Catastrophizing as a mediator of sex differences in pain: differential effects for daily pain versus laboratory-induced pain

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Abstract

Sex differences in the experience of pain have been widely reported, with females generally reporting more frequent clinical pain and demonstrating greater pain sensitivity. However, the mechanisms underpinning such differences, while subject to intense speculation, are not well-characterized. Catastrophizing is a cognitive and affective process that relates strongly to enhanced reports of pain and that varies as a function of sex. It is thus a prime candidate to explain sex differences; indeed, several prior studies offer evidence that controlling for catastrophizing eliminates the gap between men and women in reported pain. We recruited 198 healthy young adults (115 female) who took part in laboratory studies of pain responses, including thermal pain, cold pain, and ischemic pain, and who also completed questionnaires assessing catastrophizing, mood, and day-to-day painful symptoms (e.g. headache, backache). Women reported greater levels of catastrophizing, more recent painful symptoms, and demonstrated lower pain thresholds and tolerances for noxious heat and cold relative to men. Mediational analyses suggested that after controlling for negative mood, catastrophizing mediated the sex difference in recent daily pain but did not mediate the much larger sex differences in pain threshold and tolerance. These findings highlight the role of catastrophizing in shaping pain responses, as well as illuminating potentially important differences between experimental pain assessment and the clinical experience of pain.

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Keywords: Pain; Threshold; Tolerance; Catastrophizing; Sex

1. Introduction

Sex differences have been reported for many pain-related responses. For instance, women are at greater risk for pain disorders such as fibromyalgia (Unruh, 1996). Women also report more widespread pain, more pain-related affective symptoms (Keefe et al., 2000; Mullersdorf and Soderback, 2000), and more frequent daily pain (Berkley and Holdcroft, 1999; Barsky et al., 2001; Bassols et al., 2002). Sex differences have also been investigated in laboratory settings, with women demonstrating lower pain thresholds and higher pain ratings across a variety of noxious stimuli (Fillingim, 2000; Riley et al., 1998). It has been suggested (Fillingim and Maixner, 1995; Fillingim et al., 1999a,b) that these findings are linked, with greater pain sensitivity acting as a risk factor for enhanced clinical pain.

Although sex differences are well-documented, explaining these differences is more challenging. While some researchers emphasize socialization (Fearon et al., 1996) or emotional responsiveness (Riley et al., 2001) as potential mechanisms, others have highlighted biological factors (Berkley, 1997) or pain-coping (Fillingim, 2000; Myers et al., 2003; Unruh, 1996). Indeed, how one copes with pain consistently predicts important clinical outcomes, including pain severity and disability (Turk and Okifuji, 2002). Generally, the most robust predictor of pain outcomes is catastrophizing (Sullivan et al., 2001), defined as a negative emotional and cognitive response to pain involving elements of...
magnification, helplessness, and pessimism. Catastrophizing is positively correlated with pain and depression, and some laboratory studies show an association with responses to standardized noxious stimuli (Sullivan et al., 2001).

Additionally, women report more frequent catastrophic cognitions (Sullivan et al., 2001), making catastrophizing a potential contributor to sex differences in pain. In a recent osteoarthritis study, women had higher levels of pain, pain behavior, and disability. Moreover, women reported more catastrophizing, which mediated the relationship between sex and pain-related outcomes after controlling for depression (Keefe et al., 2000). A second study reported that during a cold pressor task, females reported more pain and displayed more pain behavior than males, effects which became non-significant when catastrophizing was controlled (Sullivan et al., 2000b).

The literature, however, is inconsistent, with some studies showing no sex differences in catastrophizing (Edwards et al., 2000; Unruh et al., 1999). Moreover, while pain-related sex differences in the laboratory are often large (Fillingim, 2000; Riley et al., 1998), sex differences in clinical pain are inconsistent (Turk and Okifuji, 1999). Additionally, catastrophizing as a mediator of sex differences in day-to-day pain among healthy individuals has not been studied. It is important to investigate such questions in non-clinical samples, before sex differences in pain are confounded by additional factors such as sex differences in pain treatment or sex-specific selection biases. Many researchers have also not controlled for depression when evaluating catastrophizing, which is now a standard in the field (Sullivan et al., 2001). Finally, catastrophizing likely depends on contextual factors such as the threat value of pain, which may differ in laboratory settings versus clinical environments. The present investigation, therefore, studied catastrophizing as a mediator of sex differences in both day-to-day pain and experimental pain.

2. Methods

2.1. Subjects

Participants were 198 (115 female, 83 male) individuals recruited from a University’s local community. Most (78%) were undergraduates who participated in the experiment for course credit, while the remainder (22%) consisted of healthy volunteers recruited from the community who received financial compensation for participating. Subjects were recruited using posted advertisements in a University Psychology Department. Subjects were excluded if they reported any major medical conditions or significant health risks (e.g., cardiac abnormalities, hypertension, diabetes, asthma, etc.), current pregnancy, current pain treatment, or current use of prescription analgesics, tranquilizers, antidepressants, or other centrally acting agents. The present investigation combines data from subjects who participated in several studies (e.g. Edwards and Fillingim, 2001; Edwards et al., 2003b); all participants were assessed for thermal pain responses, and some also underwent cold and/or ischemic pain tasks (see below). All procedures were approved by the University’s Institutional Review Board.

2.2. Questionnaires

Coping Strategies Questionnaire (CSQ). The CSQ is the most commonly used instrument to assess coping techniques employed by individuals with chronic pain (Rosenstiel and Keefe, 1983). It is a 48-item self-report measure that assesses cognitive (Catastrophizing, Diverting Attention, Coping Self Statements, etc.) and behavioral coping techniques. Each domain is comprised of six items and participants rate the frequency of their use of specific coping strategies on a seven-point Likert scale from zero, ‘never do that’ to six, ‘always do that.’ The CSQ’s catastrophizing subscale has been well-validated and is among the most widely used measures of catastrophizing (Sullivan et al., 2001). It assesses primarily the trait-like aspects of catastrophizing and is comprised chiefly of items tapping the dimension of helplessness in response to pain (Sullivan et al., 2001).

General Health Questionnaire (GHQ). The GHQ has been used in recent studies of pain in the general population (Fillingim et al., 1999b; Lester et al., 1994). It assesses recent pain experiences including: types of pain, number and location of pain symptoms, impact of pain, and pain severity. Participants indicated whether they experienced any of the following types of pain within the past month: headache, back pain, muscle pain, joint pain, stomach pain, dental pain and premenstrual or menstrual pain. As has been done previously, in order to avoid any over-representation of gender-related pain, premenstrual and menstrual pain were not included in the overall rating of pain experiences in this study (Fillingim et al., 1999a). For each type of pain reported, the participant indicated the number of episodes in the past month and the average severity, from 0 (no pain) to 100 (worst pain imaginable). The following variables were utilized as an index of pain experienced over the past month: Pain Sites, Number of Pain Episodes, Average Pain Severity, and a rating from 1 (‘much less than average compared to other people your age’) to 7 (‘much more than average compared to other people your age’) of the bothersomeness of aches and pains over the past month.

Profile of Mood States (POMS). The POMS (McNair et al., 1992) consists of 72 mood-related words; subjects indicate the extent to which each item describes their current mood. It has been validated against other measures of mood and is sensitive to subtle differences in affective state. The POMS measures six mood states; the subscale used in the present study was the elated-depressed scale, which has demonstrated good
psychometric properties and sensitivity in studies of stress (Sanders and Bruce, 1999).

2.3. Procedures

Upon arrival at the laboratory, subjects provided written and verbal informed consent and then completed the study questionnaires (see above). Next, all subjects underwent thermal testing procedures.

**Thermal Testing Procedures.** Thermal stimuli were delivered using a Medoc thermal sensory analyzer (TSA-2001, Ramat Yishai, Israel) as in previous studies (Edwards and Fillingim, 1999). The \(30 \times 30\) mm contact probe was applied to the left volar forearm, and affixed in place with a Velcro strap. An ascending method of limits paradigm was applied to the left volar forearm, and affixed in place with a Velcro strap. An ascending method of limits paradigm was used, with a 0.5 \({}^\circ\)C per second rate of rise, a 32 \({}^\circ\)C adapting temperature, and a maximum temperature of 52 \({}^\circ\)C. Between trials, the thermode was shifted to a different position on the ventral forearm in order to avoid sensitization or habituation. All procedures were conducted with subjects seated comfortably in a recliner. Four trials of thermal pain threshold (TPTH) were followed by four trials of thermal pain tolerance (TPTO). For trials of TPTH, subjects were instructed to press a button when the thermal stimulus first became painful, for trials of TPTO, when the pain became intolerable.

A subset of participants also underwent either an ischemic pain task (Edwards and Fillingim, 2001), a cold pressor task (Geisser et al., 1992), or both tasks. For subjects who underwent multiple pain-induction modalities, thermal pain was always assessed first, ischemic pain assessed last, and each task was separated by at least 10 min.

**Ischemic Pain Task.** For the ischemic pain task, a subject’s right arm was occluded with a standard blood pressure cuff positioned proximal to the elbow and inflated to 240 mmHg using a Hokanson E20 Rapid Cuff Inflator. The subject then performed 20 handgrip exercises of 2-s duration at 4-s intervals at 50% of maximum grip strength. Cuff inflation was maintained until the perceived pain became intolerable; the procedure was terminated by the experimenter if pain tolerance had not been achieved at 15 min following initiation of handgrip exercises. Time to ischemic pain threshold (IPTH) and time to ischemic pain tolerance (IPTO), both measured in second, were recorded.

**Cold Pressor Task.** For the cold pressor task, subjects immersed their right hands in a circulating water bath (Neslab, RTE-111, Portsmouth, NH) maintained at 5 \({}^\circ\)C. The standardized instructions for the procedure directed participants to keep their hands in the water for as long as possible but explained that if the sensations became intolerable, participants could remove their hands at any time. Each immersion lasted for a maximum of 4 min, though participants were not informed of this time limit. The time to cold pain threshold (CPTH) and tolerance (CPTO) were recorded in second.

2.4. Data reduction and analysis

Descriptive statistics are reported as means ± SD unless otherwise indicated. The significance of univariate between-group differences was determined by ANCOVA (i.e. ethnicity was utilized as a covariate) in the case of continuous variables, or \(\chi^2\) in the case of categorical variables, with significance set at \(\alpha = 0.05\). Because we used multiple variables to represent the constructs of clinical pain (i.e. pain sites, pain episodes, pain severity, bothersomeness of pain) and experimental pain (i.e. pain threshold, pain tolerance), MANOVA was used to examine associations between sex and pain (both clinical and experimental) before and after controlling for catastrophizing.

3. Results

3.1. Sex differences

Men and women in the present sample did not differ in age or educational background (see Table 1). However, more men reported their ethnic backgrounds as ‘white’ and all subsequent analyses of sex differences, therefore, control for ethnicity.

Women reported more pain sites, pain bothersomeness, and marginally more pain episodes relative to men (see Table 1). Several sex differences in pain coping emerged, with women noting significantly greater use of catastrophizing and of praying/hoping. No sex differences in POMS depressive symptoms were observed.

Sex differences were observed for some experimental pain responses. In analyses of responses to noxious thermal stimuli, TPTH and TPTO were significantly higher in men relative to women (\(P < 0.01\); see Table 1). In addition to the thermal pain procedures, 72.3% of men in the sample and 71.3% of women in the sample underwent the ischemic pain task, while cold pressor data were less common, with 31.3% of the men and 27.0% of women providing cold pressor data. No sex effects were observed for ischemic pain; although IPTO and IPTH values were slightly higher for men, this difference did not approach statistical significance (see Table 1). In contrast, even with a reduced number of participants, sex differences in cold pain were significant, with men demonstrating higher CPTH and CPTO than women (\(P < 0.05\); see Table 1).

3.2. Mediation of sex differences in recent pain experiences

After controlling for ethnicity and POMS depression subscale scores, sex showed a multivariate association with measures of day-to-day pain (\(P = 0.03\)). When this analysis was repeated, including catastrophizing as a predictor variable, catastrophizing showed a significant multivariate association with recent pain (\(P < 0.001\)), while the sex effect became non-significant (\(P > 0.1\)). See Table 2.
Because men and women differ in ethnic group representation, "G Cold pain tolerance (s) 59.5" and "G Cold pain threshold (s) 13.0" variables "n Study variables Men (n = 83) Women (n = 115) Age (years) 21.3±4.3 21.5±3.4 Ethnicity (% white) 58/83 (69.9%)* 63/114 (55.3%) Education (% current undergraduate) 69/83 (83.1%) 85/115 (72.2%) Thermal pain variables Warmth threshold 34.7±1.4* 34.1±1.2 Heat pain threshold 43.7±4.0* 42.0±3.7 Heat pain tolerance 49.2±2.3* 46.4±3.2 Clinical pain over the past 30 days Number of pain sites 1.2±1.0 1.7±1.3* Number of pain episodes 5.0±10.0 8.5±15.9Ψ Bothersomeness of pain (0–7) 3.0±1.3 3.5±1.3* Mean pain severity (0–100) 27.2±24.2 31.5±22.9 Coping strategies questionnaire subscales Diverting attention 2.1±1.3 2.1±1.3 Reinterpreting pain sensations 1.2±1.1 1.0±1.1 Coping self-statements 3.6±1.3 3.5±1.3 Ignoring pain 3.0±1.4 2.6±1.5Ψ Increasing behavioral activity 1.9±1.2 2.2±1.1Ψ Catastrophizing 0.8±1.1 1.2±1.0Ψ Praying/hoping 1.7±1.3 2.2±1.3* Ability to control pain 2.3±1.6 2.3±1.5 Ability to decrease pain 2.0±1.3 2.1±1.5 Recent mood POMS depression 23.4±5.9 24.1±4.8 Study variables Men (n = 60) Women (n = 82) Additional experimental pain variables Ischemic pain threshold (s) 148.2±159.4 132.0±120.1 Ischemic pain tolerance (s) 481.6±236.5 408.4±268.3 Study variables Men (n = 26) Women (n = 31) Additional experimental pain variables Cold pain threshold (s) 13.0±8.2* 7.3±3.0 Cold pain tolerance (s) 59.5±69.6* 24.0±40.8 *P<0.05 for the test of sex differences; ΨP<0.10 for the test of sex differences. Because men and women differ in ethnic group representation, all between-group analyses of pain, coping, and mood control for ethnicity. An ANCOVA indicated that sex remained associated with catastrophizing after controlling for ethnicity, POMS depression scores, and recent pain (P = 0.02), supporting a mediational model.

### 3.3. Mediation of sex differences in experimental pain

Simple correlations between catastrophizing and pain responses were calculated for measures of pain threshold and tolerance. Catastrophizing was significantly inversely related to TPTH (r = −0.16, P = 0.04) and TPTO (r = −0.18, P = 0.01), but showed no significant correlations with IPTH (r = 0.06, NS), IPTO (r = 0.07, NS), CPTH (r = 0.01, NS), or CPTO (r = −0.12, NS). The potential mediating influence of catastrophizing on sex differences in pain was examined separately for each modality of experimental noxious stimulation.

### Thermal Pain

After controlling for ethnicity and POMS depression subscale scores, sex showed a multivariate association with thermal pain responses (i.e. pain threshold and tolerance; P < 0.001). Interestingly, and consistent with previous data, ethnicity was also associated with thermal pain sensitivity, with African–American participants demonstrating lower HPTH and HPTO relative to white participants. When this analysis was repeated, including catastrophizing as a predictor variable, catastrophizing failed to show a significant multivariate association with experimental pain (P > 0.3), and the sex effect remained highly significant (P < 0.001), suggesting that catastrophizing did not mediate sex differences in HPTH and HPTO (see Table 3).

### Cold Pain

After controlling for ethnicity and POMS depression subscale scores, sex showed a multivariate association with cold pain responses (i.e. pain threshold and tolerance; P = 0.002). When this analysis was repeated with catastrophizing included, catastrophizing failed to show a significant multivariate association with cold pain responses (P > 0.2), and the sex effect remained highly significant (P = 0.001), suggesting no mediational effects for CPTH and CPTO (see Table 4).

### Table 3

<table>
<thead>
<tr>
<th>Study variables</th>
<th>n (males)</th>
<th>n (females)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dependent variables: number of pain symptoms, number of pain sites, average pain severity, and bothersomeness of pain (over the past 30 days).</td>
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<td></td>
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### Table 4

<table>
<thead>
<tr>
<th>Factor</th>
<th>Multivariate F</th>
<th>P-value</th>
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<tr>
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<tr>
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<td>0.94</td>
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<tr>
<td>Sex</td>
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<tr>
<td>Analysis 2 (controlling for catastrophizing)</td>
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<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>912.2</td>
<td>&lt;0.001</td>
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<tr>
<td>Ethnicity</td>
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<tr>
<td>POMS depression</td>
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<tr>
<td>Catastrophizing</td>
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<td>0.33</td>
</tr>
<tr>
<td>Sex</td>
<td>23.0</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Dependent variables: thermal pain threshold, thermal pain tolerance.
sex differences in cold pain tolerance (Sanford et al., 2002). The association between threat appraisals and cold pain tolerance was only marginally significant ($r = -0.15$); however, controlling for threat appraisals did slightly reduce the significance of sex difference in cold pressor tolerance times. The small magnitude of this association parallels our findings and suggests that catastrophizing’s relationship with experimental pain responses may be contingent on the type of response measured, with ratings of pain intensity and observation of pain behaviors (see Keefe et al., 2000), being most subject to influence by cognitive and affective processes.

We should also note that assessment of catastrophizing should include the additional dimensions of rumination and magnification, as measured by the pain catastrophizing scale (PCS) (Sullivan et al., 1995). In one prior study, only the rumination subscale of the PCS predicted pain ratings during dental hygiene treatment (Sullivan and Neish, 1998); future studies may benefit from examining the multiple dimensions of catastrophizing. It may be prudent, at this point, to recommend use of the PCS rather than the CSQ subscale in experimental pain studies; in contrast, the CSQ subscale appears to be a consistent correlate of pain outside of the laboratory, perhaps confirming that helplessness, or low self-efficacy, is among the most important psychosocial factors to assess in the context of clinical pain (Keefe et al., 2001; Turk and Okifuji, 2002). It may also be important to consider that the time frames for assessing day-to-day and laboratory pain are rather different. In the former case, individuals recall experiences over the past month, while during experimental pain testing, subjects respond to pain as it is being experienced. Pain recall is subject to many biases (Haythornthwaite and Fauerbach, 2001; Stone et al., 2004), and it may be that catastrophizing is more powerful in shaping pain recall than in determining in vivo pain responses. Indeed, in the present study, the CSQ was completed shortly after participants answered questions about recent daily pain, and their responses to questions about catastrophizing may have been influenced by those recently-recalled pain experiences.

The process of catastrophizing is closely tied to the meaning of pain (Sullivan et al., 2001); it seems probable that catastrophic interpretations are more likely to accompany daily pain relative to a brief, controllable stimulus administered in a laboratory, where subjects have foreknowledge about the stimuli (Gracely, 1999). In contrast, clinical pain is more unpredictable and threatening. For example, a study of cancer patients found that those who believed that physical therapy-induced pain was cancer-related showed higher ratings of pain intensity and unpleasantness compared to those who attributed their pain to other factors, highlighting the role of meaning and interpretation (Smith et al., 1998). Another investigation noted that pain intensity ratings are strongly affected by interpretations of stimuli as more or less tissue-damaging, a consideration that would rarely apply to most laboratory

### Table 4

Association of sex with cold pain responses after controlling for potential confounders and catastrophizing

<table>
<thead>
<tr>
<th>Factor</th>
<th>Multivariate F</th>
<th>P-value</th>
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<td><strong>Analysis 1</strong></td>
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<td>Sex</td>
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<tr>
<td><strong>Analysis 2 (controlling for catastrophizing)</strong></td>
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</tr>
<tr>
<td>POMS depression</td>
<td>1.4</td>
<td>0.25</td>
</tr>
<tr>
<td>Catastrophizing</td>
<td>1.4</td>
<td>0.25</td>
</tr>
<tr>
<td>Sex</td>
<td>7.6</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Dependent variables: cold pain threshold, cold pain tolerance.

**Ischemic Pain.** Meditational analyses were not possible for data on ischemic pain responses as no sex differences in IPTH or IPTO were observed.

### 4. Discussion

Numerous explanations have been proposed to account for sex differences in pain. While many such explanations are biological, it is becoming increasingly clear that multiple factors explain sex differences in pain experiences, including important social and psychological factors. Our results indicate that sex differences in complaints of painful day-to-day symptoms are accounted for by the significant differences between men and women in reports of catastrophizing. However, the sex differences in catastrophizing do not account for the substantially higher threshold and tolerance for thermal and cold pain observed among men.

The present study builds upon prior work by assessing catastrophizing as a mediator of male–female differences in pain responses both inside and outside of the laboratory. Several prior studies have suggested that catastrophizing at least partially mediates observed sex differences in both clinical pain (i.e. arthritis pain) (Keefe et al., 2000) and in experimental pain responses (Sullivan et al., 2000a,b). Our results are consistent with those of the former study, and suggest that catastrophizing plays an influential role in shaping sex differences in pain report in both clinical and non-clinical samples. However, it is somewhat more difficult to reconcile our findings with those of Sullivan and colleagues (Sullivan et al., 2000b), who also used a cold pressor task. They assessed pain intensity ratings and a measure of the duration of pain behaviors during the task. Correlations of between 0.33 and 0.53 were observed for the association between catastrophizing scores and pain responses. In contrast, we observed minimal correlations ($r = -0.12$ for CPTO and catastrophizing). One other recent cold pressor study evaluated threat appraisals (i.e. conceptually similar to catastrophizing) as a potential mediator of sex differences in cold pain tolerance (Sanford et al., 2002).
settings (Arntz and Claassens, 2004). Finally, a study of fibromyalgia patients reported strong correlations between catastrophizing and clinical pain but no associations with responses to noxious mechanical stimuli administered in a laboratory (Gracely et al., 2004).

Sex-related variation in catastrophizing appears to emerge relatively early in development, probably well before most individuals have had any substantive experience with chronic pain. Catastrophizing is more common among adolescent school girls than boys, and was associated with more pain and pain medication use in a survey of high school students (Bedard et al., 1997). Indeed, this sample consists of healthy college-age students with no history of chronic pain. Unfortunately, while it suggests some consequences of sex differences in catastrophizing, the present study can offer little insight into its causes; such information will require longitudinal studies with long follow-up periods in children and adolescents.

It is interesting to speculate that the findings of sex differences in catastrophizing may play a role in shaping sex differences in responses to pain treatments. Cognitive-behavioral therapy (CBT) for pain focuses on the modification of catastrophic pain-related cognitions (Pincus et al., 2002), and a recent treatment process study suggested that changes in catastrophizing paralleled changes in mood over the course of pain treatment (Jensen et al., 2001b). Furthermore, early changes in catastrophizing during multidisciplinary pain treatment contribute to later reductions in pain severity (Burns et al., 2003a,b). As some recent studies find that women show better multidisciplinary treatment outcomes than men (Edwards et al., 2003a; Jensen et al., 2001a), one might conjecture that higher initial levels of catastrophizing among women are more amenable to reduction by CBT, thus producing greater treatment-associated improvements in pain-related sequelae. Future treatment studies may benefit from the separate analysis of process variables such as catastrophizing in men and women.

Multiple limitations of the present investigation require consideration. First, only two experimental pain responses, pain threshold and pain tolerance, were studied; results may differ for other types of laboratory pain assessment, particularly if the cognitive-emotional aspects of catastrophizing are more likely to be reflected in responses such as nonverbal pain behaviors. Second, we used a unidimensional measure of catastrophizing; in future studies, the use of a multi-component measure such as the PCS may provide more complete information. Third, reports of recent daily pain were retrospective and most of these healthy subjects reported low levels of pain; it is likely that the mediating effects of catastrophizing are even more robust under the circumstances of more substantial clinical pain. In particular, since catastrophizing scores tend to be higher and more variable in clinical pain populations (Sullivan et al., 2001), the associations between catastrophizing and pain are likely to be stronger than in this non-clinical sample. Indeed, this consideration makes the present findings all the more striking, and suggests the potentially fundamental role of cognitive and affective factors in shaping sex differences in pain report. The lack of association between catastrophizing and experimental pain responses might also be explained by the low average levels of catastrophizing in this relatively healthy sample. Finally, future work should focus on identifying the mechanisms underlying sex differences in pain catastrophizing, as we are unable to shed light on the manner in which the sexes develop differential pain responses.

While sex differences in laboratory pain responses are generally robust, catastrophizing does not appear to play a role in producing these male–female discrepancies. Laboratory pain responses are related to the brain’s response to noxious stimulation (Coghill et al., 2003), to quality of life (Edwards et al., 2003c), and can prospectively predict clinical pain (Granot et al., 2003). Illuminating the mechanisms producing sex differences in experimental pain responses may therefore have important clinical implications. The fact that catastrophizing did mediate sex differences in day-to-day pain supports prior work in osteoarthritis patients. Importantly, catastrophizing mediated this association even after controlling for negative mood, highlighting catastrophizing’s unique effects. Catastrophizing is observed in community residents who report no current pain (Buer and Linton, 2002), is fairly stable across time (Sullivan et al., 2001), and is a risk factor for worsening pain (Haythornwaite et al., 2003; Keefe et al., 1989). As such, it certainly warrants further attention as a marker for individuals at high risk for the onset or worsening of pain, as a target of treatment, and as an important variable in shaping group differences in the clinical experience of pain.

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