, pain catastrophizing may lead to hyperalgesia via processes independent of spinal nociception, perhaps related to the subjective evaluation of pain (eg, memory, report bias, attention).

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