Does In Vivo Catastrophizing Engage Descending Modulation of Spinal Nociception?

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Abstract: Prior research has found that pain catastrophizing measured before pain testing is not correlated with the nociceptive flexion reflex (NFR) threshold (a measure of spinal nociception), suggesting that catastrophizing does not alter pain through descending modulatory mechanisms. However, recent evidence suggests that in vivo catastrophizing (measured during or after pain testing) is a better predictor of pain outcomes. In the present study, NFR threshold and pain sensation ratings were assessed in 78 healthy participants by delivering electric stimulations to the sural nerve. After pain testing, participants were asked to rate their affective reaction (displeasure, arousal) to electric stimuli and to report on their pain catastrophizing. Hierarchical regression analyses controlling for participant sex, pre-experiment affect, depressive symptoms, and self-efficacy were used to predict pain-related outcomes (NFR threshold, pain sensation, displeasure ratings, arousal ratings) from in vivo catastrophizing scores. Results indicated that in vivo catastrophizing was related to pain sensation but not to NFR thresholds or arousal reactions. The relation between in vivo catastrophizing and displeasure ratings was not significant after other variables were controlled. These data support prior research suggesting that catastrophizing does not alter pain by engaging descending modulatory mechanisms.

Perspective: Pain catastrophizing is an important psychological predictor of pain and pain-related functioning. The present study confirms prior reports suggesting that catastrophizing does not work by engaging mechanisms that alter pain transmission in the spinal cord before the signal travels to the brain.

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Key words: Coping, nociceptive flexion reflex, pain perception, shock, nociception, descending pain modulation.

Pain catastrophizing has emerged as one of the strongest predictors of pain-related outcomes, showing positive associations with pain report, pain behaviors, analgesic use, length of hospital stay, length of rehabilitation, pain-related disability, and supraspinal responses to noxious stimulation. In addition, interventions aimed at reducing catastrophizing have been found to improve pain. Accordingly, it is believed that catastrophizing enhances pain perception.

Several psychosocial models (for example, coping, appraisal, attentional, schema-activation, communal/interpersonal) have been used to conceptualize the catastrophizing-pain relationship; yet, little is known about the mediating physiological mechanisms. One potential mechanism is supraspinal initiation of descending central nervous system (CNS) circuitry that can inhibit or facilitate ascending nociception within the spinal cord. Catastrophizing could activate this circuitry, leading to increased nociceptive facilitation, decreased nociceptive inhibition (disinhibition), or both. Moreover, repeated or tonic activation of such mechanisms could result in chronic sensitization of central nociceptive pathways, lowered pain thresholds, and hyperalgesia. Indeed, such processes could provide a mechanism through which catastrophizing promotes the development of chronic pain. If catastrophizing exerts its influence at the spinal level, then markers of spinal nociception should be enhanced. Alternatively, cata-
Coping, especially in persons without chronic pain (for example, subjective pain, pain threshold, pain tolerance) and self-efficacy was controlled because of potential confounds between catastrophizing and negative affectivity. Depressive symptoms and pre-experiment affect were controlled because of potential confounds between catastrophizing and negative affectivity, and self-efficacy was controlled to account for efficacy beliefs. Given that in vivo catastrophizing may better assess pain-related catastrophic thoughts, it was predicted that in vivo catastrophizing would be positively correlated with subjective ratings of noxious electric stimulation (pain sensation, displeasure, arousal) and negatively correlated with NFR threshold, over and above control variables.

Materials and Methods

Data for the present study were collected during 2 recent experiments that examined the influence of emotion on the NFR. Only those methods directly relevant to the present study will be described.

Participants

Participants were 90 healthy male (28%) and female students who received course credit for their participation. Most were white non-Hispanic (68%), single (87%), and employed (59%), with an average age of 22.06 (SD = 6.22). Participants were excluded for age <18 years, cardiovascular, neurological, and/or circulatory problems, recent use of analgesic, antidepressant, or antihypertensive medication, recent psychological trauma, specific phobia of snakes or spiders (due to pictures of spiders and snakes presented during picture-viewing, see “Procedure”), problems healing, Raynaud disease, or any medical problem exacerbated by stress. Twelve participants reached pain tolerance before NFR threshold was achieved and discontinued participation. Therefore, a final sample of 78 participants is included in the present analyses. All procedures were fully approved by The University of Tulsa Ethics Review Board, and participants gave informed consent before participating.

Apparatus

All data acquisition was computer-controlled, using a PC equipped with dual monitors, A/D board (National Instruments, PCI-6036E, Austin, TX), and LabVIEW software. Computerized versions of questionnaires were presented by using LabVIEW that automatically saved data to spreadsheets. Noxious electrical stimuli were delivered by means of a Grass Instruments stimulator (Model S88, West Warwick, RI), stimulus isolation unit (Model SIU8T), constant-current unit (Model CCU1), and bipolar stimulating electrode (Nicolet, 019-401400, Madison, WI). The onset/offset of the stimulator was controlled by computer, and a computer-controlled voltage regulator varied the current to the participant (maximum current = 40 mA). Psychophysiological signals were sampled at 1000 Hz and collected/filtered with Grass Instruments Model 15LT Bipolar Amplifier with Quad AC (15A54) and Dual DC (15A12) modules. Experimenters monitored participants by video from an adjacent room.

nociceptive flexion reflex (NFR), have failed to establish a link between catastrophizing and enhanced spinal nociception. The NFR is a spinal reflex resulting from nociceptor activation, and the level of stimulation that elicits the NFR (NFR threshold) is used as a measure of spinal nociceptive sensitivity. Importantly, enhanced NFR is associated with enhanced subjective pain and chronic pain. Thus, if catastrophizing activates descending modulation to facilitate spinal nociception, then greater pain catastrophizing should be associated with lower NFR threshold (enhanced sensitivity). In France and colleagues’ studies, however, catastrophizing did not correlate with NFR threshold despite significant correlations with supraspinally mediated outcomes (for example, subjective pain, pain threshold, pain tolerance). Therefore, pain catastrophizing does not appear to activate processes that alter spinal nociception.

Recent research suggests that the context in which catastrophizing is measured may influence the catastrophizing-pain relation. Most studies, including those mentioned above, assessed catastrophizing by asking participants to report on their catastrophic thoughts before pain testing. However, catastrophizing assessed before pain testing does not predict catastrophizing measured during or after a painful situation (that is, in vivo catastrophizing). Moreover, in vivo catastrophizing correlates more strongly with experimental pain outcomes. Thus, assessment of catastrophizing during or after participants have experienced a common painful event may provide greater accuracy in the report of catastrophic emotions/cognitions associated with pain coping, especially in persons without chronic pain.
or facilitate nociception before it is registered in conscious-
ness as pain.\textsuperscript{11,32,38} To elicit the NFR, a bipolar surface stimulating elec-
trode was attached to the left ankle over the retromal-
leolar pathway of the sural nerve. Two active Ag-AgCl
electrodes were placed over the left biceps femoris mus-
cle 10 cm superior to the popliteal fossa to record biceps
femoris electromyogram (EMG) associated with the NFR.
A common ground electrode was placed over the left
lateral epicondyle of the femur. To facilitate relaxation
of the leg muscles during the experiment, participants
were seated in a recliner with a small pillow positioned
under their ankle. For this procedure, reflex detection
was defined as a mean biceps femoris EMG response in the
90- to 150-ms post-stimulus interval that exceeded
mean EMG activity during the 60-ms pre-stimulus base-
line interval by at least 1.0 standard deviation. Using the
90- to 150-ms time frame avoids potential contamination
by the non-nociceptive RII reflex, startle responses, and/or voluntary movements.\textsuperscript{7} The stimulus intensity
necessary to reliably elicit the NFR (that is, NFR threshold)
was assessed according to procedures adapted from
France and colleagues.\textsuperscript{14} Electric stimulation of the sural
nerve was repeated according to a variable interval of 8
to 12 seconds to prevent habituation and predictability.
For each stimulation, 5 rectangular wave pulses of 1-ms
duration and 3-ms interstimulus interval were delivered.
The trial began with 0 mA (current) and increased in 1.5
mA steps until an NFR was detected. Stimulus intensity
was then decreased in 0.75-mA steps until a reflex was no
longer detected. This up-down staircase procedure was
repeated 2 more times, but with 0.5-mA steps. The aver-
age stimulus intensity (in mA) of the last 2 peaks and
troughs was used to define NFR threshold. Participants
for which a reflex could not be reliably obtained were
given the choice of repeating the threshold assessment
procedure or discontinuing the experiment. The raw bi-
cepes femoris EMG signal was amplified by 20,000, and
frequencies below 10 Hz and above 300 Hz were filtered.
The signal was rectified and reduced online after each
stimulation.

Self-Report Measures

Pain Sensation Rating Scale

A previously validated computer-presented numerical
rating scale was used to rate each electric stimulation.\textsuperscript{14}
Starting from the bottom, the scale was labeled 0 (no
sensation), 1 (just noticeable), 25 (uncomfortable), 50
(pain), 75 (very painful), and 100 (maximum tolerable).
To respond, participants dragged an indicator along the
scale and submitted their answers by computer mouse.
Average ratings from the last 2 stimuli during the NFR
threshold assessment were used as a measure of pain
sensation.

In Vivo Catastrophizing

The 6-item subscale of the Coping Strategies Question-
naire (CSQ) was used to assess in vivo catastrophizing
after all exposure to noxious electric stimulations. This
subscales measures negative/catastrophic thoughts and
ideations about pain and has been shown to have good
internal and test-retest reliabilities.\textsuperscript{46}

Affect Ratings

Pre-experiment affect and affective responses to elec-
tric stimulations were rated with the use of a computer-
ized version of the Self-Assessment Manikin (SAM).\textsuperscript{29} The
SAM consists of 2 sets of 5 pictographs depicting affective valence (unpleasant-pleasant) and arousal (calm-ex-
cited). The SAM has been shown to have good validity,
correlations between SAM valence and arousal rat-
ings and the multi-item Semantic Differential scale being
r = 0.96 and r = 0.95, respectively.\textsuperscript{29} Further, SAM ratings
have been shown to be reliable, with coefficients for
valence and arousal being r = 0.94 and r = 0.94, respect-
ively.\textsuperscript{28} Participants dragged an indicator on or between
the 5 pictographs for each scale and submitted their an-
swers by computer mouse. Ratings ranged from 1 to 9 for
each dimension. The valence dimension was reverse-
scored so that higher scores reflected greater negative affect (displeasure). To rate pre-experiment affect, in-
suctions were: “Please rate how you feel right now . . . ”. To rate responses to electric stimuli, instructions
were: “Please rate how the electrical stimulations made
you feel . . . ”.

Center for Epidemiological Studies-
Depression Scale

The Center for Epidemiological Studies-Depression Scale
(CES-D)\textsuperscript{40} is a 20-item self-report measure of de-
pressive symptomatology. Items are rated on a 4-point
Likert scale indicating the degree to which the respon-
dent has experienced each symptom over the past 2
weeks. The CES-D has been shown to have good internal
consistency and test-retest reliability and adequate dis-
criminative and convergent validity.\textsuperscript{40}

Self-Efficacy for Pain Reduction

The 5-item Self-Efficacy for Pain Reduction (SEPR) was
used to measure participants’ beliefs that they could
make reductions in different levels of pain (mild, discom-
forting, distressing, horrible, excruciating) without tak-
ing medications.\textsuperscript{41} Ratings were made on 11-point Likert
scales ranging from 0 (uncertain) to 10 (certain). Items
were summed to achieve a score ranging from 0 to 50,
with higher scores indicating greater self-efficacy. Re-
search has suggested a 1-day test-retest reliability rang-
ing from 0.45 to 0.70, and correlation with cold pressor
pain tolerance was r = 0.48, suggesting adequate valid-
ity (Rhudy JL, Dubbert PM, Parker JD, Burke RS, Williams
AE. Affective modulation of pain in substance depen-
dent veterans, Pain Med [in press]). Internal consistency
in the present study was α = 0.81.

Procedure

Participants were provided an overview of the experi-
ment, informed consent was obtained, and all electrodes
were then applied. Before pain testing, participants
filled out demographics, depressive symptoms, pre-experiment affect, and self-efficacy questionnaires. There were 2 phases to the original experiments: NFR threshold assessment and emotional picture-viewing. Phase 1 involved sending electric stimuli to the ankle to determine the stimulation intensity that reliably elicited the NFR (NFR threshold). During this phase, participants viewed the computerized pain rating scale positioned next to a digital light that illuminated when they were to make a rating (after every electric stimulation). Phase 2 involved viewing 24 emotionally charged pictures, during which electric stimuli were randomly delivered. During this phase, 8 pleasant (couples in erotic poses), 8 neutral (household objects, mushrooms), and 8 unpleasant (human and animal attack scenes) pictures were each presented for 6 seconds (with varying 12- to 22-second interpicture interval). Presentation order was randomized across participants with the limitation that not more than 2 pictures of similar valence were shown consecutively. This picture-viewing paradigm was used to examine the influence of emotional valence (pleasant, neutral, unpleasant) on NFR magnitude and pain ratings resulting from noxious electric stimuli delivered during pictures. Data from phase 2 are reported elsewhere and will not be discussed further.43,44 Once phase 2 was concluded, the participants completed the pain catastrophizing measure, rated their affective reactions to electric stimulations (displeasure and arousal), were debriefed, and were thanked for their participation. It was decided to measure pain catastrophizing after phase 2 to keep from influencing coping responses (through priming, suggestion, and so forth) during phase 2 pain testing. Thus, these ratings were taken 15 to 20 minutes after the NFR threshold testing procedure.

**Data Analysis**

To examine the relations between in vivo catastrophizing and nociceptive reactions, 4 hierarchical regression analyses were conducted that predicted NFR threshold, pain sensation ratings, displeasure ratings, and arousal ratings. Analyses controlled for participant sex because of the well-established sex differences in pain,6,8,12,45 whereas depressive symptoms, self-efficacy, and pre-experimental affect were controlled to ensure that these factors did not mediate the relation between catastrophizing and pain outcomes.52 Therefore, participant sex, depressive symptoms (CES-D), self-efficacy (SEPR), and pre-experiment affect (SAM displeasure and arousal ratings) were entered into step 1, and in vivo catastrophizing scores were entered into step 2. In the 3 models predicting subjective reactions to electric stimuli (pain, displeasure, and arousal ratings), NFR threshold (stimulus intensity level) was also entered into step 1 to control for individual differences in stimulus level. All assumptions for regression were met.

**Results**

Preliminary analyses indicated that participants who discontinued the experiment were not significantly different from completers on self-efficacy ratings, depressive symptoms, or pre-experiment affect ratings (P > .05).

Table 1 provides descriptive statistics and zero-order correlations for all variables in this study. Table 2 depicts results of all 4 regression models. The regression model predicting NFR threshold was not significant [F(6, 71) = 0.372, P = .894], nor was the zero-order correlation or the β-weight for in vivo catastrophizing. Moreover, no other predictor was significantly related to NFR threshold in this sample. This suggests that in vivo catastrophizing is not related to spinal nociception.

The regression model predicting pain sensation ratings was significant [F(7, 70) = 2.83, P = .01], and in vivo catastrophizing predicted pain sensation ratings over and above control variables (R² change = 0.05, F change = 4.46, P = .038). This suggests that in vivo catastrophizing was related to the subjective evaluation of noxious electric stimuli. Stimulus level (NFR threshold) and self-efficacy were also significant predictors in the model, predicting 8% and 7% unique variance in pain sensation ratings, respectively. This suggests that persons with higher NFR thresholds also tended to have higher ratings of the threshold-level stimulus and that increased self-efficacy was associated with lower pain ratings.

The regression model predicting affective displeasure reactions to electric stimulations was significant [F(7, 70) = 2.49, P = .024]. Although the zero-order correlation between catastrophizing and displeasure ratings was significant, after controlling for other variables in the regression model, in vivo catastrophizing was not significant (R² change = 0.04, F change = 3.74, P = .057). Stimulus level (NFR threshold) was a significant predictor in the model (P = .018), explaining 7% unique variance in ratings of displeasure. Again, persons with higher NFR thresholds also tended to have higher ratings of the threshold-level stimulus. Self-efficacy approached significance as a unique predictor (P = .072) and explained 4% of the variance in displeasure ratings.

The regression model predicting affective arousal reactions to electric stimulations was not significant [F(7, 70) = 1.16, P = .335], nor was the zero-order correlation or the β-weight for in vivo catastrophizing.

**Discussion**

The present study was the first, to our knowledge, to examine the relation between in vivo catastrophizing (measured after pain testing) and NFR threshold, pain sensation ratings, and affective pain reactions (displeasure, arousal). Results suggested that in vivo catastrophizing was related to subjective pain but not to NFR threshold or subjective arousal reactions. The zero-order correlation between in vivo catastrophizing and affective displeasure was positive and significant, but the relation was not significant when other variables were controlled by multiple regression. We did not specifically test for mediation in our models; however, we did control for pre-experiment affect, depressive symptoms, and self-efficacy and still found a significant relation be-
between in vivo catastrophizing and pain sensation ratings. Thus, these data are consistent with other research that suggests the effect of catastrophizing on pain is not due solely to the mediating effects of depressive symptoms or negative affect\textsuperscript{19,23,53} and extends this work to suggest that self-efficacy does not fully explain the relation. Although the majority of studies examining the influence of pain catastrophizing have been in clinical populations, experimental studies have noted a positive association between catastrophizing and reactions to noxious stimuli including pain threshold, pain tolerance, temporal summation, and subjective reports.\textsuperscript{13-15,17,20} In an attempt to elucidate the mechanisms mediating this relation, the present study used the NFR—a measure of spinal nociception. Prior studies have found that the NFR and subjective pain are modulated in parallel by psychological factors such as emotion,\textsuperscript{43,44} stress,\textsuperscript{62} attention,\textsuperscript{61} and hypnotic suggestion.\textsuperscript{27} Ostensibly, psychological (supraspinal) factors influence the NFR through circuitry that includes the periaqueductal gray (PAG), rostroventral medulla (RVM), dorsolateral pons/tegmentum (DLPT), and descending projections through the dorsolateral funiculus—a modulatory circuit with numerous connections to brain centers involved with cognition and emotion.\textsuperscript{11,42,57} It is assumed that this spinal gating mechanism alters the ascending flow of nociception before registration at supraspinal centers associated with pain perception. At least part of the variability in subjective pain would then reflect alterations in spinal nociception (as indexed by the NFR), hence accounting for the covariation between these measures. We reasoned that if supraspinal processes associated with catastrophic thinking modulate subjective pain by activating descending circuitry, the NFR would covary with catastrophizing (that is, greater catastrophizing equals enhanced NFR). This should be true whether pain catastrophizing reflects a trait-like disposition that results in tonic activation of descending processes, or whether it results in state-like activation during, or in anticipation of, pain.\textsuperscript{52} However, our study corroborates prior work suggesting that descending modulation does not mediate the catastrophizing-pain relation because the NFR did not covary with in vivo pain catastrophizing.\textsuperscript{14,15}

As France and colleagues\textsuperscript{14} point out, though, “a failure to reject the null hypothesis can never establish that such a relationship does not exist. . .” (p. 463). Nevertheless, this is the third empirical study suggesting that catastrophizing does not significantly correlate with NFR threshold. Moreover, the effect size for the relation is consistently small across studies regardless of the method of catastrophizing assessment. In the present study, in vivo catastrophizing explained 0.3% of the variance in NFR thresholds, an effect size similar to that found in a sample of osteoarthritis patients when catastrophizing was measured before pain testing (0.4%).\textsuperscript{15} Accordingly, our findings are not likely to stem exclusively from low power or characteristics specific to our sample.

Together, these data suggest that catastrophizing modulates pain by mechanisms other than descending controls that alter spinal nociception. It is possible that

### Table 1. Descriptive Statistics for Variables in Regression Models

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>MEAN</th>
<th>SD</th>
<th>OBSERVED RANGE</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. In vivo catastrophizing (0–36)</td>
<td>8.21</td>
<td>5.65</td>
<td>0–25</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>2. NFR threshold (0–40 mA)</td>
<td>7.33</td>
<td>5.25</td>
<td>2–26</td>
<td>0.05</td>
<td>–</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>3. Pain sensation rating (0–100)</td>
<td>50.24</td>
<td>25.08</td>
<td>4–99</td>
<td>0.28*</td>
<td>0.26*</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Displeasure rating of electric stimuli (1–9)</td>
<td>7.38</td>
<td>1.47</td>
<td>4–9</td>
<td>0.30*</td>
<td>0.25*</td>
<td>0.41*</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Arousal rating of electric stimuli (1–9)</td>
<td>6.77</td>
<td>2.03</td>
<td>1–9</td>
<td>0.05</td>
<td>0.21</td>
<td>0.21</td>
<td>0.48*</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Depressive symptoms (0–60)</td>
<td>12.83</td>
<td>8.83</td>
<td>0–45</td>
<td>0.36*</td>
<td>0.03</td>
<td>0.08</td>
<td>0.14</td>
<td>0.01</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Self-efficacy (0–50)</td>
<td>23.90</td>
<td>8.72</td>
<td>3–50</td>
<td>–0.09</td>
<td>0.08</td>
<td>–0.24*</td>
<td>–0.20</td>
<td>0.09</td>
<td>0.04</td>
<td>–</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Pre-experiment displeasure (1–9)</td>
<td>3.90</td>
<td>1.44</td>
<td>1–7</td>
<td>–0.06</td>
<td>0.08</td>
<td>0.01</td>
<td>0.00</td>
<td>–0.04</td>
<td>0.10</td>
<td>–0.26*</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>9. Pre-experiment arousal (1–9)</td>
<td>3.91</td>
<td>2.31</td>
<td>1–9</td>
<td>0.10</td>
<td>–0.06</td>
<td>0.03</td>
<td>0.05</td>
<td>0.20</td>
<td>0.20</td>
<td>0.11</td>
<td>–0.01</td>
<td>–</td>
</tr>
<tr>
<td>10. Participant sex (1 = female)</td>
<td>0.09</td>
<td>0.05</td>
<td>0.05</td>
<td>0.15</td>
<td>–0.10</td>
<td>–0.07</td>
<td>–0.17</td>
<td>–0.05</td>
<td>–0.04</td>
<td>–</td>
<td>–</td>
<td></td>
</tr>
</tbody>
</table>

**Abbreviation:** NFR, nociceptive flexion reflex.

**NOTE:** Actual ranges of the scales are listed in parentheses next to the variable labels.

*Two-tailed \( P < .05. \)
catastrophic thinking simply biases pain report/behavior without altering the nociceptive signal. However, recent imaging research suggests catastrophizing during painful stimulation is correlated with activation of brain regions associated with attention to, and anticipation of, pain.\textsuperscript{19,49} Therefore, catastrophizing may engage cortico-cortical mechanisms that enhance (or remove inhibition of) nociception at the supraspinal level rather than within the spinal cord. Future research should attempt to experimentally manipulate catastrophizing while imaging supraspinal reactions to noxious stimulation to help delineate potential brain circuitry associated with pain catastrophizing.

The present study is not without its limitations, however. First, instructions for the pain catastrophizing subscale were not altered to specifically ask participants to reflect on their catastrophic thoughts during pain testing. Rather, we assumed that pain testing would provide a salient and recent experience on which participants could base their self-report. Therefore, it remains possible that catastrophizing scores would have significantly correlated with NFR thresholds had we altered the instructions, or had we assessed catastrophizing during pain testing. Second, the method of catastrophizing assessment (pre-test vs post-test) was not experimentally manipulated; thus, our design provided a weaker test of the influence of in vivo catastrophizing because we were unable to compare the 2 methods.\textsuperscript{54} Nonetheless, recent studies that did measure catastrophizing before and after pain testing suggests that pre-test and post-test assessments are poorly correlated and that post-test ratings are more strongly correlated with pain outcomes.\textsuperscript{6,10} Therefore, our study is likely to provide important insights into the relation of in vivo catastrophizing and spinal nociception.

The third limitation stems from our measure of spinal nociception. The NFR is an indirect measure of spinal nociception assessed from muscle activity corresponding to a nociception-initiated withdrawal reflex.\textsuperscript{48,50,59} As such, the NFR may not provide a complete picture of the complex processes associated with nociceptive process-

### Table 2. β-Weights, Semi-Partial Correlations (sr\textsuperscript{2}), and Inferential Statistics for Final Regression Models Predicting Pain-Related Outcomes

<table>
<thead>
<tr>
<th>Criterion (Model R\textsuperscript{2})</th>
<th>Predictors</th>
<th>β</th>
<th>sr\textsuperscript{2}</th>
</tr>
</thead>
<tbody>
<tr>
<td>NFR threshold (R\textsuperscript{2} = 0.03)</td>
<td>Sex (0 = male, 1 = female)</td>
<td>0.074</td>
<td>0.005</td>
</tr>
<tr>
<td></td>
<td>Depressive symptoms</td>
<td>0.010</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Self-efficacy</td>
<td>0.132</td>
<td>0.015</td>
</tr>
<tr>
<td></td>
<td>Pre-exp displeasure rating</td>
<td>0.115</td>
<td>0.012</td>
</tr>
<tr>
<td></td>
<td>Pre-exp arousal rating</td>
<td>−0.078</td>
<td>0.006</td>
</tr>
<tr>
<td></td>
<td>In vivo catastrophizing</td>
<td>0.063</td>
<td>0.003</td>
</tr>
<tr>
<td>Pain sensation ratings (R\textsuperscript{2} = 0.26)</td>
<td>Sex (0 = male, 1 = female)</td>
<td>−0.138</td>
<td>0.018</td>
</tr>
<tr>
<td></td>
<td>Depressive symptoms</td>
<td>−0.016</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>Self-efficacy</td>
<td>−0.290\textdagger</td>
<td>0.072\textdagger</td>
</tr>
<tr>
<td></td>
<td>Pre-exp displeasure rating</td>
<td>−0.074</td>
<td>0.005</td>
</tr>
<tr>
<td></td>
<td>Pre-exp arousal rating</td>
<td>0.056</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>NFR threshold</td>
<td>0.289\textdagger</td>
<td>0.081\textdagger</td>
</tr>
<tr>
<td></td>
<td>In vivo catastrophizing</td>
<td>0.245\textdagger</td>
<td>0.050\textdagger</td>
</tr>
<tr>
<td>Displeasure ratings (R\textsuperscript{2} = 0.20)</td>
<td>Sex (0 = male, 1 = female)</td>
<td>0.078</td>
<td>0.006</td>
</tr>
<tr>
<td></td>
<td>Depressive symptoms</td>
<td>0.058</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>Self-efficacy</td>
<td>−0.211\textasterisk</td>
<td>0.038\textasterisk</td>
</tr>
<tr>
<td></td>
<td>Pre-exp displeasure rating</td>
<td>−0.062</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>Pre-exp arousal rating</td>
<td>0.059</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>NFR threshold</td>
<td>0.262\textdagger</td>
<td>0.067\textdagger</td>
</tr>
<tr>
<td></td>
<td>In vivo catastrophizing</td>
<td>0.227\textdagger</td>
<td>0.043\textdagger</td>
</tr>
<tr>
<td>Arousal ratings (R\textsuperscript{2} = 0.10)</td>
<td>Sex (0 = male, 1 = female)</td>
<td>−0.110</td>
<td>0.011</td>
</tr>
<tr>
<td></td>
<td>Depressive symptoms</td>
<td>−0.067</td>
<td>0.004</td>
</tr>
<tr>
<td></td>
<td>Self-efficacy</td>
<td>0.031</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>Pre-exp displeasure rating</td>
<td>−0.037</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>Pre-exp arousal rating</td>
<td>0.208\textasterisk</td>
<td>0.040\textasterisk</td>
</tr>
<tr>
<td></td>
<td>NFR threshold</td>
<td>0.224\textasterisk</td>
<td>0.049\textasterisk</td>
</tr>
<tr>
<td></td>
<td>In vivo catastrophizing</td>
<td>0.051</td>
<td>0.002</td>
</tr>
</tbody>
</table>

Abbreviations: NFR, nociceptive flexion reflex; Pre-exp, pre-experiment ratings. sr\textsuperscript{2} provides the unique shared variance between a predictor and the criterion.

*P < .10.
†P < .05.
‡P < .01.
ing in the spinal cord. Indeed, numerous factors could influence motoric output of the NFR without influencing nociceptive transmission. To address these shortcomings, future research is needed that provides corroborating evidence from other measures of spinal nociception (for example, fMRI of dorsal horn activity).

Finally, methodology associated with the larger studies from which these data were drawn may have influenced our outcomes. These data came from 2 studies that examined the influence of emotional picture-viewing (phase 2) on nociception and pain. During picture-viewing, participants were exposed to 24 pictures varying in emotional content (for example, couples in sexual acts, animals attacking, household objects). NFR threshold and pain sensation ratings were assessed before picture-viewing, whereas pain catastrophizing and affective reactions to electric stimulation (displeasure, arousal) were measured after. Therefore, emotion-induction may have biased memory for, or the reporting of, catastrophizing and pain-related affective reactions.15,30,39 There are reasons to believe picture-viewing did not confound their measurement, however. Biases caused by emotional valence (pleasant vs unpleasant) should be washed out because there were equal numbers of pleasant, unpleasant, and neutral pictures presented, and all pictures were viewed by every participant. The order of picture presentation was randomized between subjects, so any effects resulting from the first or last picture (primacy or recency effects, respectively) should be washed out. Further, each picture was presented only briefly (6 seconds), and studies have shown that there is very little emotional carry-over effects after picture offset.3 Moreover, any bias caused by emotion-induction should have similarly affected the report of catastrophizing and affective reactions (given they were both measured after picture-viewing). Yet, the association between these variables was nonsignificant in the regression models. Nonetheless, it remains possible that emotion-induction had some effect on the measurement of catastrophizing and affective reactions to electric stimulation.

It is important that future research determine whether these findings replicate in clinical pain populations. Although 1 study has suggested that these results should generalize,15 a number of psychological and physiological changes occur in the development of chronic pain that could alter the catastrophizing-pain relation. For example, as pain becomes intractable, negative coping may increase, leading to greater and more frequent catastrophizing. The range of catastrophizing scores was large in our no-pain sample; however, clinical samples evidence higher mean catastrophizing and a wider range of scores.5,30,46 Catastrophizing may have a greater impact on pain and nociception at the upper tail of the catastrophizing distribution. Further, evidence suggests that persistent pain can be associated with changes in central nociceptive processing.36,56,63 These changes could modify the CNS such that neural mechanisms associated with catastrophizing gain access to descending circuitry, perhaps through deactivation of opioid receptors (given their importance in descending circuitry).11,13,64

In sum, this study suggests that in vivo pain catastrophizing is positively associated with subjective pain, even after controlling for depressive symptoms, current affect, self-efficacy, and participant sex. However, in vivo catastrophizing does not amplify the pain experience through descending modulation because catastrophizing scores did not correlate with the NFR (a measure of spinal nociception). Therefore, psychological interventions for the management of pain that target catastrophic thoughts are not likely to influence ascending nociception but rather supraspinal nociception or its interpretation.

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