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Individual and Gender Differences in Subjective and Objective Indices of Pain: Gender, Fear of Pain, Pain Catastrophizing and Cardiovascular Reactivity

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Abstract According to fear-avoidance models of pain perception, heightened fear of pain may increase disruptive effects of pain; however, the extent to which this affects self-reported pain severity versus physiological indices of pain is not well delineated. The current study examined self-report measures and physiological indices of pain during a cold pressor (CP) task. Individual differences in fear of pain and pain catastrophizing were also assessed via questionnaire. The primary aim of the current study was to examine the extent to which individual differences associated with fear and catastrophizing in response to pain influences subjective and physiological measures of pain. A secondary aim was to examine gender differences associated with response to pain. Average subjective pain ratings were higher for females than males. In contrast, males exhibited higher systolic and diastolic reactivity in response to the CP task relative to females, as well as failure to fully recover to baseline levels. Follow-up correlational analyses revealed that subjective pain ratings were positively associated with fear of pain in both sexes, but were not associated with cardiovascular indices. These results suggest that fear of pain and pain catastrophizing do not influence cardiovascular responses to induced pain. Further research is necessary in order to determine whether these gender differences in blood pressure and heart rate response profiles are due to biological or psychosocial influences. Results support the notion that fear of pain increases subjective pain ratings, but does not influence cardiovascular responses during CP pain-induction.

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Introduction

Individual differences in response to pain may have important consequences for such outcomes as the development of chronic pain, over-reliance on pain medication, as well as decreased psychosocial functioning. Numerous studies have reported that psychosocial factors are among the most important predictors of the development of chronic pain and pain-related disability (e.g., Crook et al. 2002; Linton 2000; Gatchel et al. 1995; Turk and Okifuji 2002), and of the likelihood of return to work in injured workers.

Accordingly, it is important to better understand those psychological factors that may underlie differences in pain complaints and responses to pain. Current biopsychosocial models incorporate the role of psychological and social factors in addition to biological factors in explaining the experience of pain. Biopsychosocial models that delineate attentional and emotional processes associated with heightened pain intensity and pain-related distress have been proposed (e.g., Brown 2004; Gatchel and Turk 1996; Turk and Melzac 2001; Turk and Okifuji 2002). Both "fear of pain" (Lethem et al. 1983; Slade et al. 1983) and "pain catastrophizing" (e.g., Sullivan et al. 1995) have been hypothesized to influence responses to pain.

The fear-avoidance model of exaggerated pain perception (Lethem et al. 1983; Slade et al. 1983) proposes that following acute injury, individuals with greater fear of pain tend to avoid activities that may trigger pain, leading to decreased strength, decreased flexibility, and general physical de-conditioning. Such individuals begin to view

themselves as having reduced personal control and being more disabled, and this may lead to heightened pain intensity. This process may contribute to the transition from acute injury to chronic pain. In support of this model, fear of pain has been shown to predict self-reported disability in low back pain (Fritz et al. 2001; Klenerman et al. 1995; Sieben et al. 2002).

Vlaeyen et al. (1995; Vlaeyen and Linton 2000) refined the fear-avoidance model by proposing that pain catastrophizing contributes to the development of chronic pain via beliefs that one is unable to tolerate pain and that pain is unbearable, as well as rumination on negative outcomes associated with pain. Higher levels of pain catastrophizing were hypothesized to contribute to both chronic disability and heightened pain intensity. Pain catastrophizing has been associated with higher pain ratings and with greater emotional distress during laboratory-induced pain (Sullivan et al. 1995), and with increased adverse pain-related outcomes including pain severity and pain-related disability (Quartana et al. 2009). Pain catastrophizing includes the magnification of pain, feelings of helplessness when in pain, and an inability to inhibit pain-related thoughts prior to, during, or following pain (Sullivan et al. 1995). The role of pain catastrophizing in chronic pain is supported by findings that the persistence of acute lower back pain to continued pain 6 months later was predicted by higher levels of pain catastrophizing in the acute phase (Picavet et al. 2002).

Both fear of pain and pain catastrophizing have been linked to the development of chronic pain and/or disability. Levels of fear of pain (Sieben et al. 2002) and pain catastrophizing (Burton et al. 1995) at the acute stage predicted self-reported disability across varying time spans up to a year later. Fear of pain has been reported by some to be better than self-reported pain ratings as a predictor of disability (e.g., Crombez et al. 1999), leading to the proposal that pain-related fear and avoidance play a substantial role in the progression toward chronic pain-related disability (Vlaeyen and Linton 2000).

Cardiovascular Reactivity to Pain, Fear, and Anxiety

In addition to self-reported pain severity measures, a number of studies have described physiological responses to pain, including increases in blood pressure (BP) and heart rate (HR) (e.g., Peckerman et al. 1994), caused by increased sympathetic nervous system activation (Norton and Asmundson 2003). While there is variability in the nature of cardiovascular changes depending on method of pain induction [e.g., pain via CP led to an immediate increase in heart rate during foot immersion but not during CP with forehead placement (Peckerman et al. 1994)] as well as other variations in cardiovascular response patterns to pain (Dixon et al. 2004), multiple studies have reported that blood pressure and heart rate tend to increase in response to induced pain (e.g., Lovallo 1975; Janig 1985). At the same time, a study of patients presenting to an emergency department with objectively verified diagnoses of painful conditions reported no significant associations between self-reported pain scores and BP or HR (Marco et al. 2006). In addition, both fear and anxiety have been repeatedly associated with increases in heart rate and blood pressure. Fear has been differentiated from anxiety in that it is viewed as a response to an immediate threat, whereas anxiety is typically viewed as a negative emotional state associated with anticipation of a future or impending threat (Barlow 2002; Bouton et al. 2001). However, both states involve cardiovascular reactivity, and it is not clear that researchers reporting on the cardiovascular effects of fear versus anxiety have explicitly distinguished each from the other. Accordingly, for the purposes of the current study it is assumed that (a) both "fear of pain" and "pain catastrophizing" involve some degree of increased fear or anxiety, and that (b) both increased fear and anxiety are associated with increased cardiovascular reactivity.

It may be reasonably predicted that individuals with increased tendency toward fear of pain or pain catastrophizing would experience more fear/anxiety with respect to pain and consequently demonstrate greater sympathetic activation in response to threatened or actual pain, with corresponding increases in BP and HR (Asmundson and Taylor 1996). This increased sympathetic activation associated with fear/anxiety may have additive effects above and beyond the cardiovascular reactivity attributable to pain itself. If so, greater cardiovascular reactivity on exposure to induced pain would be expected in those with greater pain-related fear/anxiety.

However, research to date does not indicate a simple relationship between higher levels of fear of pain or pain catastrophizing and pain-induced physiological reactivity. For example, some studies show increases in both HR and BP (Dixon et al. 2004), while others show increased BP for men only, and increased HR for women only (Maixner and Humphrey 1993).

George et al. (2006) used CP to induce acute pain in healthy volunteers, and found that fear of pain and pain catastrophizing measures taken before CP were associated with greater pain intensity; however, regression analyses showed that only fear of pain was a significant predictor of pain intensity at both the onset of pain and at the limit of the participant's tolerance for CP. They also found that neither fear of pain nor pain catastrophizing predicted systolic blood pressure reactivity, nor did gender.

George et al. (2006) speculated that their measure of pain catastrophizing, a Catastrophizing subscale from the coping strategies questionnaire (CSQ; Rosenstiel and

Keefe 1983; Keefe et al. 1989) may have been a poor choice for the study, and suggested that future studies should consider using the pain catastrophizing scale (PCS; Sullivan et al. 1995). However, a subsequent study using both the PCS and CSQ (Hirsh et al. 2008) found that after controlling for gender, only fear of pain was a predictor of pain threshold, tolerance and intensity. However, cardiovascular variables were not measured in this study, and the question of whether pain catastrophizing moderates cardiovascular pain responses during CP-induced pain remains unresolved.

The primary aim of the current study was to examine the relationships among fear of pain/pain catastrophizing, self-reported pain intensity, and physiological measures (HR and BP). Participants completed the PCS (Sullivan et al. 1995) as a measure of pain catastrophizing. They also completed the Fear of Pain questionnaire, third edition (FPQ-III; McNeil and Rainwater 1998). Pain ratings were taken before, during and after CP, and diastolic BP, systolic BP, and HR were measured before, during, and following termination of CP procedure. This allowed for examination of associations among self-reported pain, pain catastrophizing, fear of pain, and physiological measures. It was hypothesized that higher FPQ and PCS scores would be associated with greater BP and HR reactivity and with greater self-reported pain intensity.

Materials and Methods

Participants

Participants were undergraduates from Texas State University who either received a payment of ten dollars or extra credit in one of their psychology courses. Prospective participants were excluded if they had a history of circulatory problems (e.g., diabetes or Reynaud's Syndrome), elevated blood pressure, or if they were currently taking medications affecting pain (e.g., analgesics) or autonomic nervous system function (e.g., cold medications or decongestants). A total of 65 subjects participated in the study. Seven participants were excluded due to calibration problems with the BP monitor leaving 58 normotensive individuals (23 males, 34 females, mean age = 22.3 years). Of these individuals, 20 failed to complete the cold-pressor (CP) task (7 males, 13 females). Details of the CP task are provided below in the Procedures section. Of those who failed to complete the CP task, all withdrew their hands from the ice water bath within 30 s and thus did not provide subjective pain ratings for the task (the first rating was to be taken 60 s after immersion) or sufficient data for calculating stable estimates of BP changes during the CP task. However, their demographic and questionnaire data were retained for further analyses. The remaining participants (n = 38; 17 males, 21 females; mean age = 21.7 years) completed all 5 min of the CP task. The pattern of attrition and completion is notably distinct from what is typically observed in CP research in that (a) such a high percentage withdrew from the study so quickly, and (b) that of those who continued, all persisted for a comparatively lengthy 5 min. There is no obvious explanation for the high percentage of early dropouts from the CP task. Although it is possible that local warming of the water may have reduced pain sufficiently for participants to persist for 5 min without withdrawing, this is viewed as unlikely because (a) temperature was maintained at 2 °C throughout the procedure; and (b) pain ratings did not decrease across time, but remained stable across the entire CP task.

Procedures for human subjects were approved by the Institutional Review Board at Texas State University. Informed consent was obtained from each participant.

Self Report Measures

Subjective Pain Ratings

A 10-cm visual analog scale (VAS) was provided for participants to rate their current level of pain, which was assessed during the last minute of the baseline period, after each minute of the CP task, and after the first 2 min of the recovery period. The VAS is a psychometric scale along a continuous line which, in this study, measured subjective pain levels. The scale ranged from "0", or "none", indicating no pain, through "10", or "agonizing", indicating severe pain. Subjective pain ratings were obtained prior to beginning the CP task, once every minute during the CP, and twice (once every minute for the first 2 min) after completing the CP.

Fear of Pain Questionnaire-III (FPQ)

The fear of pain questionnaire-III (FPQ-III; McNeil and Rainwater 1998) was given to participants prior to the CP task. The FPQ-III is used to measure the fear or anxiety associated with pain. It is a 30-item scale which requires participants to respond to various statements about feelings and attitudes towards pain on a 5-point scale. The FPQ-III has demonstrated good internal consistency and test–retest reliability (McNeil and Rainwater 1998) as well as good construct, concurrent, and ecological validity (Husey and Jacks 1992). In the current sample, internal consistency measured by Cronbach's alpha was .96.

Pain Catastrophizing Scale (PCS)

The pain catastrophizing scale (PCS; Sullivan et al. 1995) is a 13-item scale that measures rumination, magnification,

and helplessness associated with pain using a 5-point Likert scale. Reported internal consistency for the three factors ranged from .60 to .87. The overall internal consistency (coefficient alpha) for the total PCS was .87. Gender differences have been reported, with women scoring significantly higher on average for rumination and helplessness, but with no significant differences for the magnification subscale (Sullivan et al. 1995). In the current sample, internal consistency measured by Cronbach's alpha was .93.

Physiological Measures

Cardiovascular measures included systolic blood pressure (SBP), diastolic blood pressure (DBP), and HR, which were measured continuously with a NIBP100A continuous blood pressure meter (Biopac; Goleta, CA). A pneumatic sensor was placed over the radial artery of the participant's non-dominant wrist, and secured with a Velcro strap. The raw data were digitized at a rate of 200 Hz and a gain of 1,000 via a Biopac MP150 SWS amplifier system connected to a Dell Optiplex G $\times$ 270 computer running Acknowledge v.3.8.1. Prior to analysis, raw data were scanned for artifact and manually cleaned using Acknowledge. Systolic BP, diastolic BP, and HR were then computed offline. Cardiovascular data were averaged into 5 min epochs corresponding to two 5-min baseline periods, one 5-min CP period, and two 5-min recovery periods. For all analyses, the second baseline measures were used as the baseline, while the second recovery measures were used as an estimate of recovery.

Procedures

Cold Pressor (CP) Task

The CP task involves immersing a limb in a container of ice water, and is a standard laboratory method of pain induction (Peckerman et al. 1994). In the current study, an 11-quart plastic bucket was filled approximately 2/3 full with ice cubes, then filled with water. Temperature was recorded to ensure that the water was at 2 °C prior to starting the cold pressor task. Temperature was recorded before, during, and following the experimental procedures, and remained at 2 °C during this period.

Before experimental procedures were conducted, subjects were informed of the study procedures and were aware of the CP task prior to obtaining consent. They were informed that the CP task would likely induce moderate pain. Per agreement with the Institutional Review Board, participants were informed that they could discontinue the CP task at any time, but were asked to continue with the CP task for as long as they were willing to persist, for up to 5 min. They then provided basic demographic information and completed the self-report questionnaires (FPQ and PCS). After the completion of the questionnaires, the BP sensor was applied and calibrated. The subjects then underwent a 10 min baseline period. The first 5 min were used to habituate participants to the testing environment, while the second 5 min of the baseline period were used to measure resting HR and BP. At the end of the baseline period, subjects were asked to rate their current level of pain using the VAS scale.

Prior to starting the CP task, a measuring tape was used on the participants' dominant arm to determine the length of the forearm (the distance in cm from the wrist to the elbow) and find the middle of the forearm, where a line was drawn to indicate how deeply to immerse their arms in the water. The CP task started upon the placement of the arm in the bucket of stirred ice water, which was maintained at 2 °C. Pain ratings using the VAS were assessed at 60 s intervals for 5 min. Participants were told that they could withdraw their arms at any time. After withdrawing their arms from the ice water at 300 s, the subjects immediately started a 10 min recovery period to assess physiological recovery from pain. Pain ratings were recorded during the first 2 min of this period at 60 s intervals. Thus, a total of eight pain ratings were recorded during the experiment.

Analytic Strategy

For participants who completed the CP task, planned comparisons were conducted to determine whether gender differences existed in the self report data. To this end, a mixed ANOVA was conducted on subjective pain ratings given over the course of the CP task, with rating ($n = 5$) as a within subjects variable and gender as a between subjects variable. To assess gender differences in self-report measures, two one-way ANOVAs with gender as a between subjects variable were conducted on FPQ and PCS scores. Gender differences in baseline cardiovascular data (systolic BP, diastolic BP, and HR) were examined with 3 one-way ANOVAs. Gender differences in cardiovascular data patterns across time (systolic BP, diastolic BP and HR) were examined with 3 mixed ANOVAs, with gender (male vs. female) as a between-subjects variable and time (baseline, CP, recovery) as a within subjects variable. Exploratory correlations were performed among subjective pain ratings, self-report variables, and cardiovascular measures (at baseline, during CP, and during recovery) to examine relationships among these variables.

Given that approximately one-third of the people who participated in the study did not complete even the first minute of the CP task, secondary analyses were also conducted to determine whether these non-completers differed

systematically from those who completed the CP. First, Chi square analyses were conducted to determine whether the gender balance differed between completers and non-completers. Next, two one-way ANOVAs (with completion status as a between subjects variable) were conducted to determine whether the two groups differed in terms of PCS and FPQ scores.

Results

Gender Differences in Self-report

Gender differences in subjective pain ratings over the course of the CP task are shown in Fig. 1. The ANOVA revealed a main effect of gender; $F(1, 37) = 4.88$, $p < .05$, indicating that overall, average pain ratings were higher for females relative to males (5.83 vs. 4.31, respectively). There was no main effect of time ($F < 2.0$), suggesting that pain ratings remained relatively stable over the course of the CP task. Similarly, no gender by time interaction was observed during the CP task ($F = 1.0$). Pain ratings after the CP were also assessed, yielding a time by gender interaction; $F(1, 37) = 4.04$, $p = .05$. Bonferroni-corrected post hoc comparisons revealed that both men and women reported decreases in subjective pain ratings from Time 1 to Time 2 of the recovery period, but the decrease in pain was larger for females (1.76 at Recovery 1 vs. 1.00 at Recovery 2 for males, 2.50 vs. .75 for females; $t(16) = 3.79$, $p = .002$, $t(21) = 4.43$, $p = .000$ for males and females, respectively).

For individuals who completed the CP, there were no differences between males and females in FPQ (67.5 for females vs. 57.8 for males) or PCS scores (22.0 for females versus 21.8 for males; F's < 1.5). When the data from non-completers was pooled with those who completed the CP and assessed for gender differences, no differences again were observed, in spite of increased statistical power.

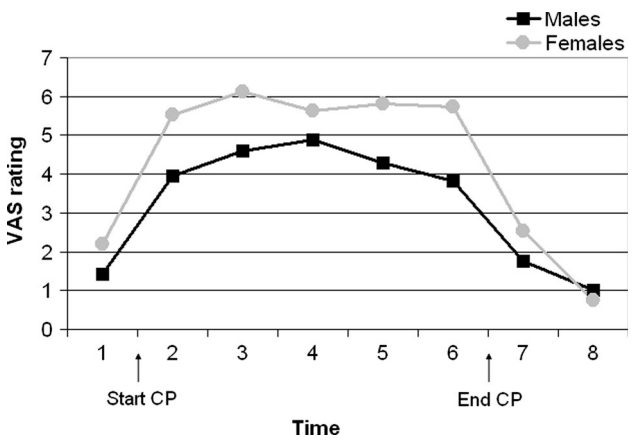

Fig. 1 Subjective pain ratings for males and females over the course of the experiment

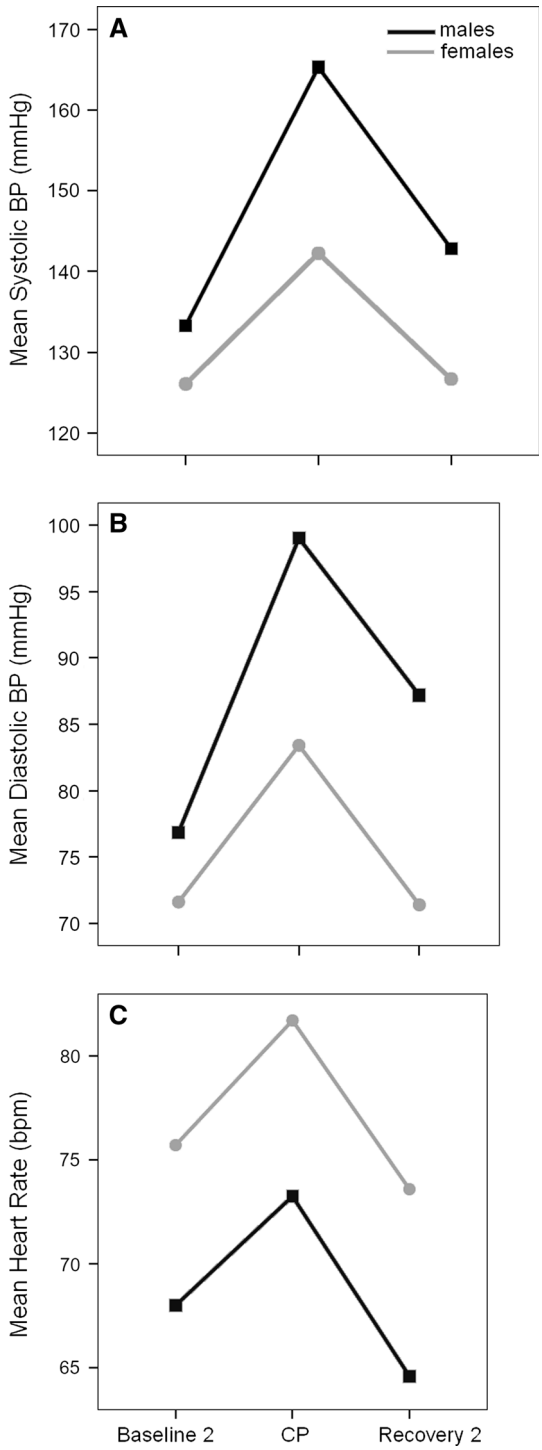

Fig. 2 Average values for **a** systolic BP, **b** diastolic BP and **c** heart rate over the course of the experiment

Cardiovascular Data

Gender differences for baseline cardiovascular data were observed for HR (68.0 for males vs. 75.3 for females; $F(1, 36) = 5.29$, $p = .027$), but were not statistically significant for either diastolic BP (76.9 for males vs. 71.6 for females) or for systolic BP (133.3 for males vs. 125.7 for females).

For systolic BP, the ANOVA revealed a main effect of gender; $F(1,35) = 7.86$, $p < .05$. As shown in Fig. 2a, males overall had higher systolic BP than females. There was also a main effect of time; $F(2,70) = 57.26$, $p < .001$, reflecting the increase in systolic BP during the CP task for both males and females, followed by a decrease over the course of the recovery period. These main effects were mitigated by a time x gender interaction; $F(2,70) = 5.57$, $p < .05$. Males exhibited higher systolic reactivity in response to the CP task relative to females, and the higher systolic BP did not fully return to baseline levels.

For diastolic BP, the ANOVA revealed a main effect of gender; $F(1,35) = 7.27$, $p < .05$, reflecting overall higher diastolic BP for males (see Fig. 2b). There was also a main effect of time; $F(2,70) = 26.15$, $p < .001$, reflecting the increase in diastolic BP during the CP task for both males and females, followed by a decrease over the course of the recovery period. There was also a marginal time × gender interaction; $F(2,70) = 3.13$, $p = .06$. As with systolic BP, males exhibited a marginally higher diastolic response to the CP task relative to females and did not fully recover to baseline levels.

For HR, the ANOVA revealed a main effect of gender; $F(1,35) = 9.12$, $p < .01$. As shown in Fig. 2c, females had higher heart rate over the course of the experiment relative to males. There was also a main effect of time; $F(2,70) = 27.96$, $p < .001$, reflecting the increase in heart rate during the CP task for both males and females, followed by a decrease over the course of the recovery period. Unlike blood pressure values, no time × gender interaction was observed; $F(2,70) = .16$, $p > .05$, indicating that HR response profiles for males and females were similar over time.

Correlational Analyses

Exploratory correlations were performed among subjective pain ratings, self-report variables, and cardiovascular measures at baseline, CP, and recovery. Pain ratings during CP task (n = 5, at one pain rating per minute) were averaged to create a mean CP pain rating. FPQ and PCS were correlated, $r = .589$, $p < .001$. Mean subjective pain ratings during the CP task correlated with FPQ in both sexes ($r = .51$, $p < .01$), and with PCS ($r = .403$, $p = .01$), but did not correlate with HR or BP indices at baseline, CP, or recovery. Neither FPQ nor PCS scores were correlated with HR or BP indices at baseline, CP, or recovery. See Table 1 for relevant correlations.

Table 1 Correlations among pain ratings, FPQ, PCS, and cardiovascular measures

Measure	1	2	3	4	5	6
1. Mean pain rating	–	.51**	.40*	.00	.00	.05
2. FPQ		–	.59**	.04	−.12	−.13
3. PCS			–	−.10	−.08	−.07
4. HR (pain)				–	−.06	−.03
5. sBP (pain)					–	−.96**
6. dBP (pain)						–

n = 38

FPQ fear of pain questionnaire, *PCS* pain catastrophizing scale, *HR (pain)* heart rate during induced pain, *sBP (pain)* systolic blood pressure during induced pain, *dBP (pain)* diastolic blood pressure during induced pain

* $p < .05$, ** $p < .01$

Completers Versus Non-completers

Chi square analyses revealed that the gender balance in the group that completed the CP was not significantly different from that of the group that did not complete the CP. ANOVAs (with completion status as a between subjects variable) conducted on PCS and FPQ scores indicated that completers and non-completers did not differ in terms of PCS and FPQ scores.

Discussion

According to the fear-avoidance model of exaggerated pain perception (Lethem et al. 1983; Slade et al. 1983), after experiencing acute pain, individuals with greater fear of pain are at increased risk to develop chronic pain due to interactions among fear, avoidance, and perceptions of reduced control and increased disability. The current study examined relationships among pain catastrophizing, fear of pain, subjective pain ratings, and physiological responses (BP and HR) during induced pain in healthy volunteers. It was hypothesized that greater fear of pain would contribute to higher pain ratings, as reported by George et al. (2006), as well as greater increases in blood pressure and heart rate. It was also hypothesized that using the PCS to measure pain catastrophizing, as recommended by George et al. (2006), would reveal a similar relationship between pain catastrophizing and both subjective pain ratings and physiological reactivity.

The current study replicated the results of George et al. (2006) in finding that fear of pain was positively correlated with subjective pain ratings for both male and female

participants. As predicted, pain catastrophizing was also found to correlate with pain ratings. Consistent with previous research, pain induction caused elevations in cardiovascular measures, which subsequently decreased on termination of pain induction. However, there was no relationship between pain catastrophizing and any of the cardiovascular dependent measures (HR, systolic BP, diastolic BP), despite using the PCS rather than the CSQ Catastrophizing subscale. Fear of pain was also unrelated to any of the physiological measures, consistent with the findings of George et al. (2006), and contrary to predictions that heightened pain-related anxiety would contribute to more intense physiological responses to pain. Generally speaking, measures of fear of pain and pain catastrophizing had no discernible influence on physiological measures of reactivity to pain induction.

Our study was motivated in part by the assumptions that individual differences in cognitive and affective responses to pain would correspond to physiological changes associated with pain induction. However, while participants on average showed increased HR and BP during pain induction, neither fear of pain nor pain catastrophizing influenced the degree of physiological reactivity. This suggests that relationships among cognitive and affective factors associated with pain do not directly or significantly influence physiological reactivity to pain. Conversely, it may be that despite the typical observation of HR and BP increases in response to pain, these measures may simply be only coarse indicators of responsiveness to pain. In other words, while HR and BP responses to the CP task may be indicative of physiological responding to the pain experience, they may not be an index of pain severity per se. This conclusion is supported by the finding that subjective pain ratings were uncorrelated with increases in BP or HR.

One explanation for the finding that PCS scores were uncorrelated with cardiovascular variables could be the timing of PCS administration, which occurred prior to beginning the CP task. Although pain catastrophizing is most often conceptualized as a trait-like variable, it is possible that the tendency to catastrophize is latent and requires some cue in order to become fully manifested (Quartana et al. 2009). Therefore, it is possible that measures like the CSQ and PCS, which require individuals to recall past pain experiences, may refer to events that are too temporally distant to permit full recollection of cognitive and affective reactions to the painful event. Supporting this possibility, Dixon et al. (2004) examined PCS scores both before and after administration of a CP task and found that post-CP scores were positively correlated with pain ratings and negatively correlated with CP tolerance, while pre-CP scores did not show these relationships. Furthermore, pre-CP PCS scores were not significantly correlated with post-CP PCS scores and were lower than post-CP PCS scores, particularly in women. These findings were replicated in a subsequent study (Edwards et al. 2005). However, a more recent study (Hirsh et al. 2008) did not replicate these results: neither pre- nor post-CP PCS scores were predictive of pain intensity, tolerance or threshold. More recently, the PCS has been adapted to specifically examine situational catastrophizing (Campbell et al. 2010). Using a 6-item adaptation of the PCS, Campbell et al. (2010) examined pain responses to heat, cold, and pressure pain and observed stronger correlations between post-pain situational catastrophizing scores and experimental pain ratings. Thus, while the extant literature is somewhat mixed, it is possible that the timing of assessment is a critical factor in whether relationships between catastrophizing and pain will be observed. In any case, failure to administer the PCS immediately following the CP task as a situational rather than state measure of pain catastrophizing may be considered a limitation of the current study, in light of previous studies finding that post-pain PCS scores are poorly correlated with pre-pain PCS scores (e.g., Dixon et al. 2004; Edwards et al. 2005).

Despite the mostly negative findings, a number of gender differences were observed. Consistent with previous research, mean subjective pain ratings during pain induction were higher for women than for men (e.g., Feine et al. 1991; Paulson et al. 1998). Males had higher systolic and diastolic BP overall, while females had higher HR overall. Despite lower subjective pain ratings, males exhibited higher systolic and diastolic BP reactivity during pain induction relative to females. Furthermore, whereas female BP increases on average returned to baseline levels following pain-induced elevation, for men the BP increases during pain induction remained significantly elevated above baseline at the end of the 10-min recovery period. This finding converges with a study by Light et al. (1993), who observed slower cardiovascular recovery in men after a series of stressors, including forehead cold-pressor, which may have been due to increased cardiac output (i.e., stroke volume) and/or increased peripheral resistance in males during the recovery period. These results suggest that the CP task was more physiologically taxing for men than women, at least with respect to cardiovascular reactivity, and may be due to the fact that relative to women, young men are at higher risk for the development of hypertension (Light et al. 1993). Given that the BP responses to the cold-pressor are associated with vasoconstriction and increased risk for hypertension, and are thought to be mediated by the psychological properties of the task (see Brownley et al. 2000 for a review), individual differences in other psychological factors like rumination may be a fruitful area for future inquiry.

The dissociation between physiological and subjective indices is curious and worthy of further examination. As

mentioned previously, cardiovascular responses during the CP task may not be an index of pain per se, and as such, may not be related to the subjective experience of pain. Alternatively, men may have used coping strategies like reappraisal or distraction to reduce experienced pain more effectively than women. Another possibility is that men were reluctant to report their actual levels of discomfort and under-reported their pain levels during the CP task. At the same time, interpretation of these findings is complicated by the well-documented finding that current hypertension (as well as future risk for hypertension) is associated with hypalgesia (France 1999). Thus, reduced pain ratings in those with high BP readings may be explained in part by the antinociceptive effect that accompanies hypertension or risk for hypertension (e.g., Ghione et al. 1988; Ghione 1996).

Further, it should be noted that a large study of Emergency Department patients presenting with verified pain-inducing tissue pathology, primarily kidney stone or fracture, reported only a very small correlation between pain rating and HR ($r = .08$), which was deemed clinically insignificant (Marco et al. 2006). Accordingly, a clear association between subject pain experience and cardiovascular reactivity cannot be unconditionally assumed.

Limitations of the current study primarily involve a relatively small sample size. In addition to being a smaller study to begin with ($n = 68$), the loss of 7 participants due to problems with instrument sensitivity and an additional 20 participants dropping out due to pain intolerance early in the CP task resulted in a sample size of 38. As a result, statistical power is lower than would be preferable. In addition, the clinical relevance of the current study is somewhat limited due to the acute nature of the experimental pain induction. While the CP task is thought to be a reasonable experimental model of chronic pain (e.g., Rainville et al. 1992), it may not have been aversive, prolonged, or threatening enough to elicit psychological reactions equivalent to those associated with chronic pain conditions (see Hirsh et al. 2008 for a discussion of this issue). While the current study did not examine the perceived threat of the CP task, participants were informed that they could terminate the task at any time and that the time of immersion would not exceed 5 min. Therefore, it is possible that the task was not perceived as threatening, or that coping strategies were used to decrease subjective pain levels.

Nonetheless, observed gender differences in subjective pain ratings and the relationship between pain ratings and fear of pain replicate previous research, and robust gender differences in cardiovascular profiles in response to pain induction were noted. Future research should include examinations of the extent to which such differences reflect biological versus psychosocial differences. Future studies may also examine the generalizability of these results to other forms of pain induction including thermal, tourniquet, and electric shock. Additional research is necessary to determine the role of individual differences in other pain-related constructs such as pain-related anxiety, locus of control, self-efficacy, and somatization. Finally, inclusion of other measures of personality traits (e.g., neuroticism) or forms of psychopathology (e.g., depression, anxiety, as well as chronic pain patients) would help to refine our understanding of the possible influence of such psychological factors in the relationships among self-reported pain intensity and physiological reactivity to pain.

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Conflict of interest All authors declare that no conflicts of interest exist related to the current study.

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