Pain catastrophizing is a maladaptive coping strategy that exerts a significant negative influence on pain and pain-related outcomes. Catastrophizing is associated with hypersensitivity to noxious stimuli, greater pain-related disability and suffering, and increased risk for, and maintenance of, chronic pain conditions. In addition, pain catastrophizing is positively correlated with affective distress, such as depressive symptomatology, negative affectivity (trait negative affect), expectation of pain and psychological distress, and exaggerated emotional response to aversive stimuli. Taken together, there appears to be significant relationships between catastrophizing, affective processes, and pain.

Recent attempts have been made to determine the mechanisms associated with the pain-catastrophizing relationship. For example, catastrophizing has been found to correlate with cerebral activation during noxious stimulation, suggesting enhanced pain may stem from amplified supraspinal nociception.
In contrast, spinal nociceptive processes do not appear to be influenced by catastrophizing, because catastrophizing does not significantly correlate with nociceptive flexion reflex (NFR) threshold, a physiological marker of spinal nociceptive processes.\textsuperscript{15,16,41} This suggests catastrophizing does not facilitate pain by activating descending (brain–to–spinal cord) circuitry to modulate ascending spinal nociception. However, the search for a significant correlation between spinal nociception and catastrophizing assumes catastrophizing exerts a direct effect on descending circuits. Alternatively, pain catastrophizing could engage descending circuits indirectly through emotional processes.

Using a procedure referred to as emotional controls of nociception (ECON), we have shown that negative affect facilitates spinal nociception and pain, whereas positive affect inhibits spinal nociception and pain.\textsuperscript{38,39,42} In the ECON procedure, emotionally charged pictures are presented during which noxious electrodermal stimulations are delivered to elicit nociceptive reactions (nociceptive flexion reflex [NFR], heart rate [HR] acceleration, skin conductance response [SCR], and pain report). When noxious stimuli are delivered during unpleasant pictures (attack scenes), nociceptive reactions are enhanced (displeasure-induced facilitation); but, when noxious stimuli are delivered during pleasant pictures (erotica), nociceptive reactions are inhibited (pleasure-induced inhibition).\textsuperscript{38–40,42} Thus, catastrophizing could enhance nociceptive processes by augmenting negative emotion and/or reducing positive emotion.

ECON studies suggest emotion engages descending modulatory mechanisms, because the NFR (a spinal reflex) is modulated. The circuit responsible for this modulation probably includes the amygdala (in conjunction with the periaqueductal gray and rostral ventromedial medulla)\textsuperscript{36–38} because of its importance in emotional processing\textsuperscript{28} and nociception modulation.\textsuperscript{14,20,30,33} Interestingly, evidence suggests amygdala activation by emotional pictures does not necessarily correlate with subjective emotion.\textsuperscript{17} This implies pain catastrophizing could enhance pain by altering the neural processes associated with emotion without influencing emotional report. For example, catastrophizing could alter amygdala activity to cause enhanced displeasure-induced facilitation and/or reduced pleasure-induced inhibition, without influencing affective ratings of pictures. Thus, catastrophizing could moderate the relationship between emotion and nociception without moderating subjective reactions to emotional stimuli.

The present study used ECON procedures to address 2 questions: (1) Does pain catastrophizing moderate affective reactions to emotionally charged pictures, and (2) Does pain catastrophizing moderate the relationship between emotion and nociceptive reactivity? Given research suggesting catastrophizing is best measured during or after a pain-evoked stimulus,\textsuperscript{9,12} catastrophizing was assessed at the completion of pain testing. If pain catastrophizing indirectly activates descending circuits via emotional processes, then catastrophizing should be associated with greater negative affect in response to unpleasant pictures and/or less positive affect in response to pleasant pictures but also enhanced displeasure-induced nociceptive facilitation and/or reduced pleasure-induced nociceptive inhibition. However, if the influence of catastrophizing on emotional processes occurs outside of awareness, then only the latter effects would be observed. Alternatively, pain catastrophizing may not influence pain and nociception via emotional processes.

Materials and Methods

Data for the present study were derived from 2 previous experiments investigating affective modulation of pain and nociception.\textsuperscript{38,39} In the second experiment, half of the participants received predictable modulation of nociception,\textsuperscript{39} whereas the first study only presented unpredictable stimuli.\textsuperscript{38} Given that stimulus predictability was found to influence descending modulation,\textsuperscript{39} only data from unpredictable stimuli in both experiments were used in the present study.

Participants

A total of 53 (15 male, 38 female) undergraduate psychology students were participants. Most were white, non-Hispanic (76%), single (87%), and employed (60%), with an average age of 22.04 years (SD = 5.64). Participants were excluded for age <18 years; neurological, cardiovascular, and/or circulatory problems; recent use of analgesic, anxiolytic, or antidepressant medication; specific phobia of snakes or spiders (due to picture-viewing); chronic pain diagnosis; recent psychological trauma as defined by the DSM-IV\textsuperscript{1}; or Raynaud’s disease.

Procedure

All participants were provided an overview of the experiment before informed consent was obtained. Demographic information and health status were then assessed by questionnaires, and then eligibility was determined by a brief interview. Next, participants were instrumented and familiarized with the Self-Assessment Manikin (SAM) to rate affective reactions to pictures, as well as the numerical rating scale (NRS) to rate pain. Testing consisted of 2 phases. During phase 1, the nociceptive flexion reflex (NFR) threshold was determined (see below) to establish the stimulus intensity level (120% NFR threshold) that would be used during phase 2 (picture-viewing). Throughout phase 1, the NRS was presented on the participant’s computer screen and a green light was illuminated next to the scale when the participant was to make a pain rating (after each stimulation). During phase 2, participants viewed erotic, neutral, and threat-related (attack) pictures presented in pseudorandom order (no more than 2 pictures of similar content were shown consecutively). During picture-viewing, electric stimuli were randomly delivered during 50% of pictures (balanced across contents) 3 to 5 seconds after picture onset (latency randomly determined), and during 6 randomly determined inter-picture intervals (to reduce associations between pictures and shock). Each picture...
was presented for 6 seconds and inter-picture intervals varied randomly between 12 and 22 seconds. Psychophysiological recording trials began 3 seconds before each picture and continued for 2 seconds after picture offset (11 seconds total). After each picture was presented, participants rated their affective valence (pleasure) and arousal reactions to each picture using the computer-presented SAM. At the conclusion of phase 2, participants filled out the catastrophizing subscale of the Coping Strategies Questionnaire. Participants were then debriefed and provided course credit. All procedures were fully approved by the University of Tulsa ethics review board.

Apparatus

Data acquisition and stimulus and questionnaire presentation were controlled by a PC equipped with dual monitors, A/D board (PCI-6036E National Instruments, Austin, TX), and LabVIEW software. A 17-inch flat-panel monitor situated 0.5 meter from the participant presented questionnaires and picture stimuli. Physiological signals and experimental timing were monitored by the experimenter from an adjacent control room by an additional 17-inch 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; West Warwick, RI), stimulus isolation unit (Model SIU8T), constant current unit (Model CCU1), and bipolar stimulating electrode (Nicolet, 019-401400; Madison, WI) were used to deliver noxious electrodermal stimuli to the left ankle over the retromalleolar pathway of the sural nerve. Biceps femoris electromyogram (EMG) associated with the NFR was recorded by attaching 2 active Ag-AgCl electrodes over the left biceps femoris muscle 10 cm superior to the popliteal fossa. A common ground electrode was attached to the lateral epicondyle of the left femur. To initiate relaxation of the leg muscles, participants were seated in a recliner and a small pillow was situated underneath the left ankle. The procedures for phase 1 of the experiment (NFR threshold assessment) were adapted from France and colleagues. Electric stimulations (trains of 5, 1-ms rectangular wave pulses at 250 Hz) were delivered to the sural nerve according to a variable interval of 8 to 12 seconds. The first trial started at 0 mA (current) and increased in 1.5 mA steps until an NFR was detected. The NFR 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 prestimulus baseline interval by at least 1 standard deviation. Potential contamination by the non-nociceptive RII reflex, startle responses, and/or voluntary movement is avoided by using the 90- to 150-ms timeframe. The stimulation intensity was then decreased in .75-mA steps until an NFR was no longer observed. This up-down staircase process was repeated 2 more times, but with the use of .5-mA steps. The average stimulation intensity (in mA) of the last 2 peaks and troughs was multiplied by 120% to establish the intensity of stimulation used throughout phase 2 (picture-viewing). NFR magnitude was inspected and calculated off-line for stimulations delivered during phase 2 (picture-viewing) by subtracting mean activity in the 60 ms prior to electrical stimulus onset from mean activity in the 90- to 150-ms post-stimulation window.

Skin Conductance Response

Skin conductance response (SCR, a measure of sympathetic nervous system activation) was measured from 2 electrodes filled with isotonic paste (EC33, Grass Instruments) that were applied to the distal volar surface of the middle and ring fingers on the participant’s nondominant hand after the skin had been washed and dried. Initially, skin conductance level during the 11-second recording trial was averaged by 0.5-second epochs, and then the mean activity in the 1-second pre-shock interval was subtracted from each 0.5-second post-shock ep-
SCR is defined as the maximum skin conductance increase in the 1- to 4-second post-event interval\(^7\); therefore, the .5-second post-shock epoch with the greatest positive change within the 1- to 4-second post-shock interval was used as the response definition.\(^{40,42}\)

### Heart Rate Acceleration

The ECG was measured by applying two Ag-AgCl electrodes with conductive gel (EC60, Grass Instruments) to the right and left forearms. ECG was converted off-line to heart rate (HR) in beats per minute from interbeat intervals. The HR waveform was then averaged by 0.5-second epochs and the mean activity in the 1-second pre-shock interval was subtracted from each 0.5-second post-shock epoch. HR acceleration was defined as the maximum increase in the 1- to 5-second post-shock interval.\(^{40,42}\)

### Subjective Pain Ratings

A computer-presented numerical rating scale (NRS) was used to assess subjective pain. 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.

### Questionnaires

#### Catastrophizing

The 6-item subscale of the Coping Strategies Questionnaire (CSQ) was used to assess catastrophizing at the end of pain testing. This subscale measures catastrophic thoughts about pain and has good internal and test-retest reliabilities.\(^{43}\) Scores range from 0 to 36. The mean catastrophizing score in the current sample was 8.11 (SD = 5.57), with an observed range of 0 to 25. Standard CSQ instructions were used in the present study.

#### Affective Ratings: Self-Assessment Manikin

A computerized version of the Self-Assessment Manikin (SAM)\(^7\) was used to evaluate affective reactions to each picture.\(^{38}\) Two sets of 5 pictographs illustrating affective valence/pleasure (unpleasant-pleasant) and arousal (calm-excited) comprise the SAM. To respond, participants dragged an indicator on or in between any of the five pictographs for each scale and submitted their answers by computer mouse. Ratings between 1 and 9 were generated for each dimension with higher scores typifying greater pleasure or arousal.

### Data Reduction and Analyses

Four nociceptive reactions to noxious electric stimuli were assessed (NFR magnitude, SCR, HR acceleration, subjective pain ratings). These nociceptive reactions were transformed to \(z\) scores within-subjects and then averaged by picture content. Converting to \(z\) scores allows the reactions to be placed on a common metric (standard deviation units), thus easing interpretability and comparison across the reactions. Additionally, the \(z\) score transformation can help the distributional properties of the variables (ie, normality), improves statistical power by removing arbitrary between-subject variance that is not explained by the independent variables from the ANOVA error terms, and allows comparison to other studies of emotional modulation.\(^{38,39,42}\) However, conversion to \(z\) scores eliminates the ability to determine the main effect of pain catastrophizing on emotional and nociceptive reactivity, because the between-subject variance in the dependent variables is set to zero.

To determine whether pain catastrophizing moderated reactions to emotional picture stimuli, SAM pleasure (valence) and arousal ratings were each analyzed using ANCOVAs with picture valence (unpleasant, neutral, pleasant) and participant sex (male, female) entered as the within-subject variables, and pain catastrophizing as a continuous covariate.

Although the 4 nociceptive reactions index different response systems, it is assumed that they tap into a single latent construct (eg, nociceptive responding). To verify this, we initially conducted a principal components analysis on the standardized reactions. The Bartlett’s test of sphericity suggested there was significant covariance among the reactions (\(P < .001\)), and an examination of the scree plot and eigenvalues suggested a single component should be retained that explained 50% of the variance in the reactions. Therefore, to determine whether pain catastrophizing moderated the emotional-pain relationship, a single 3 (Picture Valence) \(\times\) 2 (Participant Sex) \(\times\) 4 (Nociceptive Reactions: NFR, SCR, HR, pain) ANCOVA model was created with catastrophizing entered as a continuous covariate. The use of a single model (which is facilitated by the \(z\) score transformations) also reduces family-wise type I error rate and provides a statistical test of the Picture Valence \(\times\) Nociceptive Reaction interaction that determines whether the reactions are modulated in parallel by emotional picture-viewing. [For comparison, however, we conducted 3 (Picture Valence) \(\times\) 2 (Participant Sex) repeated-measures ANCOVAs, with pain catastrophizing entered as the covariate, separately for each unstandardized nociceptive reaction. The results of these analyses generally agree with those reported on the standardized reactions analyzed together. Pain catastrophizing did not moderate the influence of emotional picture-viewing on nociceptive reactions. Moreover, the main effect of sex, and the interactions of Sex \(\times\) Catastrophizing and Picture Valence \(\times\) Sex \(\times\) Catastrophizing were nonsignificant in all models (\(Ps > .05\)). The Picture Valence \(\times\) Sex interaction was nonsignificant in all models (\(Ps > .05\)), except the model predicting NFR (\(P = .04\)). This was due to a single male outlier value that was corrected by \(z\)-score transformation (thus this effect is not present in the transformed data). The main effect of pain catastrophizing was nonsignificant in all models (\(Ps > .05\)), except the model predicting pain ratings (\(P = .02\)). Higher catastrophizing was associated with higher pain ratings.]

All ANCOVA models included a Picture Valence \(\times\) Pain Catastrophizing interaction to specifically test study hy-
potheses. It is important to enter pain catastrophizing as a continuous covariate (rather than creating arbitrary groups) because it makes use of the total variance of the scale. However, to report the Picture Valence $\times$ Catastrophizing data in Tables, 3 arbitrary groups were formed based on catastrophizing score tertiles. The group numbers as well as means and standard deviations of catastrophizing scores for each group were: low catastrophizers ($N = 22; M = 3.18, SD = 1.59$), medium catastrophizers ($N = 16, M = 8.19, SD = 1.80$), and high catastrophizers ($N = 15, M = 15.27, SD = 3.88$). Partial eta-squared ($\eta^2$) was used as the effect size for $F$ tests and Cohen's $d$ was used for mean comparisons. Cohen's $d$ provides guidelines for interpreting $\eta^2$ (small = .01, medium = .06, large = .14) and $d$ (small = .2, medium = .5, large = .8). To overcome problems with sphericity, Greenhouse-Geisser corrections were used and epsilons ($\epsilon$) are reported when appropriate.

Results

Affective Reactions to Pictures

Table 1 reports the Pain Catastrophizing $\times$ Picture Valence interactions for SAM valence/pleasure and arousal ratings. The analysis of pleasure ratings resulted in a significant main effect of picture valence $[F(2,98) = 25.93, P < .001, \eta^2 = .35, \epsilon = .85]$ but not a Pain Catastrophizing $\times$ Picture Valence interaction $[F(2,98) = 2.49, P = .10, \eta^2 = .048, \epsilon = .85]$. Compared with neutral pictures, erotic and attack pictures elicited greater arousal ($Ps < .001, d > 1.09$). The main effects of sex and pain catastrophizing, and all interactions with these independent variables were non-significant ($Ps > .05$). Together, results of SAM ratings suggest pain catastrophizing does not moderate affective reactions to standardized emotional stimuli.

Table 1. Means and Standard Errors of Pleasure and Arousal Ratings in Response to Different Picture Contents by Catastrophizing Subgroups

<table>
<thead>
<tr>
<th></th>
<th><strong>ATTACK</strong></th>
<th><strong>NEUTRAL</strong></th>
<th><strong>EROTIC</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MEAN</strong></td>
<td><strong>SEM</strong></td>
<td><strong>MEAN</strong></td>
<td><strong>SEM</strong></td>
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<tr>
<td>Pleasure (valence ratings 1–9)</td>
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<td></td>
<td></td>
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<tr>
<td>Low catastrophizing</td>
<td>2.91</td>
<td>0.25</td>
<td>4.76</td>
</tr>
<tr>
<td>Med catastrophizing</td>
<td>2.22</td>
<td>0.37</td>
<td>4.94</td>
</tr>
<tr>
<td>High catastrophizing</td>
<td>2.30</td>
<td>0.38</td>
<td>3.98</td>
</tr>
<tr>
<td>Arousal ratings (1–9)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low catastrophizing</td>
<td>5.68</td>
<td>0.41</td>
<td>2.25</td>
</tr>
<tr>
<td>Med catastrophizing</td>
<td>5.47</td>
<td>0.60</td>
<td>2.64</td>
</tr>
<tr>
<td>High catastrophizing</td>
<td>5.88</td>
<td>0.61</td>
<td>2.99</td>
</tr>
</tbody>
</table>

NOTE: Analyses treated catastrophizing as a continuous variable, but for tabular reporting, catastrophizing subgroups (Low, Med, High) were created based on tertiles.

Nociceptive Reactions

Fig 1 illustrates the effect of picture valence on each standardized nociceptive reaction and Table 2 reports the Pain Catastrophizing $\times$ Picture Valence interaction for standardized and unstandardized (for comparison) nociceptive reactions.

Figure 1. Influence of picture-viewing (attack, neutral, erotic) on z-score–transformed nociceptive reactions (pain ratings, nociceptive flexion reflex [NFR] magnitude, skin conductance response [SCR], heart rate [HR acceleration]). Superimposed line represents the main effect of picture valence on the multivariate combination of the 4 nociceptive reactions. Attack pictures facilitated nociceptive reactions (displeasure-induced facilitation) and erotic pictures inhibited reactions (pleasure-induced inhibition). Pain catastrophizing did not moderate the influence of emotional picture-viewing on nociceptive reactivity. NFR = nociceptive flexion reflex; SCR = skin conductance response; HR Accel = heart rate acceleration; Combined = multivariate combination of the 4 nociceptive reactions.
reactions. The analysis of nociceptive reactions resulted in a significant main effect of picture valence \(F(2,100) = 10.34, P < .001, \eta^2 = .17\) but not a Pain Catastrophizing \(\times\) Picture Valence interaction \(F(2,100) = .78, P = .46, \eta^2 = .015\). The main effects of sex, pain catastrophizing, and nociceptive reaction and all interactions with these independent variables were nonsignificant \((Ps > .05)\). Compared with neutral pictures, nociceptive reactions were inhibited during erotic pictures \((Ps < .001, d = 1.46)\) and facilitated during attack pictures \((Ps < .05, d = .46)\) (Fig 1). Because the interactions of Picture Valence \(\times\) Nociceptive Reaction and Picture Valence \(\times\) Nociceptive Reaction \(\times\) Pain Catastrophizing were nonsignificant, this suggests all reactions were modulated in parallel. As an additional safeguard against the possibility of a type II error (failure to find a true interaction) because all nociceptive reactions were analyzed together, ANCOVA analyses were conducted on each nociceptive reaction separately to examine the Pain Catastrophizing \(\times\) Picture Valence interaction (only this effect was examined to minimize type I error rate). The Pain Catastrophizing \(\times\) Picture Valence interaction was nonsignificant in all 4 analyses: Pain ratings \((P = .40, \eta^2 = .018)\), NFR \((P = .82, \eta^2 = .004)\), SCR \((P = .69, \eta^2 = .008)\), HR acceleration \((P = .58, \eta^2 = .011)\). Together, these analyses imply catastrophizing does not influence nociceptive processing by moderating the influence of emotion.

Discussion

Prior research suggests associations between pain catastrophizing, affective processes, and pain.\(^{22,24,49,55}\) Given these relationships, we reasoned that one mechanism by which pain catastrophizing could enhance pain and nociception is through emotional processes. The current study used an experimental paradigm to determine the influence of pain catastrophizing on (1) subjective emotional reactions to erotic, neutral, and threat-related picture stimuli, and (2) the relationship between emotion and nociceptive reactions. Results suggest affective reac-

| Table 2. Means and Standard Errors of Standardized and Unstandardized Nociceptive Reactions Evoked During Different Picture Contents by Catastrophizing Subgroups |
|---------------------------------|-----------------|-----------------|-----------------|
|                                  | **ATTACK**      | **NEUTRAL**     | **EROTIC**      |
|                                  | **MEAN**        | **SEM**         | **MEAN**        | **SEM**         | **MEAN**        | **SEM**         |
| Standardized reactions           |                 |                 |                 |
| Pain ratings \(z\) score         |                 |                 |                 |
| Low catastrophizing              | 0.21            | 0.10            | 0.22            | 0.10            | -0.43           | 0.09            |
| Med catastrophizing              | 0.26            | 0.12            | -0.02           | 0.12            | -0.24           | 0.11            |
| High catastrophizing             | 0.22            | 0.13            | 0.07            | 0.12            | -0.29           | 0.12            |
| Nociceptive flexion reflex \(z\) |                 |                 |                 |
| Low catastrophizing              | 0.22            | 0.10            | 0.23            | 0.09            | -0.46           | 0.09            |
| Med catastrophizing              | 0.26            | 0.13            | -0.02           | 0.11            | -0.25           | 0.11            |
| High catastrophizing             | 0.23            | 0.13            | 0.04            | 0.12            | -0.27           | 0.11            |
| Skin conductance response \(z\) |                 |                 |                 |
| Low catastrophizing              | 0.11            | 0.10            | 0.07            | 0.09            | -0.18           | 0.10            |
| Med catastrophizing              | 0.08            | 0.12            | -0.08           | 0.11            | 0.00            | 0.13            |
| High catastrophizing             | 0.32            | 0.12            | -0.07           | 0.12            | -0.25           | 0.13            |
| Heart rate acceleration \(z\)   |                 |                 |                 |
| Low catastrophizing              | 0.05            | 0.09            | 0.23            | 0.07            | -0.28           | 0.07            |
| Med catastrophizing              | 0.17            | 0.11            | 0.01            | 0.09            | -0.17           | 0.09            |
| High catastrophizing             | 0.18            | 0.11            | 0.11            | 0.09            | -0.29           | 0.10            |
| Unstandardized reactions         |                 |                 |                 |
| Pain ratings \(0–100\)           |                 |                 |                 |
| Low catastrophizing              | 36.21           | 4.55            | 36.42           | 4.51            | 30.87           | 4.34            |
| Med catastrophizing              | 61.48           | 5.68            | 58.31           | 5.63            | 57.14           | 5.42            |
| High catastrophizing             | 57.35           | 5.81            | 54.97           | 5.77            | 51.87           | 5.54            |
| Nociceptive flexion reflex \(\Delta \mu V\) |                 |                 |                 |
| Low catastrophizing              | 6.79            | 0.94            | 8.28            | 1.21            | 4.58            | 0.73            |
| Med catastrophizing              | 6.71            | 1.18            | 6.31            | 1.51            | 4.85            | 0.91            |
| High catastrophizing             | 6.29            | 1.21            | 6.69            | 1.54            | 4.53            | 0.93            |
| Skin conductance response \(\Delta \mu S\) |                 |                 |                 |
| Low catastrophizing              | 0.74            | 0.18            | 0.75            | 0.19            | 0.60            | 0.15            |
| Med catastrophizing              | 0.55            | 0.23            | 0.39            | 0.23            | 0.53            | 0.18            |
| High catastrophizing             | 0.60            | 0.23            | 0.48            | 0.24            | 0.47            | 0.19            |
| Heart rate acceleration \(\Delta\) |                 |                 |                 |
| Low catastrophizing              | 4.82            | 0.90            | 6.29            | 0.88            | 3.66            | 0.79            |
| Med catastrophizing              | 6.68            | 1.12            | 5.83            | 1.10            | 4.76            | 0.99            |
| High catastrophizing             | 8.09            | 1.15            | 7.52            | 1.12            | 4.68            | 1.01            |
tions were not moderated by catastrophizing. Pleasure ratings were highest for erotic pictures and lowest for attack pictures, and arousal was higher during erotic and attack pictures relative to neutral pictures. These results were similar across all levels of pain catastrophizing (Table 1). Moreover, results suggest catastrophizing does not moderate the influence of emotion on pain and nociception. Nociceptive reactions were facilitated by unpleasant (attack) pictures and inhibited by pleasant (erotic) pictures. Again, the results were similar across all levels of pain catastrophizing (Table 2). Together, these results suggest that catastrophizing does not influence supraspinal nociception via emotional processes. They also provide further evidence that pain catastrophizing does not engage descending mechanisms to modulate spinal nociception. Indeed, post hoc analyses on separate nociceptive reactions found the interaction with catastrophizing explained less than 0.7% of the variance in NFR.

Recent imaging studies have reported a correlation between pain catastrophizing and cortical activation during noxious stimulation, implying that supraspinal processing of nociception is altered. Thus, pain catastrophizing may exert its effects through cortico-cortical mechanisms that enhance (or remove inhibition of) supraspinal nociception. Our results suggest catastrophizing does not influence supraspinal nociception via emotional processes, however. We used heart rate (HR) acceleration and skin conductance (SCR) reactions as indirect measures of supraspinal nociception, because autonomic outflow is mediated by supraspinal centers (e.g., hypothalamus and brainstem nuclei). Pain catastrophizing did not moderate how these measures were influenced by emotional picture-viewing. Nonetheless, research is needed to replicate our effects with other indices of supraspinal nociception, e.g., cortical evoked potentials in response to noxious shocks.

Given that emotional processes and pain catastrophizing were not found to interact to influence nociceptive reactivity, this may mean emotional processes and pain catastrophizing modulate pain through independent mechanisms. This finding is clinically relevant, in that, interventions designed to manage pain should target each of these variables independently. Although research has found a positive relationship between pain catastrophizing and negative affect, it cannot be assumed that reducing one will eliminate the pain enhancing effects of the other. A comprehensive approach to pain management that reduces pain catastrophizing, reduces negative emotions, and increases positive emotions, is likely to produce the most positive outcomes.

It is important that future research determine whether our findings extend to persons suffering from chronic pain, however. The development of chronic pain can lead to a number of psychological and physiological changes that may alter emotion and/or pain modulation circuitry. Such changes could facilitate a stronger relationship between catastrophizing, affective processing, and pain. Additionally, pain catastrophizing may have a greater impact on pain in clinical populations. This idea is supported by research demonstrating that clinical samples tend to catastrophize more, and that there is a wider range of individual differences in catastrophizing in clinical populations. Therefore, pain catastrophizing may modulate pain indirectly via emotional processes in chronic pain populations.

It is prudent to interpret null results with caution. Our analyses may have been underpowered to detect the interaction with pain catastrophizing (type II error). While this is a possibility, our sample size was reasonably large (N = 53), and, more importantly, our effect size estimates for the interaction with pain catastrophizing were small, explaining 4.8% of the variance or less (1.5% in nociceptive reactions). The effect sizes were also small when the nociceptive reactions were analyzed separately in post hoc analyses (1.8% or less). These effect size estimates suggest: (1) our results are not likely due to type II error, and (2) the moderating effect of pain catastrophizing is not likely to be clinically meaningful even if a type II error was made.

Our study was not designed to examine the main effect of catastrophizing on nociceptive reactivity. Rather, we wanted to test whether pain catastrophizing moderated within-subject changes in our dependent variables. Therefore, prior to analyses, all nociceptive reactions were converted to a z score within subjects. This technique has several advantages, such as placing the variables on a common metric to facilitate comparisons across measures, improving the distributional properties of the variables, allowing a single ANCOVA model to analyze all nociceptive reactions simultaneously (reducing type I error rate and providing a statistical test of parallel modulation), and removing arbitrary between-subject variance while retaining within-subject variance (increasing power). In doing so, every participant’s mean response was set to zero. This eliminated the possibility that individual differences in catastrophizing could be correlated with individual differences in nociceptive reactivity in the analyses of standardized data, because the between-subject variance was zero. However, for comparison, preliminary analyses did test the main effect of catastrophizing and found that it was only significant in the analysis of pain ratings (higher catastrophizing = higher pain ratings). This is consistent with previous studies that have found catastrophizing predicts subjective pain, but not the NFR.

**Study Limitations**

A few limitations of the present study are worth noting. Pain catastrophizing was assessed by the 6-item subscale of the Coping Strategies Questionnaire (CSQ). CSQ catastrophizing primarily taps the helplessness component of catastrophizing. In other conceptualizations, catastrophizing is seen as a multidimensional construct including helplessness, but also magnification and rumination components. It is possible that magnification and rumination would have moderated emotion and/or emotional modulation of nociception. A follow-up study is underway to test this hypothesis.

A second limitation stems from our use of standardized
emotional pictures to evoke emotional responses. Cata-
strophizing may only moderate reactions to emotional
stimuli if the stimuli represent a viable and specific threat
to physical integrity (eg, pain cue). Although unpleas-
ant pictures depicted threatening scenes (eg, humans
with weapons attacking the viewer, snakes attacking the
viewer), these may not have been personally-relevant
and/or threatening enough for pain catastrophizing to
have exerted an influence. Indeed, pain catastrophizing
may modulate emotional reactions only to stimuli that
represent pain.

Based on evidence suggesting catastrophizing should
be assessed during or after pain testing, we chose to
assess catastrophizing at the end of the experiment,
after pain testing was completed. However, unlike
these other studies, our paradigm involved an emotion
manipulation that may have biased participants cata-
strophizing ratings. There are reasons to believe
emotion-induction did not bias the measurement of
catastrophizing, however. Most importantly, our analy-
yses suggested catastrophizing did not influence affective
reactions. Had emotional picture-viewing influenced
catastrophizing, a relationship between affective re-
tions and catastrophizing would have been expected.
Furthermore, emotional biases due to a particular affective
state (pleasant versus unpleasant) should have been
eliminated by our design. There were equal numbers of
erotic, attack, and neutral pictures, all pictures were
viewed by every participant, and the order of pictures
was randomized. So, any primacy or recency effects
should have been washed out across participants. More-
over, each picture was only presented for 6 s. Studies
have shown there is very little emotional carry-over ef-
fects with pictures of this duration. Thus, there is little
evidence to suggest catastrophizing scores were biased.

Summary

In summation, results from this study indicate that pain
catastrophizing did not moderate subjective reactions to
affective stimuli, nor did it moderate the influence of
emotion on nociceptive reactions (NFR, SCR, HR, subject-
ive pain). This suggests pain catastrophizing does not
modulate pain indirectly through emotional processes.
This also implies that catastrophizing and emotion may
alter pain through distinct processes. Thus, clinical inter-
ventions aimed at managing pain may prove more suc-
cessful if emotional distress and catastrophic cognitions
are addressed separately in treatment.

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