Published in final edited form as:
Pain. 2002 October ; 99(3): 459–463.

Catastrophizing is related to pain ratings, but not nociceptive flexion reflex threshold

Christopher R. France[a,*], **Janis L. France**[a], **Mustafa al'Absi**[b], **Christopher Ring**[c], and **David McIntyre**[c]

a *Department of Psychology, Ohio University, 245 Porter Hall, Athens, OH 45701, USA*

b *Department of Behavioral Sciences, University of Minnesota School of Medicine, Duluth, MN, USA*

c *School of Sport and Exercise Sciences, University of Birmingham, Birmingham, UK*

Abstract

Catastrophizing is reliably associated with increased reports of clinical and experimental pain. To test the hypothesis that catastrophizing may heighten pain experience by increasing nociceptive transmission through spinal gating mechanisms, the present study examined catastrophizing as a predictor of pain ratings and nociceptive flexion reflex (NFR) thresholds in 88 young adult men (n = 47) and women (n = 41). The NFR threshold was defined as the intensity of electrocutaneous sural nerve stimulation required to elicit a withdrawal response from the biceps femoris muscle of the ipsilateral leg. Participants completed an assessment of their NFR threshold and then provided pain ratings using both a numerical rating scale (NRS) and the short-form McGill pain questionnaire (SF-MPQ). Pain catastrophizing was assessed using the catastrophizing subscale of the coping strategies questionnaire (CSQ). Although catastrophizing was positively related to both NRS and SF-MPQ pain ratings, catastrophizing was not significantly related to NFR threshold. These findings suggest that differential modulation of spinal nociceptive input may not account for the relationship between catastrophizing and increased pain.

Keywords

Pain; Nociceptive flexion reflex; Catastrophizing

1. Introduction

Catastrophizing – the tendency to ruminate, magnify, or feel helpless about pain (Rosenstiel and Keefe, 1983; Sullivan et al., 1995; Keefe et al., 1989; Keefe et al., 2000) – has been shown to contribute to the experience of pain in a number of ways. In individuals with pain disorders, the tendency to focus on and exaggerate the threat value of painful stimuli and/or to negatively evaluate one's ability to deal with pain has been shown to predict increases in pain reported over time (Keefe et al., 1989), severity of reported pain (Tan et al., 2001), emotional distress in response to pain (Severeijns et al., 2001, Turner et al., 2001), and disability in response to pain (Sullivan et al., 1998; Severeijns et al., 2001; Turner et al., 2001). Further, interventions that lead to improvements in catastrophizing, and in related psychological variables such as self-efficacy and perceived control over pain, have been shown to be associated with reductions in chronic pain (Parker et al., 1988; Keefe et al., 1990a,b; Jensen et al., 2001). Higher levels of catastrophizing are also associated with greater pain reports during experimental pain

* Correspondence author. Tel.: +1-740-593-1079; fax: +1-740-593-0579. *E-mail address:*france@ohio.edu (C.R. France)..

(Sullivan et al., 1995, Sullivan et al., 1997) and painful medical or dental procedures (Sullivan et al., 1995, Sullivan and Neish, 1998).

In a recent paper, Sullivan et al. (2001) attempt to place the consistent relationships observed between catastrophizing and pain within a broader theoretical context. They posit that conceptually overlapping models of schema-activation and appraisal both suggest that catastrophizing may contribute to heightened pain experience by increasing attentional focus on pain and/or by increasing emotional reactivity to pain. They further suggest that, consistent with the gate control theory of pain (Melzack and Wall, 1984), cognitive and emotional responses associated with catastrophizing may heighten pain experience by increasing nociceptive transmission through spinal gating mechanisms. Although the existing literature provides convincing evidence that catastrophizing is associated with higher levels of reported pain, it is yet to be determined if catastrophizing is associated with alterations in supraspinal modulation of nociceptive inputs. The nociceptive flexion reflex (NFR) paradigm allows for a non-invasive assessment of this hypothesis. The NFR is a polysynaptic spinal reflex subserving withdrawal from potentially noxious stimuli. Individual differences in the intensity of electrocutaneous sural nerve stimulation required to elicit the NFR provide indirect evidence of supraspinal modulation, with higher thresholds suggesting enhanced descending inhibition and lower thresholds suggesting decreased inhibition of spinal nociceptive transmission (Willer et al., 1979; Kiernan et al., 1995; Danziger et al., 1998). For example, Willer et al. (1979) reported that NFR was dampened during performance of a mental arithmetic task, and enhanced during anticipation of an intense noxious stimulus. More recent studies have examined the effects of hypnosis on NFR (Kiernan et al., 1995; Danziger et al., 1998), and have demonstrated that hypnotic suggestion of analgesia results in strong NFR inhibition in some participants while others show reflex facilitation. Animal studies provide convincing evidence of supraspinal modulation of nociceptive flexion reflexes, with the periaqueductal gray playing a major role in the control of spinal nociceptive neurons through relays in the rostral ventromedial medulla (Willis, 1988; Fields and Basbaum, 1999). As the periaqueductal gray receives direct input from the medial prefrontal cortex, the insular cortex, and the hypothalamus (Fields and Basbaum, 1999), higher cognitive functions are likely to be involved in this descending modulation pathway.

To sum up, based on the existing empirical and physiological evidence, it is conceivable that individuals who are high and low on pain catastrophizing show differential supraspinal modulation of nociceptive input when faced with potentially painful stimulation. To address the possibility that elevated levels of pain catastrophizing may be associated with reductions in descending inhibition of spinal nociceptive neurons, the present study examined the relationship between pain catastrophizing and NFR threshold in healthy young adults.

2. Methods

2.1. Participants

One hundred young adult men and women met inclusion criteria and participated in the laboratory protocol. To be eligible, participants had to be in good physical health as indicated by the absence of chronic or acute illness. The sample had a mean age of 19.5 (SD = 1.7) years, body mass index of 24.5 kg/m^2 (SD = 3.9), and was 79% White, 9% Asian, 7% Black, 3% Hispanic, and 2% American Indian. Participants were recruited from Ohio University and the University of Minnesota and received $20/h for their participation.

Data from 12 participants were not included in the final analyses either because they failed to provide complete pain ratings ($n = 2$), reached their tolerance for electrocutaneous stimulation before an NFR was observed ($n = 5$), or did not show a reflex prior to the maximum stimulation

intensity of 40 mA (n = 5). These participants were eliminated from all analyses, resulting in a final sample of 88 (47 men and 41 women).

2.2. Initial screening

Potential participants completed an initial screening questionnaire designed to identify healthy men and women. Those who indicated a history of major medical problems or routine use of medication (other than birth control) were not recruited. Interested participants completed a brief medical screening to confirm the absence of any medical contraindications to testing. Following the medical screening, participants were scheduled for a laboratory testing session. Participants were asked to refrain from caffeine, nicotine, alcohol, and strenuous exercise for at least 4 h before their arrival at the laboratory, and from analgesic medication for 24 h before testing. To control for the potential effects of menstrual cycle phase on pain, women were tested within 7 days of onset of menses.

2.3. Laboratory testing procedure

To begin the protocol, informed consent and measurements of height and weight were obtained. Participants were excluded if their body mass index (kg/m^2) exceeded 35, as high levels of subcutaneous fat can interfere with accurate assessment of the nociceptive flexion reflex (no participant met this exclusion criterion). Participants then completed a brief questionnaire to assess adherence to the requested dietary and exercise restrictions, and a series of questionnaires including the coping strategies questionnaire (CSQ). As part of a separate investigation, participants were randomly assigned to receive a gel capsule that contained either placebo or 50 mg of naltrexone 70 min prior to the NFR assessment. At the time of testing, neither the experimenter nor the participant was aware of the gel capsule contents.

2.3.1. Electrode attachment—NFR recording and stimulation electrode sites were cleaned and abraded with Omni Prep electrode paste, and an impedance of less than 10,000 Ohm was verified using a UFI Checktrode (model MKII). Electromyographic (EMG) activity was recorded using a DelSys, Bagnoli-2 EMG amplifier. For the NFR recording, the differential EMG electrode was placed over the left biceps femoris muscle 10 cm superior to the popliteal fossa, with a reference electrode attached over the lateral epicondyle of the femur. EMG was recorded and processed using a CED Micro1401 analog-to-digital converter and Spike2 software. To elicit the NFR, a Nicolet bar electrode (anode inferior) was attached to the left leg over the retromalleolar pathway of the sural nerve. Electrical stimulation was delivered using a Digitimer, DS7A constant-current stimulator.

2.3.2. Assessment of NFR—After electrode placement, participants were seated in a Hi-Seat rehabilitation chair (model 2000) with a left leg rest adjusted to maintain knee flexion at approximately 60° from horizontal position . Electrical stimulation was applied over the sural nerve according to a variable interval schedule of 20 s (range 15–25 s) to prevent stimulus habituation and predictability. Each stimulation trial consisted of a volley of five 1 ms rectangular pulses with a 3-ms interpulse interval (total duration = 17 ms). Using an up–down staircase method (Levitt, 1971), stimulation intensity was increased in 4 mA increments until an NFR was detected (or a maximum intensity of 40 mA was reached) and then was decreased in 2 mA increments until a reflex was no longer detected. Continuing from this intensity, the procedure was then repeated using 1 mA increments so that the reflex appeared and subsided two more times. NFR occurrence was defined as a mean EMG response in the 90–150 ms post-stimulation interval that exceeded mean EMG activity during the 60 ms pre-stimulation baseline (from −65 to −5 ms) interval by at least 1.5 standard deviations. As in previous studies conducted in our laboratory (Page and France, 1997; France and Suchowiecki, 2001), the 90–150 ms interval was chosen because it avoids possible contamination by the low-threshold cutaneous flexor reflex, startle reactions, and voluntary movements. NFR threshold (in mA)

was defined as the average of the peaks during the last two ascending sequences (current intensity that elicited a reflex) and troughs during the last two descending sequences (current intensity that no longer elicited a reflex).

During the NFR threshold assessment, participants rated the perceived magnitude of each stimulation using a numerical rating scale. The scale, which was displayed in front of the participant throughout the NFR assessment, included a vertical line graphic labeled with intensity-denoting adjectives and numbers. Starting from the bottom of the graphic, the scale was labeled one (sensory threshold), 25 (uncomfortable), 50 (painful), 75 (very painful), and 100 (maximum tolerable). Similar rating scales have been used in previous NFR studies (Willer et al., 1979; Chan and Dallaire, 1989; Page and France, 1997; France and Suchowiecki, 1999, 2001; Anderson et al., 2000), and individual pain ratings have been shown to correlate significantly (r = 0.58–0.95) with NFR thresholds (Chan and Dallaire, 1989; Porchet et al., 1990; Boureau et al., 1991; Dowman, 1991). In general, numerical pain rating scales have been shown to have high levels of reliability and validity in the assessment of pain intensity, and scores obtained from such scales can be treated as ratio data (Jensen and Karoly, 1992). In the event that a participant provided a rating of 100, the procedure was discontinued. Immediately following NFR threshold assessment, participants provided a retrospective rating of pain during the NFR assessment using the pain rating scale anchors described above and the pain rating index of the short-form McGill pain questionnaire (SF-MPQ).

2.3.3. Electrocutaneous pain threshold and tolerance assessment—

Approximately 20 min after the NFR threshold assessment, pain threshold and tolerance were assessed during electrocutaneous sural nerve stimulation using the same stimulation parameters described above (i.e. variable interval schedule of 20 s; volley of five 1 ms rectangular pulses with a 3-ms interpulse interval). Participants rated the perceived intensity of each stimulation pulse using the aforementioned pain-rating scale. Stimulation intensity was increased in 2 mA increments until either a maximum intensity of 40 mA was reached or the participant gave a rating of 100. Pain threshold was defined as the first stimulation intensity that received a rating of 50 (painful) or greater. Pain tolerance was defined as the stimulation intensity corresponding to a rating of 100 (maximum tolerable). Participants then completed a retrospective rating of the pain tolerance assessment procedure using SF-MPQ. Upon completion of the questionnaire, all the electrodes were removed and participants were given an opportunity to view their data and ask questions.

2.3.4. Questionnaires—The coping strategies questionnaire (CSQ) is a widely used measure of pain coping strategies that can be completed by respondents with and without current pain (Rosenstiel and Keefe, 1983; Lefebvre et al., 1995). Although participants in the present study completed the full CSQ, the findings reported in this manuscript focus exclusively on responses to the catastrophizing subscale. The catastrophizing subscale is composed of six items that assess negative thoughts related to pain as well as catastrophic thoughts and ideations about pain. The CSQ catastrophizing subscale has demonstrated good internal reliability (Rosenstiel and Keefe, 1983) and a high degree of stability over time (Keefe et al., 1989). A recent study has also shown that the catastrophizing subscale is the most powerful predictor of pain severity among the CSQ subscales (Tan et al., 2001).

The short-form McGill pain questionaire (SF-MPQ) allows quantitative, multidimensional pain ratings to be obtained in a brief period of time (Melzack, 1987). In this study, participants completed the pain rating index of the SF-MPQ. Specifically, respondents rated 15 pain descriptors on a scale from 0 (none) to 3 (severe) and a sum of all rankings was used to compute a total pain rating index score. The SF-MPQ is a reliable and valid instrument commonly employed in clinical and research applications (Melzack, 1987).

3. Results

3.1. Data analysis

Hierarchical regression analyses were conducted to examine the relationship between catastrophizing scores and dependent measures obtained during (1) NFR threshold assessment and (2) electrocutaneous pain threshold and tolerance assessment. For each analysis, sex (male, female) and the drug administered (placebo, naltrexone) were forced into the equation on step 1, and then catastrophizing score was forced into the equation on step 2. Table 1 provides descriptive statistics for measures of catastrophizing, NFR threshold, and pain ratings obtained during both NFR threshold assessment and electrocutaneous pain threshold and tolerance assessment.

3.2. NFR threshold assessment

As can be seen in Table 2, results of the regression analyses revealed that neither sex, the drug administered, nor catastrophizing was significantly related to NFR threshold. Further, although neither sex nor drug was significantly related to pain ratings in response to NFR assessment, catastrophizing was positively related to both NFR pain rating scale scores (Beta = 0.45, $P < 0.001$) and SF-MPQ pain rating index scores (Beta = 0.49, $P < 0.001$). That is, higher catastrophizing was associated with higher pain ratings.

3.3. Electrocutaneous pain threshold and tolerance assessment

As can be seen in Table 3, results of the regression analyses revealed that neither sex nor drug administered was significantly related to electrocutaneous pain threshold; however, higher levels of pain catastrophizing were associated with lower pain thresholds (Beta = −0.26, $P < .05$). A similar pattern of findings was observed for SF-MPQ pain ratings in response to electrocutaneous threshold and tolerance assessment, with individuals who scored high on catastrophizing reporting greater pain (Beta = 0.45, $P < 0.001$). None of the predictor variables were significantly related to pain tolerance.

4. Discussion

The results of the present study confirm and extend existing evidence of the relationship between catastrophizing and pain reports. Specifically, a significant positive relationship was observed between catastrophizing and electrocutaneous pain as assessed using the pain-rating scale, the McGill pain questionnaire, and electrocutaneous pain threshold level. In contrast, catastrophizing was not significantly related to nociceptive flexion reflex threshold levels. Therefore, in the present context, catastrophizing appears to be associated with heightened experience and/or reports of pain without significant evidence of enhanced nociceptive transmission through spinal gating mechanisms. As pain is a multidimensional experience, in the presence of equivalent nociceptive input, catastrophizing may contribute to heightened pain experience by increasing attentional focus on pain and/or emotional reactivity to pain. For example, those who score high on measures of catastrophizing, may 1) perceive electrocutaneous pain as more threatening, 2) and are less able to distract themselves during NFR assessment, and 3) suffer greater anxiety as a consequence. Regardless of the mechanism of the relationship between catastrophizing and elevated pain ratings, this effect does not appear to rely on elevated nociceptive input.

It is important to emphasize that the current findings are based on an acute pain manipulation in a non-clinical sample. As a result, the possibility remains that catastrophizing may be related to differential modulation of nociceptive transmission in individuals who suffer from more intense and enduring clinical pain. To address this issue, we are currently examining the relationship between catastrophizing and NFR activity in men and women with osteoarthritis

pain. It is also important to note that our conclusion that catastrophizing may not influence nociceptive input is based on the failure to observe a significant relationship between catastrophizing scores and NFR threshold. Since a failure to reject the null hypothesis can never establish that such a relationship does not exist, our findings must be interpreted with appropriate caution.

Acknowledgements

This work was supported by a grant from the National Heart, Lung, and Blood Institute of the National Institutes of Health (NHLBI R01 HL64794).

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Table 1

Descriptive statistics for measures of catastrophizing, NFR threshold, and pain ratings obtained during NFR threshold and electrocutaneous pain threshold and tolerance assessments[a]

Variable	Mean	SD	Range
Catastrophizing (units)	5.8	4.9	0–21
NFR assessment			
NFR threshold (mA)	15.0	8.9	4–40
Pain rating scale (units)	48.1	21.3	4–100
Pain rating index of the SF-MPQ (units)	10.0	7.2	1–34
Pain threshold and tolerance assessment			
Electrocutaneous pain threshold (mA)	17.5	8.9	4–40
Electrocutaneous pain tolerance (mA)	31.3	8.8	6–40
Pain rating index of the SF-MPQ (units)	12.9	8.2	2–36

[a] Note: SF-MPQ, short form McGill pain questionnaire.

Table 2

Results of regression analyses conducted for NFR assessment[a]

Equation R^2	Predictor variable(s)	Beta	*t*-value	*P*
NFR threshold				
0.06	Sex	0.11	1.04	ns
	Drug	− 0.15	− 1.47	ns
	Catastrophizing	0.12	1.14	ns
Pain rating scale				
0.23	Sex	0.09	0.95	ns
	Drug	− 0.06	− 0.68	ns
	Catastrophizing	0.45	4.63	< 0.001
Pain rating index of the SF-MPQ				
0.28	Sex	0.07	0.74	ns
	Drug	− 0.13	− 1.43	ns
	Catastrophizing	0.49	5.19	< 0.001

[a] Predictor variables include sex (male = 0, female = 1), drug (naltrexone = 0, placebo = 1), and pain catastrophizing score.

Table 3

Results of regression analyses conducted for the electrocutaneous pain threshold and tolerance assessment[a]

Equation R^2	Predictor variable(s)	Beta	*t*-value	*P*
Electrocutaneous pain threshold				
0.08	Sex	0.00	0.03	ns
	Drug	−0.12	−1.19	ns
	Catastrophizing	−0.26	−2.50	< 0.05
Electrocutaneous pain tolerance				
0.08	Sex	−0.20	−1.87	ns
	Drug	−0.02	−0.19	ns
	Catastrophizing	−0.19	−1.80	ns
Pain rating index of the short form of MPQ				
0.21	Sex	−0.13	−1.34	ns
	Drug	−0.09	−0.92	ns
	Catastrophizing	0.45	4.63	< 0.001

[a] Predictor variables include sex (male = 0, female = 1), drug (naltrexone = 0, placebo = 1), and pain catastrophizing score.