Cold Pressor Pain and Psychophysiological Activation in High and Low Pain Catastrophizers

by

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A thesis submitted to the Department of Psychology

in conformity with the requirements for

the degree of Master of Science

Queen's University

Kingston, Ontario, Canada

July, 2008

Abstract

Pain catastrophizing is a robust predictor of heightened pain report. However, theoretical models addressing mechanisms of catastrophizing are lacking. The purpose of this study was to examine whether high and low catastrophizers manifest differential psychophysiological arousal patterns in an experimental laboratory pain setting. Gender differences and covariates were also examined. Participants were 102 undergraduate students (56 female, 46 male) at Queen's University recruited from the Psychology 100 subject pool aged 17-28 years (M = 18.44, SD = 1.40). After deleting participants with missing data, n = 95 for pain report/psychological variables, and n = 93 for psychophysiological measures. Participants were pre-screened using the Pain Catastrophizing Scale (PCS) from which individuals were selected from the top third (i.e., PCS score $\geq$ 24) and the bottom third (i.e., PCS score $\leq$ 13) of the distribution to represent high and low catastrophizers respectively. Participants completed measures of depression and anxiety before participating in a cold pressor task. Participants' heart rate (HR) and galvanic skin response (GSR) were recorded during periods of baseline (3 minutes), pain induction (1 minute), and pain recovery (10 minutes). The Short-Form McGill Pain Questionnaire (SF-MPQ) was completed following the recovery period. Results showed that catastrophizing was related to greater pain intensity during pain induction and during pain recovery, as well as greater retrospective sensory and affective reports of pain. Women reported greater pain intensity during pain induction, during pain recovery, and greater retrospective reports of sensory pain. These effects were significant even when controlling for depression and anxiety. With regard to psychophysiological arousal, catastrophizing was related to higher HR during pain induction. There also

appeared to be trends suggesting that catastrophizing may also be associated with HR before and after pain induction. Results provide partial support for an appraisal model of catastrophizing and may be useful in informing future models implicating catastrophizing in the development and maintenance of chronic pain.

Acknowledgements

First, I would like to thank my supervisor, Dr. Dean Tripp, for his guidance, support, and encouragement throughout the time that we have been working together.

I would also like to thank my committee members, Dr. Tom Hollenstein and Dr. Patricia Minnes. Your feedback and suggestions were greatly appreciated. I would also like to thank Dr. Uzma Rehman for her advice and suggestions in the proposal phase of this project.

Thank you to my research assistants, Karen Zhang and Monica Armanious, for their assistance with data collection and data entry. Thank you also to Natalie Stechyson for her assistance with data entry.

A special thank you to my labmates, Mary Acreman, Jess Ginting, and Annie Hsieh, as well as my friends and family for supporting me through this process.

Lastly, thank you to Aaron, for his ongoing support over the years.

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Chapter 1: Introduction

Understanding pain determinants and associations is a mandated research goal because pain is widely prevalent, exerts a huge emotional and social impact on its sufferers and their loved ones, and is an ever-increasing financial burden. Indeed, pain is a central health care issue and is the primary complaint in approximately 80% of physician visits in Canada (Canadian Pain Consortium, 2001). Greater pain has been linked to poorer quality of life, increased prevalence and incidence of depression and anxiety, problems with interpersonal relationships, and increased health care utilization (e.g., Becker et al., 1997; Catala et al., 2002; Moulin, Clark, Speechley, & Morley-Foster, 2002; Von Korff, Dworkin, Le Resche, & Kruger, 1988). Pain also accounts for a significant portion of health care spending in Ontario, with pain-related illness costing 5 billion dollars annually (Canadian Pain Consortium, 2001).

Pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (Merskey & Bogduk, 1994, p. 209), indicating that the pain experience is influenced by both physical and psychological factors. As described in what are now classic publications, pain has both sensory and affective qualities, is almost always seen as a threat to the individual organism, and is associated with strong desires and behaviours to stop, diminish, or avoid its occurrence (e.g., Hardy, Wolf, & Goodell, 1952; Melzack & Casey, 1968; Melzack & Wall, 1965). With the introduction of the Gate Control Theory of Pain, Melzack and Wall (1965) crafted a new model of pain that employed medical and psychological concepts of pain as reciprocating inputs with bottom-up and top-down components. Thus, pain not only includes components of noxious stimulation (i.e.,

intensity), but also negative affective responses and the influence of cognitive processes that individualize the pain experience. The Gate Control Theory of Pain acted as a stepping-stone for volumes of future studies, suggesting that pain is modulated by a host of psychological factors (e.g., Melzack & Casey; Melazack & Wall).

Catastrophizing

Although many pain-associated psychological variables have been examined, research has consistently identified catastrophizing as a major individual difference factor in pain reports. Originally defined as a cognitive distortion found in depressed patients (Beck, 1976), catastrophizing has long been a construct that represents profound negative ruminations about one's ability to cope or self-regulate distress. In reference to pain, catastrophizing was presented as a pervasive set of dysfunctional beliefs or appraisals of one's ability to manage or tolerate pain (Jensen, Turner, Romano, & Karoly, 1991; Sullivan, Bishop, & Pivik, 1995). In what is arguably the defining paper on the construct of pain catastrophizing, Sullivan, Thorn, et al. (2001, p. 53) defined catastrophizing as "an exaggerated negative mental set brought to bear during actual or anticipated pain experience". Further, it has been suggested that pain catastrophizing consists of three inter-related factors: rumination (e.g., "I cannot stop thinking about the pain"), magnification (e.g., "I wonder whether something serious may happen"), and helplessness (e.g., "There is nothing I can do to stop the pain") (Sullivan et al., 1995, Sullivan, Thorn, et al., 2001).

Regardless of whether one examines clinic-, laboratory-, or epidemiological-based research, pain catastrophizing is a robust individual difference factor (e.g., Picavet, Vlaeyen, & Schouten, 2002; Sullivan, Thorn, et al., 2001; Tripp et al., 2006).

Catastrophic thinking about pain is associated with greater acute pain in clinical populations (e.g., post-operative patients and dental hygiene patients; Jacobsen & Butler, 1996; Sullivan, Thorn, Rodgers, & Ward, 2004) and in healthy individuals undergoing laboratory-based pain induction tasks (e.g., France, France, al'Absi, Ring, & McIntyre, 2002; Sullivan et al., 1995; Sullivan, Rodgers, & Kirsch, 2001; Sullivan et al., 2004). As well, catastrophizing is predictive of greater pain in medical conditions where pain is persistent, such as low back pain (Smeets, Vlaeyen, Kester, & Knottnerus, 2006), rheumatoid arthritis/fibromyalgia (Edwards, Bingham, Bathon, & Haythornthwaite, 2006), and pelvic pain disorders in men (Tripp et al., 2006). Catastrophizing is not only robustly predictive of pain intensity reports, but is also associated with greater pain behaviors, such as verbal pain reports, the number of visits to health care professionals, the use of analgesic (i.e., pain-reducing) medications, and disability (Sullivan, Stanish, Waite, Sullivan, & Tripp, 1998; Sullivan, Thorn, et al., 2001; Tripp et al., 2006). In addition to its strong association with pain report, catastrophizing is a stable construct, showing little change in test-retest scores without intervention (Sullivan et al., 1995, Sullivan, Thorn et al., 2001; Turner, Mancl, & Aaron, 2004).

Theoretical Perspectives on Catastrophizing

Although catastrophizing is a robust predictor of various pain and pain-associated outcomes, there has been little work published on theoretical perspectives addressing how catastrophizing may function in the elevation of pain report. Indeed, Thorn, Ward, Sullivan, and Boothby (2003) argued that much of the previous pain and catastrophizing research has been conducted without a guiding theoretical model and that little was

known about how catastrophizing is associated with existing models of health and distress.

In their review of the literature, Sullivan, Thorn, et al. (2001) suggested that catastrophizing acts to amplify pain report via an appraisal process, sharing features of the primary and secondary appraisal processes described in Lazarus and Folkman's (1984) Transactional Model of Stress (TMS). The TMS suggests that the manner in which one appraises a situation will affect the level of stress the individual experiences and manifests (Lazarus & Folkman, 1984). Further, the TMS suggests that primary appraisals are employed to assess the general threat value of a potential stressor, which is followed by a secondary appraisal examining whether the person believes they can manage or cope with the threat as it has been perceived. This process is suggested to be recursive, acting in a cyclical manner termed reappraisal, indicating that the appraisal process is ongoing.

As suggested by Sullivan, Thorn, et al. (2001), the pain catastrophizing factors are consistent with the primary and secondary appraisals described in the TMS. Specifically, the rumination and magnification factors of catastrophizing are suggested to be conceptually similar to primary appraisals (i.e., perceiving the situation as threatening), whereas the helplessness component of catastrophizing is thought to mimic secondary appraisals (i.e., perceiving oneself not to have enough resources to cope with the pain stressor) (e.g., Sullivan et al., 1995). Support for the conceptualization of catastrophizing as an appraisal process has been previously offered (e.g., Jensen et al., 1991; Sullivan et al., 1995; Sullivan, Thorn, et al., 2001). Furthermore, Unruh, Ritchie, and Merskey (1999) found that catastrophizing was associated with report of threat appraisals in

patients with pain. This notion has also been supported more recently by Thorn (2004), who discussed catastrophizing in chronic pain with regard to primary and secondary appraisals in the stress-appraisal-coping model.

What is Stress?

Stress is a general term used to describe a state of physiological or emotional alarm associated with a physical or perceived threat to bodily homeostasis (Johnson, Kamilaris, Chrousos, & Gold, 1992). Stress can be reported verbally, but can also be indexed physiologically through the autonomic nervous system (ANS), which is the component of the peripheral nervous system associated with activation of the internal organs during times of perceived threat. The ANS is composed of the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS), which usually act to counterbalance one another resulting in bodily homeostasis (Thayer & Brosschot, 2005). The SNS is commonly referred to as the system responsible for engaging the "fight-or-flight" response. For example, in response to an actual or perceived threat, blood pressure, heart rate (HR), and perspiration increase in order to help mobilize the body for strong physical actions like physical confrontation or rapid departure from the threatening situation (Salpolsky, 2007; Thayer & Brosschot). The PNS is responsible for the return to homeostasis component of the ANS, which attempts to down-regulate the body to its resting state (commonly referred to as the "rest and digest" response). For example, PNS activation would be demonstrated by decreased HR and activation of hormones responsible for enhancing energy storage (Salpolsky; Thayer & Brosschot). For a more detailed discussion on stressors and physiological arousal see Johnson et al (1992).

Psychophysiological measures are commonly used to measure the stress response due to the relative ease of data collection compared to other measures (e.g., cortisol). Commonly-used measures of stress-related arousal used when assessing pain include HR and galvanic skin response (GSR) (Flor, 2001). HR (i.e., the electrical activity of the heart measured in beats per minute) is a measure of both SNS and PNS activity (Flor). GSR (i.e., the electrical activity of the skin, or the amount of sweat produced) is a measure of SNS activity (Flor). Thus, both HR and GSR are indicators of SNS arousal (Lazarus, Speisman, and Mordkoff, 1963).

Appraisals and Stress

The TMS suggests that when an individual is confronted with a situation that is appraised as threatening, stress ensues if the individual judges himself or herself as having insufficient resources to cope with the threat (Lazarus & Folkman, 1984). Data indicate that people who appraise situations as threatening report greater perceived stress and manifest greater psychophysiological arousal. For example, Maier, Waldstein, and Synowski (2003) showed that individuals who appraised a mental arithmetic test as threatening not only reported greater perceived stress, but also had elevated psychophysiological arousal as assessed by diastolic blood pressure. Furthermore, Tomaka, Blascovich, Kelsey, and Leitten (1993) found that threat appraisals of a scary movie were related to greater subjective stress and corresponding increases in psychophysiological arousal assessed by increased HR and GSR. Thus, threatening appraisals are associated with greater psychophysiological arousal. As such, because catastrophizing has been theorized to act as a threat appraisal, it is reasonable to consider psychophysiological arousal when investigating differences in catastrophizing.

Catastrophizing and Stress

Although catastrophizing has been proposed to act similarly to primary and secondary appraisal processes (Sullivan, Thorn, et al., 2001), the relationship between pain catastrophizing and psychophysiological arousal has yet to be examined in acute pain. The linkage between catastrophizing and stress-related arousal is further strengthened by research examining catastrophic-like thoughts and arousal. Described as perseverative cognitions, ruminative types of cognitions have been associated with stress-related negative health outcomes (Brosschot, Gerin, & Thayer, 2006; Brosschot, Pieper, & Thayer, 2005). Perseverative cognitions have been shown to be associated with greater and prolonged stress responses as evidenced by heightened SNS and decreased PNS activation (Brosschot et al., 2006). Studies have also linked trait rumination and worry with longer HR recovery times following stressful tasks and fear-provoking situations (e.g., Roger & Jamieson, 1988; Segerstrom, Glover, Craske, & Fahey, 1999). Further, Brosschot and Thayer (2003) showed that experiencing negative emotions was associated with elevated arousal as assessed by HR as long as 5 minutes following the emotional onset. They suggested that this prolonged cardiovascular activation was due to perseverative negative emotions. Finally, perseverative cognitions are also associated with other indicators of the stress response, such as poorer immune system functioning and somatic complaints (Brosschot et al., 2006).

Recently, the relationship between coping styles and physiological measures has been examined in the pain literature. Edwards and Fillingim (2005) found that active coping was associated with decreases in mean arterial pressure (i.e., average blood pressure) and cardiac contractility (i.e., strength of heart beat) from baseline to post-pain

induction, whereas catastrophizing was associated with increases in cardiac contractility from baseline to post-pain induction. However, this study did not measure arousal during the pain induction task. An important consideration when studying pain catastrophizing is the pain experience. Melzack (1999) suggested that modernized pain theory may have been so heavily geared toward examination of the once ignored psychological aspects that promote negative pain experiences that the stress/arousal response is often overlooked when considering pain. However, pain disrupts the homeostatic balance of the body, thereby enabling stress-related pain disorders, including chronic pain disorders (e.g., fibromyalgia), to ensue. Thus, it is important to consider the relationship between pain and arousal when considering pain catastrophizing and arousal. Literature regarding pain and the stress response is reviewed in the following section.

Pain and Stress

Psychophysiological responses in both acute and chronic pain have been investigated in the literature. However, although both acute and chronic pain are discussed, it is important to make the distinction between the two types of pain. Acute pain generally lasts for a short period of time, whereas chronic pain is more persistent, typically defined as pain lasting longer than 3 months (Sternbach, 1989).

Both acute and chronic pain are associated with increased psychophysiological arousal. Indeed, when either acute or chronic pain is present, stress physiology is activated (Melzack, 1999). For example, Dowling (1983) found that acute pain was associated with increases in both HR and GSR. Furthermore, greater pain is associated both with heightened blood pressure and increased GSR. Specifically, greater pain intensity corresponds strongly to increases in SNS activation (e.g., GSR) in healthy

participants (Donaldson et al., 2003). Induction of acute cold pressor pain is also associated with increases in blood pressure in healthy participants (e.g., al'Absi & Petersen, 2003; Dixon, Thorn, & Ward, 2004). As well, acute pain is associated with greater GSR reactions in patients with chronic low back pain, but not in control participants (e.g., Peters & Schmidt, 1991).

Why Study Catastrophizing and Arousal in Relation to Pain?

There are several impetuses to promote the study of arousal and catastrophizing in pain. As suggested in the TMS-relevant literature, appraisals exert significant influence on individual responses to real or even imagined events. Although catastrophizing has been suggested to act as an appraisal process (i.e., Jensen et al., 1991; Sullivan, Thorn, et al., 2001), a stress response profile has yet to be examined across a catastrophizing versus non-catastrophizing control group. There is also associated research suggesting that threat appraisals, which include catastrophizing-like constructs such as perseverative cognitions, are associated with measures of arousal. Finally, given that pain is a stressor associated with elevated psychophysiological arousal, examining the association between catastrophizing and differential psychophysiological arousal is warranted.

Thorn et al. (2003) noted that catastrophizing has long been investigated in the pain literature without significant linkages to existing theoretical models. As Thorn et al. asserted, having theoretical models to aid in understanding pain catastrophizing and how it may operate to influence heightened pain reports is a necessary step in the pain literature. Thus, using a stress-appraisal framework of pain catastrophizing along with psychophysiological assessment is a warranted and theoretically-supported research endeavor (e.g., Maier et al., 2003; Tomaka et al., 1993). In addition, investigating

correlates of catastrophizing will allow for greater investigation of methods for targeting catastrophic thoughts in clinical pain treatment (e.g., Sullivan, Tripp, & Santor, 2000).

A Model of Catastrophizing, Stress, and Pain

Based on the previous TMS and pain literature reviewed, a model of catastrophizing, pain, and psychophysiological arousal was proposed (see Figure 1). This model was created due to the lack of previous research examining a mechanism of the relation between catastrophizing and heightened pain report. The model describes three time periods in which catastrophizing may have an effect on arousal and pain report. In this model, there is consideration of the period before pain is actually induced or a *baseline period*. There would also follow a period in which the individual experiences pain, aptly referred to as the *pain induction period*. Finally, upon termination of the painful stimulus, the final period can be considered a *pain recovery period*.

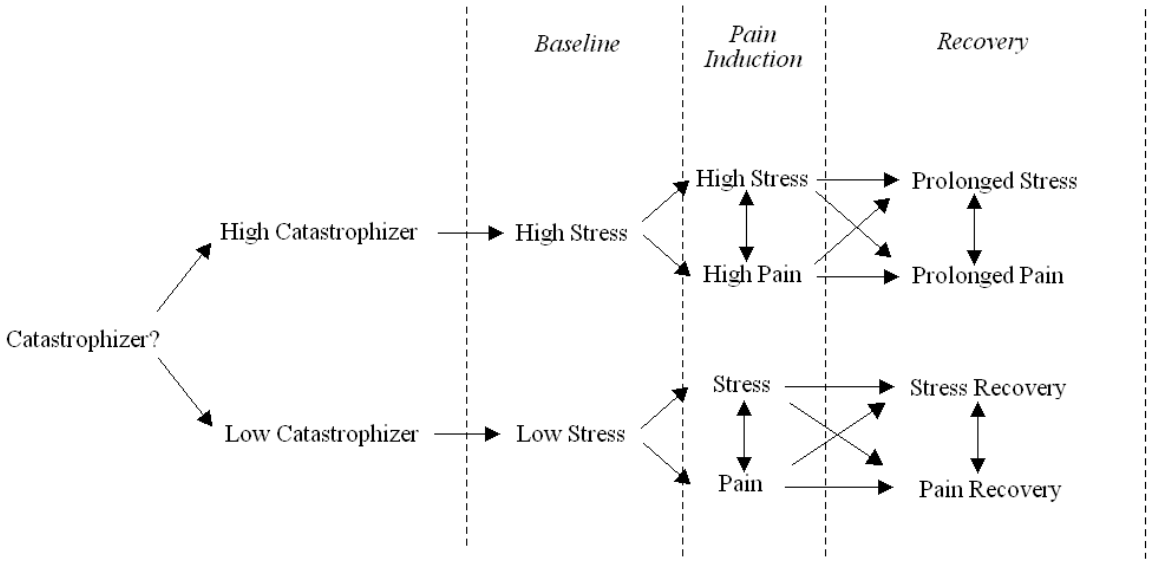

Figure 1. Model depicting the relationship between catastrophizing, pain, and psychophysiological arousal across baseline, pain induction, and pain recovery periods.

Based on an appraisal model of catastrophizing, it would be expected that catastrophizing would be associated with greater arousal at all three periods. In particular, at the baseline period, an appraisal model of catastrophizing suggests that catastrophizing acts as a threat appraisal as an individual awaits a painful experience and that such a threat would be associated with heightened psychophysiological arousal (e.g., Maier et al., 2003; Tomaka et al., 1993). This prediction is consistent with an appraisal model of pain catastrophizing (Sullivan, Thorn, et al., 2001), where pain is suggested to be an attention-grabbing threat to those rated high in catastrophizing. The proposed model also suggests that psychophysiological arousal would be elevated for catastrophizers during and following a pain event. Indeed, these predictions are consistent with the literature suggesting that catastrophizing and catastrophizing-like cognitions are associated with higher arousal in proximal response to a stressor as well as prolonged arousal following the cessation of a stressor (Brosschot & Thayer, 2003; Edwards & Fillingim, 2005; Thayer, Friedman, & Borkovec, 1996). However, as mentioned previously, the relationship between catastrophizing and arousal in response to pain has not been previously examined.

The proposed model in Figure 1 also depicts other relationships of interest in addition to the associations between catastrophizing and arousal in the time periods surrounding pain. Not only does the proposed model outline the associations between catastrophizing and arousal, it also outlines the associations previously found in the pain literature regarding the relationship between catastrophizing and pain, namely that catastrophizing is associated with greater pain (Edwards et al., 2006; Jacobsen & Butler, 1996; Sullivan et al., 1995, 1998, 2001, 2004; Smeets et al., 2006; Tripp et al., 2006).

Furthermore, the model proposes arousal activation concurrent with pain, which is consistent with evidence indicating that stress and pain are associated (e.g., al'Absi & Petersen, 2003; Dixon et al., 2004; Donaldson et al., 2003). The proposed model also suggests that arousal at earlier time points would be associated with arousal at later time periods and that pain at earlier time periods would be related to pain at later time periods. These predictions are consistent with the results of Dixon et al.'s study, which indicated that blood pressure before a pain induction task was related to blood pressure after pain induction and also with results of studies indicating that pain during pain induction is related to pain following the stimulus (al'Absi & Petersen, 2003). In addition, the model speculates that arousal at earlier time periods would be associated with pain at later time periods and that pain at earlier time periods would be associated with arousal at later time periods.

Although there are no direct studies examining the associations of catastrophizing, pain and psychophysiological assessment during acute pain, there are possibilities in sequencing the associations between catastrophizing and arousal for acute pain that might be assessed with mediational analyses. As previously stated, based on an appraisal model of catastrophizing, the logical suggestion is that individuals higher in catastrophizing may appraise impending pain induction as more threatening and would likely have greater arousal in the period preceding pain induction (see Figure 1). This anticipatory arousal might then be associated with greater pain report. Thus, perhaps the association between catastrophizing and heightened pain report is mediated by arousal.

The suggestion that arousal may mediate the association between catastrophizing and heightened pain report is supported by stress and pain research. For example,

Brosschot et al. (2006) proposed in their perseverative cognition hypothesis that perseverative cognitions lead to prolonged stress responses, which then in turn are related to disease. Further, Salpolsky (2007) noted that activation of the stress response can lead to stress-related disease via PNS suppression. In addition, Melzack (1999) proposed that stress experienced in response to pain might be related to the development of chronic pain disorders. In light of the evidence suggesting that situations appraised as threatening lead to stress (e.g., Maier et al., 2003; Tomaka et al., 1993) and that catastrophizing-like cognitions are related to greater stress (e.g., Brosschot et al., 2006), it is therefore suggested that catastrophizing results in heightened arousal, which may in turn lead to greater pain report both during and following pain induction. That is, it is hypothesized that arousal may mediate the association between catastrophizing and heightened pain report.

Gender Differences

Gender differences are an important consideration when examining the relationship between catastrophizing and pain. Pain research indicates that women report greater pain and catastrophizing than men (e.g., Unruh, 1996; Sullivan et al., 2000). However, when examining the relationship among catastrophizing, gender, and pain, there are some mixed results (e.g., Edwards, Haythornthwaite, Sullivan, & Fillingim, 2004; Sullivan et al., 2000). In experimental pain research, Sullivan et al. (2000) found that when catastrophizing was included as a covariate in the analysis, the relationship between gender and pain was no longer significant. Edwards et al. (2004) failed to replicate this effect of catastrophizing for experimental pain, but did find similar results for reported daily pain. Given these mixed results, and with the bulk of the research

showing gender differences in catastrophizing and pain, effects of gender on pain and interactions between catastrophizing and gender in predicting pain were examined in the present study.

Furthermore, although gender differences were not seen in threat appraisals (e.g., Unruh et al., 1999), gender is associated with greater psychophysiological activation in regard to pain, with women exhibiting lower blood pressure and reporting greater pain during a pain induction task than men (Dixon et al., 2004). Thus, it is expected that results for psychophysiological activation will follow a similar pattern to those for pain (i.e., females will have greater psychophysiological arousal than males).

The Present Study

Using the proposed model of catastrophizing and psychophysiological arousal (see Figure 1), this study sought to examine individual differences in arousal between people self-reported to be high and low in catastrophizing during a laboratory-based acute pain induction task. To examine the relationship between catastrophizing and pain report, participants were monitored for psychophysiological arousal before, during, and following immersion of their forearm into a cold water tank. Participants reported pain intensity and pain unpleasantness during pain induction and pain recovery periods. Both pain intensity and pain unpleasantness were collected to obtain sensory and affective descriptions of pain during the pain induction and pain recovery periods. As well, participants provided retrospective reports of sensory and affective pain from the pain induction period following completion of the pain recovery period in the form of a questionnaire.

As previously suggested, although catastrophizing has been conceptualized as an appraisal process, differences in psychophysiological activation during acute pain between individuals high and low in catastrophizing remain unexamined. Examining the association between catastrophizing and the psychophysiological stress response allows for the initial modeling of the differential arousal pattern that high catastrophizers may exhibit throughout their pain experience when compared to low catastrophizers. Furthermore, until recently (e.g., Aslaksen, Myrbakk, Hoifodt, & Flaten, 2007) some previous pain-arousal study methodologies have been limited in their assessment of psychophysiological activation to before and/or after a pain task (Dixon et al., 2004; Edwards & Fillingim, 2005) or at separate chosen time points during a pain task (al'Absi & Petersen, 2003). It is suggested that a continuous measurement of psychophysiological activation (i.e., HR and GSR) before, during, and after pain experience in an acute pain induction task is important to consider because it allows for continuous assessment of changes over time, which may provide a more robust test of the arousal indices.

Objectives and Hypotheses

Based on the literature reviewed and the model proposed in Figure 1, four main objectives were proposed for the present study. The objectives, and the hypotheses corresponding to each of these objectives, are listed below:

The first objective was to examine the associations between the pain and arousal outlined in the model proposed in Figure 1. Hypothesis 1 outlines the particular aspects of direct study under this objective (see following page).

Hypothesis 1. It was expected that associations between pain and arousal outlined in the model would be significantly correlated. More specifically, it was expected that:

a. Pain reported during the pain induction period would be positively associated with pain during the pain recovery period. Also, pain intensity and pain unpleasantness during the pain induction period would be positively associated as would pain intensity and pain unpleasantness during the pain recovery period.
b. Arousal (i.e., HR and GSR) during the baseline period would be positively associated with arousal during the pain induction period. Also, arousal during pain induction would be positively associated with arousal during the pain recovery period. Finally, HR and GSR would be positively associated within the baseline, pain induction, and pain recovery periods.
c. Pain reported during the pain induction period would be positively associated with arousal during pain induction. Likewise, pain reported during the pain recovery period would be positively associated with arousal during the pain recovery period.
d. Arousal during the baseline period would be positively associated with pain during the pain induction period. As well, arousal during the pain induction period would be positively associated with pain during the pain recovery period.

e. Pain during the pain induction period would be positively associated with arousal during the pain recovery period

The second objective was to replicate previous findings documenting relationships between catastrophizing, gender, and pain report. This objective included extending the findings to include pain measured after participating in a pain induction task during a recovery period and also retrospective reports of sensory and affective pain. Hypotheses 2-4 outline the particular aspects of direct study under this second objective (see below).

Hypothesis 2. It was expected that catastrophizing and gender would be related to reported pain during the pain induction period. More specifically:

a. High catastrophizers would report greater pain intensity and pain unpleasantness during the pain induction period than low catastrophizers.
b. Women would report higher pain intensity and pain unpleasantness during the pain induction period than men.
c. The interaction between catastrophizing and gender was also examined. It was expected that catastrophizing and gender would interact, such that for women, high catastrophizers would report greater pain than low catastrophizers, but this effect would not be as large for men.

Hypothesis 3. It was expected that catastrophizing and gender would be related to reported pain during the pain recovery period. More specifically:

a. High catastrophizers would report greater pain intensity and pain unpleasantness during the pain recovery period than low catastrophizers.
b. Women would report greater pain intensity and pain unpleasantness during the pain recovery period than men.
c. The interaction between catastrophizing and gender was also examined. It was expected that catastrophizing and gender would interact, such that for women, high catastrophizers would report greater pain intensity and pain unpleasantness than low catastrophizers, but this effect would not be as large for men.

Hypothesis 4. It was expected that catastrophizing and gender would be related to pain reported retrospectively following the pain recovery period. More specifically:

a. High catastrophizers would report greater sensory pain and affective pain than low catastrophizers.
b. Women would report greater sensory pain and affective pain than men.
c. The interaction between catastrophizing and gender was also examined. It was expected that catastrophizing and gender would interact, such that for women, high catastrophizers would report

greater sensory pain and affective pain than low catastrophizers, but this effect would not be as large for men.

The third objective was to examine the relationship between catastrophizing, gender, and psychophysiological arousal over time. Hypothesis 5 outlines the particular aspects of direct study under the third objective and specifies particular interactions (see below).

Hypothesis 5. It was expected that catastrophizing and gender would be related to greater arousal (i.e., HR and GSR) during baseline, pain induction, and pain recovery periods. More specifically:

a. High catastrophizers would have higher arousal than low catastrophizers across baseline, pain induction, and pain recovery periods.
b. Women would have higher arousal than men across baseline, pain induction, and pain recovery periods.
c. Arousal would be higher during the pain induction period than during baseline or pain recovery periods.

Interactions between catastrophizing, gender, and time were also examined. Predictions regarding each of these potential interactions are outlined on the following page:

d. Catastrophizing and gender would interact, such that for women, high catastrophizers would display higher arousal than low catastrophizers, but this effect would be smaller for men.

e. Catastrophizing and time would interact, such that high catastrophizers would have higher arousal during pain induction than baseline, but there would be no difference in arousal between pain induction and pain recovery periods. Low catastrophizers, in contrast, would have higher arousal during pain induction than during baseline, but there would be an expected decrease in arousal during the pain recovery period.

f. The gender by time interaction was considered exploratory, as Dixon et al. (2004) found conflicting results surrounding interactions between gender and time such that there were gender by time interactions for some psychophysiological measures (e.g., diastolic blood pressure), but not others (e.g., systolic blood pressure). However, based on the evidence indicating that women typically report higher pain than men (e.g., Unruh, 1996), and pain is related to SNS psychophysiological activation (e.g., Dixon et al., 2004), it is suggested that similar to high catastrophizers, females may have greater arousal during pain induction than during baseline; however, female arousal would not decrease as drastically as for males during the pain recovery period.

g. The three-way interaction between catastrophizing, gender, and time was also exploratory. However, based on the literature reviewed, it

was hypothesized that catastrophizing, gender, and time would interact, such that for low catastrophizing females, arousal would decrease slightly during the recovery period, but would not decrease for the high catastrophizing females. In contrast, for low catastrophizing males, arousal would decrease over time to a greater extent than for the low catastrophizing females, and would decrease only slightly for the high catastrophizing males.

The fourth and final objective was to examine arousal as a mediator of the relationship between catastrophizing and pain. Hypothesis 6 outlines the particular aspects of direct study under this fourth objective (see below).

Hypothesis 6. It was expected that arousal would mediate the relationship between catastrophizing and pain (see Figure 2). More specifically:

a. Arousal in the baseline phase would fully mediate the relationship between catastrophizing and pain during the pain induction period.
b. Arousal in the pain induction period would partially mediate the relationship between catastrophizing and pain during the pain induction period.
c. Arousal in the pain induction period would partially mediate the relationship between catastrophizing and pain in the pain recovery period.

d. Arousal in the pain recovery period would partially mediate the relationship between catastrophizing and pain during the pain induction period.

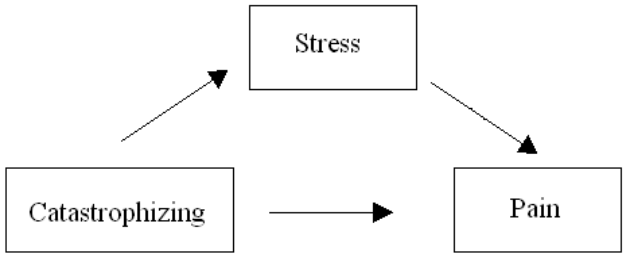

Figure 2. Mediation model depicting arousal (i.e., stress) mediating the relationship between catastrophizing and pain.

Covariates

Depressive symptoms and anxiety were also assessed and covaried out of any significant analyses due to their association with catastrophizing (Edwards et al., 2006; Sullivan et al., 1995) and the documented relationships between depressive symptoms/anxiety with pain (e.g., Asmundson, Jacobson, Allerdings, & Norton, 1996; Banks & Kerns, 1996; McCraken, Zayfert, & Gross, 1992; Robinson & Riley, 1999) and arousal (Johnson et al., 1992; Thayer et al., 1996; Thayer, Smith, Rossy, Sollers, & Friedman, 1998).

Although depression and anxiety may seem to share similar components with catastrophizing (e.g., rumination, helplessness), studies have shown that catastrophizing is a distinct construct, separate from depression and anxiety (Sullivan, Thorn, et al., 2001). For example, one study found that when controlling for depression at time 1,

catastrophizing still predicted depression at time 2 (6 months later), indicating that depression and catastrophizing are not redundant constructs (Keefe, Brown, Wallston, & Caldwell, 1989). Catastrophizing is also distinct from anxiety, as studies show that catastrophizing contributes significant unique variance when predicting pain, whereas anxiety does not (Sullivan et al., 1995). Because catastrophizing is distinct from depression and anxiety, it was expected that all hypotheses would be significant even when controlling for depression and anxiety.

Previous pain experiences were also assessed, as it is reasonable to suggest that a participant's number of previous encounters with pain could be related to their pain report and their psychophysiological response to pain. Indeed, one study found that when rating another person's perceived pain, ratings were higher when the observer completed a cold pressor task before rather than after observing another person (Robinson & Wise, 2004). If the number of previous pain experiences was related to pain or arousal, then it was covaried out of the appropriate analyses.

Furthermore, some studies show that females in the ovulatory and luteal phases have greater pain sensitivity than women in the follicular phase (Edens & Gil, 1995) and females in the luteal phase have higher HR than women in the follicular phase (al'Absi & Peterson, 2003). Therefore, females were asked when they had their last menstrual period and this was also covaried out of the appropriate analyses if it was significantly related to pain report or arousal.

In summary then, based on research indicating that catastrophizing is one of the strongest predictors of pain outcomes (Sullivan, Thorn, et al., 2001; Tripp et al., 2006), it was expected that results would still be significant when controlling for these factors.

Chapter 2: Method

Participants

Participants were 102 undergraduate students (56 female, 46 male) at Queen's University recruited from the Psychology 100 subject pool aged 17-28 years (M = 18.44, SD = 1.40). From this total sample, data from four participants were not used due to improper calibration of the equipment. Data from three additional participants were excluded due to illness, early termination of the pain induction task, and suspicion of marijuana intoxication. These seven participants were deleted from all analyses. Two additional participants were deleted from analyses involving psychophysiological variables because the equipment was malfunctioning (during the baseline period for one and during the pain induction period for the other). Thus, a sample consisting of 95 participants was available for analyses involving pain report or psychological measures (26 high catastrophizing females, 19 high catastrophizing males, 25 low catastrophizing females, 25 low catastrophizing males). For analyses involving psychophysiological variables, 93 participants were available (24 high catastrophizing females, 19 high catastrophizing males, 25 low catastrophizing females, 25 low catastrophizing males). Of the total sample remaining after deletions (N = 95), 89.5% (n = 85) were right handed. The sample was predominantly Caucasian (71.6%; n = 68); 22.1% of the sample was Asian (n = 21), and 6.3% were classified as another ethnicity (n = 6).

All potential participants were screened and excluded from participating if they reported medical conditions that could be aggravated by participating in a cold pressor task (e.g., frostbite, hypertension, negative cardiovascular history, chronic pain; Sullivan et al., 1995) or if they had previously participated in a cold pressor task. Participants were

compensated with course credit (i.e., 1% towards their final grade in a first year Psychology course) or $10.

Materials

Demographics Questionnaire. The demographics questionnaire consisted of 7 questions regarding gender, age, ethnicity, dominant hand, and family/personal pain experiences (see Appendix A). The questionnaire also asked female participants to report when they had their last menstrual period to determine whether the time of cycle was related to pain and/or arousal.

Pain Catastrophizing Scale. The Pain Catastrophizing Scale (PCS; Sullivan et al., 1995) is a 13-item self-report measure used to assess catastrophizing (see Appendix B). All items begin with the phrase, "When I'm in pain…". Statements include: "I worry all the time about whether the pain will end," "I keep thinking of other painful events," and "There's nothing I can do to reduce the intensity of the pain". Items are rated on a 5-point scale ranging from 0 (*Not at all*) to 4 (*All the time*). Scores are summed to obtain a total score of catastrophizing. Scores on the PCS range from 0 to 52, with higher scores indicating greater pain catastrophizing. The PCS also provides a score for three components of catastrophizing: rumination, magnification, and helplessness. The PCS has been shown to have high internal reliability (Cronbach's alpha was .93 for the total PCS score, .91 for rumination, .75 for magnification, and .87 for helplessness; Osman et al., 1997). Test re-test reliabilities are moderately high (r = .75 for six weeks; r = .70 for ten weeks; Sullivan et al., 1995). In this study, Cronbach's alpha was .92 for the total PCS score, indicating high internal consistency. The Cronbach's alphas for the subscales were .89 for rumination, .65 for magnification, and .86 for helplessness.

Pain Intensity. Pain intensity was assessed using an 11-point numerical rating scale (NRS) ranging from 0 (*No Pain*) to 10 (*Extreme Pain*). High pain intensity indicates greater pain experience. Participants verbally rated their pain intensity every 20 seconds when prompted by the experimenter. Jensen and Karoly (2001) recommended the use of the NRS because it is easy to administer and is a valid measure of pain intensity, correlating positively with other measures of pain intensity (e.g., Jensen, Karoly, O'Riordan, Bland, & Burns, 1989; Wilkie, Lovejoy, Dodd, & Tesler, 1990).

Pain Unpleasantness. To obtain an affective description of pain throughout the task, pain unpleasantness was assessed using an 11-point NRS ranging from 0 (*Not Unpleasant*) to 10 (*Extremely Unpleasant*). As with pain intensity, participants verbally rated their pain unpleasantness every 20 seconds when prompted by the experimenter. This measure of pain unpleasantness has been previously used in the pain literature when assessing acute pain induction (e.g., Pukall, Binik, & Khalife, 2004).

Short-Form McGill Pain Questionnaire. The Short-Form McGill Pain Questionnaire (SF-MPQ; Melzack, 1987) is a 15-item questionnaire used to assess two dimensions of pain: sensory and affective, as well as overall pain severity (see Appendix C). Participants reflected on their current pain experience and rate each of 15 pain descriptors such as "throbbing" or "tender" on a 4-point intensity scale ranging from 0 (*None*) to 3 (*Severe*). Scores on the total SF-MPQ (Pain Rating Index-Total; PRI-T) range from 0 to 45, where higher scores indicate higher perceived pain severity. The measure also has two subscales: the sensory subscale, which ranges from 0 to 33 (Pain Rating Index-Sensory; PRI-S) and the affective subscale, which ranges from 0 to 12 (Pain Rating Index-Affective – PRI-A). The SF-MPQ has high validity and reliability. The SF-MPQ

total score and subscales are correlated highly with the full-length MPQ total score and subscales in a variety of pain situations (Burckhardt & Jones, 2003; Melzack, 1987). Furthermore, the SF-MPQ is highly reliable, with internal consistency values ranging from .73-.89 (Burckhardt & Jones). In the present study, the SF-MPQ had a Cronbach's alpha value of .82, indicating a high internal consistency.

Center for Epidemiological Studies & Depression Scale. The Center for Epidemiological Studies and Depression Scale (CES-D; Radloff, 1977) is a 20-item measure used to assess current depressive symptoms based on reports from the last week (see Appendix D). The CES-D evaluates both physical and cognitive symptoms of depression. All items begin with the phrase, "During the past week…". An example of an item that assesses physical symptoms is: "I had crying spells". An example of an item that assesses cognitive symptoms is: "I felt that people disliked me". Items are rated on a 4-point scale ranging from 0 (*Rarely or none of the time*) to 3 (*Most or all of the time*). Four of the items must be reverse-scored and scores are summed to obtain a total score of depressive symptoms. Scores on the CES-D rage from 0-60, with a higher score indicating greater depressive symptoms. The CES-D has been widely used and is shown to have high internal consistency and test-retest reliability (values ranging between .84 and .90 and between .48 and .67, respectively; Radloff, 1977). In this study, Cronbach's alpha was .87, indicating high internal consistency.

State-Trait Anxiety Inventory – Form Y. The State-Trait Anxiety Inventory (STAI; Spielberger, Gorusch, Lushene, Vagg, & Jacobs, 1983) is a 40-item measure of anxiety (see Appendix E). The STAI evaluates both state anxiety (i.e., situational anxiety) and trait anxiety (i.e., dispositional anxiety). For state anxiety, participants are asked how

they feel “right now at the present moment”, whereas for trait anxiety participants are asked how they “generally feel” on items such as “I feel calm”. Items are rated on a 4-point scale ranging from 1 (*Not at all*) to 4 (*Very much so*). Scores are summed to obtain a total score of state anxiety and a total score of trait anxiety. The STAI is shown to have high internal consistency, with Cronbach’s alpha values ranging from .89 to .92 (Speilberger et al., 1983). In the present study, the internal consistencies for the state anxiety and trait anxiety scales were high, with respective Cronbach’s alpha values for state anxiety and trait anxiety scales of .93 and 91.

Cold Pressor Task Apparatus. A cold pressor machine (CPM) was used to induce acute pain. The CPM (see Figure 3) is a re-circulating, double-bucket system with a built-in refrigeration unit. The CPM consists of a 22 inch by 29 inch outer casing that houses a 10 by 12 inch bucket which was filled to the brim with water. An internal thermostat held the water at a temperature of approximately 1-3^{o}C. A Fisher scientific thermometer was used to verify water temperature and to calibrate temperature settings. In the cold pressor task (CPT), participants immerse their non-dominant hand and forearm in the water with their fingers spread and touch the bottom of the water bucket. The cold pressor paradigm was designed to safely induce pain, has been used in many studies investigating acute pain, and is shown to be a highly reliable and valid measure of pain (Edens & Gil, 1995). Benefits of using a cold pressor to induce pain are that: 1) participants have control over their exposure to the stimulus and can remove their arm from the water if the sensation becomes too uncomfortable, and 2) discomfort associated with cold water rapidly dissipates once the forearm is removed (Edens & Gil, 1995).

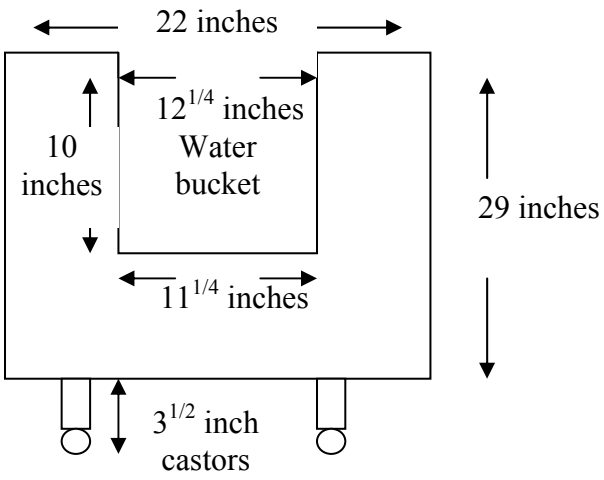

Figure 3. Cold pressor machine (CPM).

Psychophysiological Measurement Apparatus. Psychophysiological assessment included continuous monitoring of HR, respiration rate, and GSR. The apparatus used in this data collection consisted of a computer, an amplifier (BioPac™ Systems MP150), a battery pack, and sensors. The software used to capture the raw physiological data was Acqknowledge™ Version 3.8.2, while Noldus Observer XT™ was used to record video of the participant completing the task and to synchronize psychophysiological data recorded in Acqknowledge™ with the video input. The role of the amplifier was to amplify the sensor signals from the participant.

There were a total of five sensors used to measure the various arousal responses. Two sensors were used to measure HR and GSR, whereas only one was used to measure respiration rate. To measure respiration rate, a sensor attached to a stretchy fabric strap was placed around the participants' chest, at approximately the top of the rib cage. To measure HR, one sensor was placed on right side of the chest, approximately 2 inches below the collarbone. Another sensor was placed on the left side of the abdomen in

between the bottom of the rib cage and the hip bone. GSR was measured by placing a sensor on each of the participants' middle and ring fingers on their right hand. All sensors fed into a battery pack. Located on the battery pack were gain and filter controllers, which act to indicate and adjust or alter the strength of signal. There were three channels, one for HR (channel A), one for GSR (channel B), and one for respiration (channel C). For the present study, the gain on the battery pack was set to 1K for channel A, 10K for channel B, and 1K for channel C. The filters on the battery pack were set to .5 Hz for channel A, Direct Current (DC) for channel B, and DC for channel C, consistent with BioPac™ manufacturer recommendations.

Audiotaped Instructions. A standardized audiotape script was used to prompt participants when to verbally report their pain ratings during the pain induction and pain recovery periods. Instructions on the audiotape directed participants to put their arm in the water, to report pain at set intervals, and when to remove their arm from the water. The audiotape was played using a portable cassette player.

Procedure

All potential study participants completed the PCS as a pre-screening questionnaire that is included in a subject pool screening package completed yearly by first year undergraduate psychology students. From the potential participants completing the PCS pre-screening, individuals in the top third (i.e., PCS score ≥ 24) and the bottom third (i.e., PCS score ≤ 13) were identified as high and low catastrophizers respectively and were selected for participation in order to draw maximum differences in catastrophizing for comparisons. Although test-retest reliability for the PCS is reported as adequate (.70 for 10 weeks; Sullivan et al., 1995), as a confirmation check those

individuals selected for the study were contacted by email and/or telephone and asked to complete a second PCS to confirm their eligibility for the study (i.e., to ensure that their catastrophizing score still fell within the original top or bottom third of the distribution). Participants whose second PCS scores did not fall within the top or bottom ranges were excluded from participating in the study. Qualified participants were then scheduled for the experiment by email or telephone contact. For this first portion of the study (i.e., the confirmation check of the PCS score), participants were provided with a letter of information (Appendix F) and a consent form (Appendix G) before completing the PCS, and a debriefing form (Appendix H) following completion of the PCS.

Each participant was tested individually with the same female experimenter. Upon arrival, the experimenter greeted participants. To ensure that all participants received the same set of instructions, a script was utilized during all contact with participants (Appendix I). Participants read the letter of information (Appendix J) and consent form (Appendix K), had any questions answered, and then proceeded with the study following consent. Participants first completed demographic questions, the CES-D, and the STAI. These measures were presented in random order to control for order effects. The psychophysiological sensors were then attached to the participants. The experimenter explained what each of the sensors measured as each one was attached. The sensor measuring respiration rate was attached first, followed by the sensors used to measure HR, and finally the sensors to measure GSR.

Once connected to the sensors, participants were asked to remain still, relax, and breathe normally for 8-10 minutes, during which time the sensors became accustomed to measuring their psychophysiological responses (as directed by the Biopac^{TM}

recommendations). Immediately following this, participants were asked to complete a paced breathing exercise, in which they timed their breathing with a figure on a computer screen of a red bar moving up and down at a rate of 9 cycles per minute (e.g., Rottenberg, Wilhelm, Gross, & Gotlib, 2002). During the paced breathing exercise, participants inhaled as the red bar moved up and exhaled as the red bar moved down. The paced breathing exercise was performed to familiarize participants with the equipment, and to obtain a standardized breathing rate for all participants. A standardized breathing exercise is useful in that it allows these data to be further analyzed in a future study examining between-subjects differences in respiratory sinus arrhythmia (RSA; i.e., the variability of the heart rate for each respiratory cycle) among high and low catastrophizers.

Following the paced breathing task, participants were instructed to remain still, relax, and breathe normally for 3 minutes alone in the room while their psychophysiological responses were recorded for the baseline period. Following the 3-minute baseline period, participants completed the CPT by placing their left hand in the cold pressor machine and spreading their fingers to touch the bottom of the water bucket. Participants were instructed to leave their hand in the water for 1 minute, at which time they were instructed to remove their hand from the water.

During the pain induction period, participants were instructed to rate their pain intensity and pain unpleasantness on the rating scales ranging from 0 (*no pain/not unpleasant*) to 10 (*extreme pain/extremely unpleasant*) every 20 seconds when the audiotape indicated “report”. A rating scale placed at eye level was provided as a visual aid across from where the participants were seated.

Participants' pain reports were recorded and also captured on the video segment. If participants did not wish for their data to be used in further analyses, their videotaped data were deleted after ensuring that the physiological data did not have any artifacts that needed to be cross-checked with the video. After removing their arm from the cold pressor apparatus, participants were instructed to rest their arm on a towel on the edge of the CPM. Participants were instructed to remain still and breathe normally for 10 minutes while their psychophysiological responses were recorded throughout the pain recovery period. Participants were also asked to report pain intensity and unpleasantness each minute during this pain recovery period for a total of 10 reports. Following the pain recovery period, the sensors were removed and participants completed the SF-MPQ. Finally, participants were debriefed (Appendix L) and any questions they had were answered.

During the study, there was a second experimenter in an adjacent room where the data were recorded who was responsible for recording the psychophysiological data. The participants never saw this experimenter, but they were informed of his or her presence at the commencement of the study. The second experimenter began recording data for the paced breathing and baseline periods when the primary experimenter had left the room (after giving the instructions for the task) and stopped recording once the appropriate amount of time had elapsed. The pain induction and pain recovery periods were recorded together (i.e., there was no break in recording). After study completion, the pain induction and pain recovery periods were divided.

Data Reduction and Analysis

From the physiological data obtained, there were a number of data reduction steps. First, the raw electrocardiogram (ECG) data were filtered using a high pass filter of 2 Hz to eliminate any noise in the data (i.e., anything that might interfere with optimal peak detection). By using a high pass filter of 2 Hz, anything with a frequency less than 2 Hz was eliminated, thus eliminating slow waves such as respiration. If any abnormalities were seen in the data after filtering (e.g., large single increases or decreases in the HR) the waveforms were edited to eliminate the abnormalities (e.g., artifacts due to participant movement, coughing, etc.). During the baseline period, only 7% of cases required editing. During the pain/pain recovery periods, 25% of the cases required editing. However, of these cases that required editing, 77% required altering of only one or two peaks.

After editing the raw ECG data, the HR was calculated in Acqknowledge™ by using peak detection (i.e., detecting the number of R-waves to obtain the number of beats per minute). The psychophysiological data were then clipped to obtain the correct amount of time for each study period (i.e., baseline – 3 minutes, pain induction – 1 minute, and pain recovery – 10 minutes). Because of a lag in linking Acqknowledge™ to Noldus Observer XT™, the first 1.479 seconds were deleted from each segment from the baseline period and the pain/pain recovery period. The baseline period only required clipping at the end of that period (i.e., after 3 minutes to obtain a 3-minute long segment). Because the pain and pain recovery periods were recorded together, the two periods needed to be split into two segments at the appropriate points. To determine the appropriate start and stop points for the pain and pain recovery periods, the videos were viewed. The pain induction period was defined as the time when the participant first

placed their fingertips in the water and was terminated 1 minute afterwards. The pain recovery period was defined as the time from when the participant had entirely removed their hand from the water until 10 minutes from that point.

Once the data were clipped to the appropriate length, the average, minimum, and maximum HR and GSR values were obtained for each time period (i.e., baseline, pain induction, and pain recovery). This was done in Acqknowledge^{TM} by highlighting the data for the entire period and specifying the values to be calculated (i.e., average, minimum, and maximum). In addition, the average values for HR and GSR for each 20-second interval during the pain induction period were calculated. As well, for the pain recovery period, the average values for HR and GSR for each 1-minute interval during the 10-minute long pain recovery period were calculated.

After reducing the psychophysiological data, all data were examined for missing data. As mentioned previously, seven cases (i.e., pilot participants and/or participants with large portions of missing data) were deleted from all analyses. There were an additional two cases deleted from analyses involving psychophysiological data due to recording equipment malfunction. With regard to the psychological measures, there were only two cases that had missing data. One case had one item from the CES-D missing and one case had one item missing from the SF-MPQ, which were replaced with the mean for that variable (Tabachnick & Fidell, 2007).

For the pain intensity and pain unpleasantness variables, averages were taken for each time period (i.e., for pain induction and pain recovery). For the psychophysiological variables, reactivity (i.e., the difference between the variable during pain induction and

baseline) and deactivation (i.e., the difference between the variable during pain recovery and pain induction) variables were also created.

Normality was assessed for each dependent variable by dividing the skewness and kurtosis values by their respective standard errors with values exceeding an absolute value of three identified as violations of normality (Tabachnick & Fidell, 2007). Violations of normality were corrected using the appropriate transformations outlined in Tabachnick & Fidell (2007, p. 89). All analyses were performed using both the transformed and untransformed data[1]. No differences in significance were found between results using the transformed versus the untransformed data in any of the analyses. Thus, untransformed values were presented in this study to facilitate interpretation.

Outliers that had been identified in the boxplots produced by SPSS were identified as significant outliers if they were greater than three standard deviations from the mean, which is a conservative estimate of an extreme outlier (Tabachnick & Fidell, 2007). There was only one outlier in the data set. This outlier was for the average HR during the cold pressor task and it was replaced with the value of three standard deviations from the mean (Tabachnick & Fidell).

Finally, correlations among related dependent variables (specified in the results section) were also examined to check for potential multicollinearity or construct redundancy. Redundant constructs, defined as correlations between variables > .70, were reduced by using only one of the redundant variables (Tabachnick & Fidell, 2007).

Analyses pertaining to each of the hypotheses are described in the results section, which follows.

[1] Note that when conducting analyses which involved a time-series where one or more of the variables in the time-series required transformation, the other variables were also transformed to maintain consistency.

Chapter 3: Results

Descriptives

Table 1 displays the descriptive statistics for the full sample for each of the dependent variables. All values were within expected ranges given the population of young, healthy adults.

Table 1

Descriptive Statistics for Dependent Variables and Covariates

Variable	Range	*M*	*SD*	*N*
Average Pain Intensity (Pain Induction)	1.33-10.00	7.08	1.95	95
Average Pain Intensity (Pain Recovery)	0.00-4.30	0.85	0.78	95
Average Pain Unpleasantness (Pain Induction)	2.67-10.00	7.62	1.91	95
Average Pain Unpleasantness (Pain Recovery)	0.00-5.70	1.12	1.24	95
SF-MPQ Pain Rating Index – Total	6.00-45.00	19.90	8.26	95
SF-MPQ Pain Rating Index – Sensory	4.00-33.00	16.39	6.13	95
SF-MPQ Pain Rating Index – Affective	0.00-12.00	3.52	3.24	95
Average HR (bpm) (Baseline)	57.77-104.97	80.03	10.58	94
Average GSR (Baseline) (μmho)	0.45-39.34	13.89	8.77	94
Average HR (bpm) (Pain Induction)	68.20-135.14	92.08	14.40	94
Average GSR (μmho) (Pain Induction)	6.45-88.77	36.14	17.89	94
Average HR (bpm) (Pain Recovery)	56.77-103.46	74.47	10.61	94
Average GSR (μmho) (Pain Recovery)	3.30-55.48	18.48	11.22	94
Average HR Reactivity (bpm)	-12.51-43.22	11.87	11.18	93
Average GSR Reactivity (μmho)	3.95-72.89	22.41	12.20	93
Average HR Deactivation (bpm)	-50.43-5.25	-17.62	11.31	94
Average GSR Deactivation (μmho)	-40.32-1.72	-17.66	9.10	94
CES-D Total Score	0.00-38.00	11.73	7.81	95
STAI (State Form) Total Score	20.00-74.00	31.64	9.82	95
STAI (Trait Form) Total Score	21.00-62.00	36.18	9.88	95
Number of Previous Pain Experiences	0-9	1.78	1.61	95
Days Since Start of Last Menstruation*	0-32	14.04	7.92	49

Note. SF-MPQ = Short-Form McGill Pain Questionnaire; HR = heart rate; bpm = beats per minute; GSR = galvanic skin response; μmho = micromho; Reactivity = difference between average arousal during pain induction from baseline; Deactivation = difference between average arousal during pain recovery from pain induction; CES-D = Centre for Epidemiological Studies in Depression; STAI = State Trait Anxiety Inventory.

* This measure was for female participants only.

Correlations Between Dependent Variables and Covariates

As shown in Table 2, there were no significant correlations between the covariates and pain intensity or unpleasantness during the pain induction period. However, the positive correlation between trait anxiety and pain intensity during pain induction approached significance. There were significant small positive correlations between pain intensity/unpleasantness during the recovery period and measures of depression and state/trait anxiety. There were also significant small positive correlations between the SF-MPQ scales and measures of depression and state/trait anxiety. There were no significant correlations between the number of previous pain experiences and any of the dependent variables, nor were there correlations involving the days since the start of last menstruation. There were no significant correlations between any of the covariates and the arousal variables.

Thus, because there were relationships involving depression and anxiety and the pain report variables, depression and anxiety were covaried out of analyses involving pain report variables. Covariates were not used in any of the other analyses.

Table 2

Correlations Between Dependent Variables and Covariates

	CES-D	STAI State Form	STAI Trait Form	Number of Previous Pain Experiences	Days Since Start of Last Menstruation
Average Pain Intensity (Pain Induction)	.15	.19[a]	.16	.05	.14
Average Pain Intensity (Pain Recovery)	.34**	.27**	.33**	.13	.01
Average Pain Unpleasantness (Pain Induction)	.17	.19	.16	.12	.04
Average Pain Unpleasantness (Pain Recovery)	.27**	.31**	.32**	.19	.10
SF-MPQ Pain Rating Index – Total	.35**	.32**	.19**	.01	.11
SF-MPQ Pain Rating Index – Sensory	.29**	.24**	.21*	-.02	.12
SF-MPQ Pain Rating Index – Affective	.35**	.34**	.33**	.06	.05
Average HR (bpm) (Baseline)	.002	.05	.04	-.13	.04
Average GSR (μmho) (Baseline)	.003	-.03	-.005	.05	.03
Average HR (bpm) (Pain Induction)	.16	.17	.17	.06	-.06
Average GSR (μmho) (Pain Induction)	.01	.05	.02	.06	-.13
Average HR (bpm) (Pain Recovery)	.06	.14	.06	-.14	.09
Average GSR (μmho) (Pain Recovery)	.06	.04	.04	.07	-.11

Note. SF-MPQ = Short-Form McGill Pain Questionnaire; HR = heart rate; bpm = beats per minute; GSR = galvanic skin response; μmho = micromho; Reactivity = difference between average arousal during pain induction from baseline; Deactivation = difference between average arousal during pain recovery from pain induction; CES-D = Centre for Epidemiological Studies in Depression; STAI = State Trait Anxiety Inventory. For correlations involving CESD, STAI, and number of previous pain experiences: $N = 95$ for pain report variables, $N = 94$ for HR and GSR. For correlations involving days since start of last menstruation: $N = 49$ for pain report variables, $N = 48$ for HR and GSR.

* $p < .05$, ** $p < .01$, [a] $p < .06$.

Hypothesis 1

The first hypothesis suggested several associations between pain and psychophysiological activation that were detailed in the study model depicted in Figure 1. The results pertaining to each of these associations are described in the appropriate sections, which follow below.

Hypothesis 1a. Pearson correlation analyses were performed to assess the associations among all of the pain variables assessed during the pain induction and pain recovery periods (see Table 3). As mentioned in the data analysis section, pain intensity and pain unpleasantness showed strong positive correlations during pain induction and also during pain recovery (*r*s > .80), indicative of construct redundancy. To manage these redundant data, pain intensity was selected as the representative variable for use in all analyses (e.g., Tabachnick & Fidell, 2007). Pain intensity was chosen because of its ease of identification and estimation (Jensen & Karoly, 2001).

Pain intensity and unpleasantness during the pain induction period showed moderate positive associations with pain intensity and unpleasantness during the pain recovery period. Pain intensity and unpleasantness during the pain induction period also showed strong positive associations with retrospective reports of pain experienced during the pain induction period (i.e., the SF-MPQ total scale and sensory/affective subscales). Pain intensity and unpleasantness during the pain recovery period showed moderate to strong positive associations with the total scale and both the sensory and affective subscales of the SF-MPQ. In addition, the SF-MPQ total scale showed strong positive associations with the affective and sensory subscales of the SF-MPQ. Finally, the sensory and affective subscales of the SF-MPQ were also strongly positively correlated.

Table 3

Intercorrelations Among Pain Variables

	Pain 1	Pain 2	Pain 3	Pain 4	Pain 5	Pain 6	Pain 7
Pain 1. Average Pain Intensity (Pain Induction)	---						
Pain 2. Average Unpleasantness (Pain Induction)	.82**	---					
Pain 3. Average Pain Intensity (Pain Recovery)	.46**	.42**	---				
Pain 4. Average Unpleasantness (Pain Recovery)	.41**	.46**	.86**	---			
Pain 5. SF-MPQ PRI – Total	.72**	.67**	.52**	.48**	---		
Pain 6. SF-MPQ PRI – Sensory	.68**	.63**	.47**	.41**	.94**	---	
Pain 7. SF-MPQ PRI – Affective	.55**	.51**	.44**	.45**	.77**	.51**	---

Note. $N = 95$. SF-MPQ = Short-Form McGill Pain Questionnaire; PRI = pain response index.
** $p < .001$.

Pearson correlations were also performed among the three pain intensity reports during the pain induction period to examine the degree of association between these indices (see Table 4). As well, the average pain intensity was included in the correlation analysis to examine its association with each of the three pain intensity reports across the pain induction period. Due to the strong associations between pain intensity and pain unpleasantness during the pain induction period, only pain intensity variables were reported. Average pain intensity across the pain induction period showed a strong positive association with pain intensity reported at 20 seconds, 40 seconds, and 60 seconds. As well, the pain intensity ratings at each of the time points during the pain induction period were strongly positively correlated with one another.

Table 4

Intercorrelations Among Pain Intensity Variables During the Pain Induction Period

	Average Pain Intensity	Pain at 20s	Pain at 40s	Pain at 60s
Average Pain Intensity	---			
Pain at 20s	.93**	---		
Pain at 40s	.98**	.88**	---	
Pain at 60s	.93**	.74**	.92**	---

Note. $N = 95$; s = seconds.
** $p < .001$.

In addition, Pearson correlations were calculated to examine the associations among the average and the individual pain intensity reports recorded during the pain recovery period (see Table 5). Pain unpleasantness was also strongly associated with the average pain intensity during the pain recovery period, thus only pain intensity results were reported. Results of this analysis indicated a strong positive association between pain intensity during the pain recovery period and pain ratings at each of the 1-minute time points during the pain recovery period, with the exception of the last time point (pain intensity at 10 minutes), with which a moderate positive association was found. The pain intensity ratings for the first 9 minutes of the pain recovery period were all moderately to strongly correlated with one another. The last time point (i.e., pain intensity reported at 10 minutes after pain induction) was not correlated with the first two time points, was marginally to weakly positively correlated with the third, fourth, and fifth time points, and was moderately positively correlated with the last four time points.

Table 5

Intercorrelations Among Pain Intensity Variables During the Pain Recovery Period

	P1	P2	P3	P4	P5	P6	P7	P8	P9	P10	P11
P1. Average Pain Intensity	---										
P2. Pain 1 min	.83**	---									
P3. Pain 2 min	.89**	.82**	---								
P4. Pain 3 min	.89**	.68**	.85**	---							
P5. Pain 4 min	.86**	.54**	.73**	.85**	---						
P6. Pain 5 min	.86**	.50**	.65**	.79**	.95**	---					
P7. Pain 6 min	.78**	.43**	.51**	.61**	.76**	.86**	---				
P8. Pain 7 min	.64**	.34**	.32**	.42**	.57**	.68**	.89**	---			
P9. Pain 8 min	.64**	.33**	.34**	.42**	.54**	.67**	.83**	.90**	---		
P10. Pain 9 min	.64**	.33**	.34**	.43**	.54**	.67**	.78**	.81**	.96**	---	
P11. Pain 10 min	.34**	.13	.14	.19[a]	.19[a]	.24*	.30**	.33**	.40**	.42**	---

Note. $N = 95$. min = minutes.
* $p < .05$; ** $p < .01$; [a] $p < .07$.

Hypothesis 1b. A Pearson correlation analysis examined the associations among the averages for each period of the arousal variables (i.e., HR and GSR) as well as respective indices of reactivity (i.e., the difference between the average arousal during pain induction and baseline periods) and deactivation (i.e., the difference between the average arousal during the pain recovery and pain induction periods) (see Table 6)[2]. Average HR during baseline, pain induction, and pain recovery periods showed strong positive associations with each other. Similar to HR, average GSR during baseline, pain induction, and pain recovery periods were strongly positively correlated.

Reactivity and deactivation variables were also robustly associated with one another for GSR and HR. HR reactivity showed a strong negative association with HR deactivation. As well, greater HR reactivity and lower HR deactivation were associated with higher HR during the pain induction period, but not during baseline or pain recovery periods. As with HR, GSR reactivity showed a strong negative association with GSR deactivation. However, unlike HR, higher GSR reactivity and lower GSR deactivation were strongly correlated with average GSR during baseline, pain induction, and pain recovery periods.

Upon examining the relationships between HR and GSR, there were no significant correlations, with the exception of the weak positive correlation between HR during pain recovery and GSR during baseline (see Table 6).

[2]Please note that correlations between the maximum and minimum values were also calculated for HR and GSR. These variables were very strongly correlated with the averages for each of the variables (*r*s > .80), indicating construct redundancy. Because they were highly correlated, only the results for the average score during each time period were presented and used in further analyses for clarity.

Table 6

Intercorrelations Among Psychophysiological Variables

	HR1	HR2	HR3	HR4	HR5	GSR1	GSR2	GSR3	GSR4	GSR5
HR 1. Average HR (bpm) (Baseline)	---									
HR 2. Average HR (bpm) (Pain Induction)	.64**	---								
HR 3. Average HR (bpm) (Pain Recovery)	.92**	.63**	---							
HR 4. HR Reactivity (bpm)	-.13	.68**	-.03	---						
HR 5. HR Deactivation (bpm)	.04	-.68**	.14	-.92**	---					
GSR 1. Average GSR (μmho) (Baseline)	.16	.16	.21*	.05	-.004	---				
GSR 2. Average GSR (μmho) (Pain Induction)	.04	.10	.09	.11	-.05	.79**	---			
GSR 3. Average GSR (μmho) (Pain Recovery)	.05	.07	.11	.06	.01	.79**	.90**	---		
GSR 4. GSR Reactivity (μmho)	-.06	.05	-.03	.13	-.01	.44**	.90**	.76**	---	
GSR 5. GSR Deactivation (μmho)	-.005	-.11	-.04	-.15	.11	-.59**	-.85**	-.54**	-.83**	---

Note. $N = 93$ for correlations between anticipation period and pain period, $N = 94$ for all other correlations. HR = heart rate; bpm = beats per minute; GSR = galvanic skin response; μmho = micromho; Reactivity = difference between average arousal during pain induction from baseline; Deactivation = difference between average arousal during pain recovery from pain induction.
* $p < .05$; ** $p < .01$.

Although there were no relationships between HR and GSR using the correlational method described above, Lazarus et al. (1963) found that conducting intra-individual correlations as opposed to inter-individual correlations produced significant correlations between different measures of autonomic indices. The inter-individual method is correlating the average for one period (e.g., the recovery period) for HR and GSR for each subject. The intra-individual method (suggested by Lazarus et al.) splits up the period into smaller sections, averages each section across participants, and then correlates the variables at each of the time points. In the present study, there were 10 time points for the pain recovery period for which this method could be applied. Indeed, after taking the average across subjects and conducting a correlation to assess the relationship between HR and GSR using the averages for each of the minute-long time periods, a strong negative association between HR and GSR was found, $r(8) = -.75$, $p = .01$.

Hypothesis 1c. Pearson correlation analyses examined the associations between psychophysiological variables (i.e., HR and GSR) and pain variables during the pain induction period. Results of the correlation analyses showed that pain rating variables were not correlated with the raw HR variables (see Table 7). However, there were positive associations that approached significance between average pain intensity and average HR for the last 20-second interval of the pain induction period (i.e., 40-60 seconds). Positive associations between pain intensity rated at 40 seconds during pain induction and average HR for the last 20-second interval of the pain induction period also approached significance.

With regard to the HR reactivity variables, there were significant associations with several pain variables (see Table 7). Specifically, average HR reactivity (i.e.,

average HR during pain induction less the average HR during baseline) was positively associated with pain intensity reported at 20 seconds into the pain induction period. Positive associations between average HR reactivity and average pain intensity and pain intensity reported at 60 seconds into the pain induction period all approached significance. Furthermore, higher HR reactivity for the last 20-second interval (i.e., average HR during 40-60 seconds of pain induction less the average HR during baseline) was positively associated with higher average pain intensity and pain intensity at 20, 40, and 60 seconds.

Table 7

Correlations Between Pain Variables and HR During the Pain Induction Period

	Average Pain Intensity	Pain at 20s	Pain at 40s	Pain at 60s	SF-MPQ Total
Average HR	.11	.10	.13	.08	.10
Average HR 0-20s	.05	.06	.07	.01	.04
Average HR 20-40s	.08	.07	.10	.05	.09
Average HR 40-60s	.18[a]	.16	.20[b]	.15	.16
Average HR Reactivity	.20[c]	.23*	.18[a]	.14	.15
HR Reactivity 0-20s	.11	.16	.09	.06	.06
HR Reactivity 20-40s	.14	.17	.14	.10	.12
HR Reactivity 40-60s	.27**	.29**	.26*	.22*	.21*

Note. $N = 93$ for correlations involving HR reactivity variables, $N = 94$ for all other correlations. HR = heart rate; s = seconds; Average HR Reactivity = difference between average HR during pain induction from baseline; HR Reactivity 0-20, 20-40, 40-60 = difference between average HR for the interval from average HR during baseline. HR was measured in beats per minute.
[a] $p = .08$; [b] $p = .05$; [c] $p = .06$.
* $p < .05$; ** $p < .01$.

The Pearson correlation analysis between pain report variables and GSR indicated no significant associations (see Table 8). Even when the GSR reactivity variable was used, there were no significant correlations between pain and GSR.

Table 8

Correlations Between Pain Variables and Galvanic Skin Response (GSR) During the Pain Induction Period

	Average Pain Intensity	Pain at 20s	Pain at 40s	Pain at 60s	SF-MPQ Total
Average GSR	.009	.01	.006	.008	.01
Average GSR 0-20s	-.009	-.007	-.01	-.007	.006
Average GSR 20-40s	.008	.01	.005	.007	.009
Average GSR 40-60s	.03	.03	.02	.02	.02
Average GSR Reactivity	.03	.02	.03	.05	.03
GSR Reactivity 0-20s	.009	-.01	.008	.03	.02
GSR Reactivity 20-40s	.03	.02	.03	.05	.02
GSR Reactivity 40-60s	.06	.04	.06	.07	.04

Note. $N = 93$ for correlations involving GSR reactivity variables, $N = 94$ for all other correlations. GSR = galvanic skin response; s = seconds; Average GSR Reactivity = difference between average GSR during pain induction from baseline; GSR Reactivity 0-20, 20-40, 40-60 = difference between average GSR for the interval from average GSR during baseline. GSR was measured in micromhos (μmho).

A correlation analysis was also conducted to examine the relationship between arousal and pain intensity during the pain recovery period. Due to the large number of correlations, a critical value of .001 was used to facilitate interpretation. As shown in Table 9, none of the correlations between HR and pain during the recovery period were significant at the .001 level. Likewise, as shown in Table 10, none of the correlations between GSR and pain during the recovery period were significant at the .001 level.

Table 9

Intercorrelations Between Pain Variables and HR During the Pain Recovery Period

	Average Pain Intensity	Pain 1 min	Pain 2 min	Pain 3 min	Pain 4 min	Pain 5 min	Pain 6 min	Pain 7 min	Pain 8 min	Pain 9 min	Pain 10 min
Average HR	-.01	.02	-.02	-.05	-.07	-.09	-.06	-.03	.03	.06	.11
Average HR 0-1 min	.09	.17	.07	.04	.03	-.01	.01	.04	.04	.07	.05
Average HR 1-2 min	.02	.07	.01	.-.03	-.04	-.07	-.03	-.004	.04	.06	.09
Average HR 2-3 min	-.02	.03	-.02	-.06	-.06	-.08	-.06	-.02	.03	.06	.06
Average HR 3-4 min	-.05	.004	-.04	-.08	-.10	-.12	-.10	-.06	-.003	.06	.06
Average HR 4-5 min	-.01	.01	-.01	-.04	-.06	-.08	-.04	-.03	.04	.07	.11
Average HR 5-6 min	-.03	-.02	-.03	-.06	-.07	-.09	-.06	-.04	.03	.06	.11
Average HR 6-7 min	-.04	-.02	-.06	-.06	-.09	-.11	-.08	-.04	.03	.05	.14
Average HR 7-8 min	-.03	-.01	-.04	-.06	-.10	-.11	-.08	-.04	.03	.06	.18
Average HR 8-9 min	-.06	-.04	-.06	-.09	-.11	-.12	-.09	-.05	.02	.05	.18
Average HR 9-10 min	-.02	.01	-.03	-.07	-.09	-.10	-.06	-.03	.04	.07	.14
Average HR Deactivation	-.08	-.17	-.11	-.02	-.06	-.01	-.05	-.04	.01	.05	.16
HR Deactivation 0-1 min	-.04	.02	.02	-.08	-.05	-.07	-.02	-.04	-.02	-.09	-.13
HR Deactivation 1-2 min	.04	.11	.08	.01	.03	-.005	.03	.02	-.02	-.06	-.14
HR Deactivation 2-3 min	.08	.16	.11	.03	.05	.004	.05	.03	-.01	-.06	-.11
HR Deactivation 3-4 min	.11	.18	.13	.05	.08	.04	.09	.06	.02	-.02	-.11
HR Deactivation 4-5 min	.07	.18	.10	.02	.04	-.01	.04	.04	-.02	-.06	-.16
HR Deactivation 5-6 min	.09	.20	.11	.03	.06	.01	.05	.05	-.01	-.05	-.15
HR Deactivation 6-7 min	.09	.19	.13	.03	.07	.02	.06	.05	-.002	-.04	-.17
HR Deactivation 7-8 min	.09	.19	.12	.03	.07	.03	.07	.05	-.01	-.05	-.21*
HR Deactivation 8-9 min	.11	.22*	.14	.06	.09	.03	.08	.05	.004	-.04	-.21*
HR Deactivation 9-10 min	.08	.18	.12	.04	.08	.02	.05	.04	-.02	-.06	.18

Note. $N = 94$. HR = heart rate; min = minutes; Average HR Deactivation = difference between average HR during pain recovery from pain induction; HR Reactivity for each minute = difference between average HR for the interval from average HR during pain induction. HR was measured in beats per minute.
* $p < .05$.

Table 10

Intercorrelations Between Pain Variables and Galvanic Skin Response (GSR) During the Pain Recovery Period

	Average Pain Intensity	Pain 1 min	Pain 2 min	Pain 3 min	Pain 4 min	Pain 5 min	Pain 6 min	Pain 7 min	Pain 8 min	Pain 9 min	Pain 10 min
Average GSR	-.09	-.11	-.06	-.03	-.01	-.06	-.18	-.13	-.14	-.14	.11
Average GSR 0-1 min	-.13	-.14	-.11	-.06	-.04	-.08	-.19	-.15	-.16	-.16	.04
Average GSR 1-2 min	-.12	-.14	-.10	-.05	-.03	-.08	-.19	-.15	-.16	-.15	.07
Average GSR 2-3 min	-.11	-.12	-.09	-.04	-.02	-.08	-.19	-.14	-.15	-.15	.08
Average GSR 3-4 min	-.07	-.09	-.04	-.01	.02	-.04	-.18	-.12	-.14	-.14	.08
Average GSR 4-5 min	-.07	-.10	-.05	-.02	.004	-.05	-.18	-.12	-.14	-.14	.11
Average GSR 5-6 min	-.08	-.11	-.05	-.02	.001	-.06	-.18	-.12	-.13	-.14	.12
Average GSR 6-7 min	-.07	-.10	-.04	-.01	.01	-.05	-.18	-.12	-.13	-.13	.12
Average GSR 7-8 min	-.06	-.10	-.03	-.005	.01	-.05	-.17	-.12	-.12	-.13	.14
Average GSR 8-9 min	-.06	-.10	-.03	-.005	.01	-.05	-.17	-.11	-.12	-.13	.18
Average GSR 9-10 min	-.08	-.12	-.06	-.02	-.03	-.09	-.17	-.12	-.13	-.14	.18
Average GSR Deactivation	.19	.10	.18	.21*	.17	.17	.17	.09	.14	.15	.12
GSR Deactivation 0-1 min	-.16	-.08	-.14	-.20	-.15	-.17	.17	.09	-.12	-.15	-.04
GSR Deactivation 1-2 min	-.17	-.09	-.14	-.19	-.14	-.16	-.18	-.08	-.13	-.15	-.07
GSR Deactivation 2-3 min	-.17	-.10	-.15	-.19	-.14	-.16	-.18	-.09	-.14	-.15	-.08
GSR Deactivation 3-4 min	-.20*	-.13	-.20	-.22*	-.19	-.19	-.17	-.09	-.13	-.15	-.08
GSR Deactivation 4-5 min	-.20	-.12	-.19	-.21*	-.17	-.17	-.17	-.09	-.14	-.15	-.11
GSR Deactivation 5-6 min	-.19	-.10	-.19	-.20*	-.16	-.16	-.17	-.09	-.14	-.15	-.12
GSR Deactivation 6-7 min	-.20	-.10	-.19	-.21*	-.17	-.17	-.16	-.09	-.14	-.15	-.12
GSR Deactivation 7-8 min	-.20	-.11	-.20	-.22*	-.17	-.17	-.16	-.09	-.14	-.14	-.15
GSR Deactivation 8-9 min	-.20	-.10	-.20	-.21*	-.17	-.16	-.16	-.09	-.14	-.14	-.20
GSR Deactivation 9-10 min	-.17	-.09	-.17	-.19	-.12	-.12	-.16	-.09	-.13	-.14	-.18

Note. $N = 94$. GSR = galvanic skin response; min = minutes; Average GSR Deactivation = difference between average GSR during pain recovery from pain induction; GSR Reactivity for each minute = difference between average GSR for the interval from average GSR during pain induction. GSR was measured in micromhos (μmho). * $p < .05$.

Hypothesis 1d. Pearson correlation analyses examined whether the relationship between arousal during the baseline period was associated with pain during the pain induction period. Results of the correlation analysis indicated that average HR during the baseline period was not associated with average pain intensity reported during the pain induction period, $r(92) = -.05$, $p = .63$. Also, average GSR during the baseline period was not associated with average pain intensity reported during the pain induction period, $r(92) = -.04$, $p = .74$. Although the raw data were not associated, when considering the HR reactivity, there was a positive association between HR reactivity and average pain intensity reported during the pain induction period that approached significance (refer to Table 7). However, there was no association between GSR reactivity and pain intensity reported during the pain induction period (refer to Table 8).

Correlations also examined the relationship between arousal during the pain induction period and pain intensity during the pain recovery period. Average HR during the pain induction period was not associated with average pain intensity reported during the pain recovery period, $r(92) = .05$, $p = .64$. As well, average GSR during the pain induction period was not associated with average pain intensity reported during the pain recovery period, $r(92) = -.15$, $p = .15$. There were no significant associations between HR or GSR deactivation and pain intensity during the pain recovery period (see Tables 9 and 10, respectively).

Hypothesis 1e. The final correlation analysis examined the association of average pain intensity reported during the pain induction period with HR and GSR recorded during the recovery period. Results showed that average pain intensity during the pain induction period was not associated with average HR during the recovery period,

$r(92) = -.04$, $p = .71$. Further, average pain intensity during the pain induction period was also not associated with average GSR during the recovery period, $r(92) = .01$, $p = .93$.

Hypothesis 2

To examine the hypothesis that catastrophizing and gender would be related to greater pain intensity, a 2 (Catastrophizing Group) x 2 (Gender) x 3 (Time) mixed-model ANOVA was conducted using pain intensity ratings at each of the 20-second intervals during the pain induction period as the dependent variable.

As can be seen from Figure 4, results of the ANOVA indicated a significant main effect of catastrophizing (see Table 11). To follow up the significant main effect of catastrophizing, pairwise comparisons were examined. Pairwise comparisons revealed that high catastrophizers reported greater pain intensity than low catastrophizers at 20 seconds, 40 seconds, and 60 seconds (see Table 12).

Table 11

ANOVA for Pain Intensity over the Pain Induction Period

Source	*df*	*F*	Partial η^2	*p*
Between subjects				
Catastrophizing	1	17.41	.16	< .001
Gender	1	12.26	.12	.001
Catastrophizing x Gender	1	0.46	.005	.50
Error	91			
Within subjects				
Time	1.29	154.80	.63	< .001
Time x Catastrophizing	1.29	1.49	.02	.23
Time x Gender	1.29	0.68	.01	.44
Time x Catastrophizing x Gender	1.29	1.50	.01	.23
Error	117.19			

Note. df for within subjects analyses were corrected using Greenhouse-Geisser correction due to violations of sphericity assumption.

Table 12

Pairwise Comparisons of Pain Intensity Ratings Between High and Low Catastrophizers Over the Pain Induction Period

Time	Level of Catastrophizing	*M*	*SD*	*F*	Partial η^2	*p*
20 seconds	High	6.78	1.89	15.06	.14	< .001
	Low	5.12	2.22			
40 seconds	High	8.20	1.50	18.68	.17	< .001
	Low	6.56	2.11			
60 seconds	High	8.69	1.33	12.08	.12	.001
	Low	7.36	2.16			

Note. df(1, 91) for all comparisons.

Results of the ANOVA also indicated a significant main effect of gender (see Table 11). Pairwise comparisons revealed that women reported greater pain intensity than men at 20 seconds, 40 seconds, and 60 seconds (see Table 13).

Table 13

Pairwise Comparisons of Pain Intensity Ratings Between Women and Men Over the Pain Induction Period

Time	Gender	*M*	*SD*	*F*	Partial η^2	*p*
20 seconds	Women	6.07	2.47	6.64	.07	.01
	Men	5.25	2.23			
40 seconds	Women	8.00	1.73	12.98	.12	.001
	Men	6.57	2.06			
60 seconds	Women	8.65	1.65	13.97	.13	< .001
	Men	7.23	1.96			

Note. df(1, 91) for all comparisons.

Using the Greenhouse-Geisser correction due to violation of the assumption of sphericity, results of this ANOVA indicated a significant main effect of time (see Table

11). Helmert contrasts indicated that pain intensity reported at 20 seconds into pain induction period ($M = 5.91$, $SD = 2.22$) was significantly lower than pain intensity reported at later times, $F(1, 91) = 183.32$, $p < .001$, partial $\eta^2 = .67$. Furthermore, pain intensity reported at 40 seconds ($M = 7.34$, $SD = 2.01$) was significantly lower than pain intensity reported at 60 seconds ($M = 7.99$, $SD = 1.93$), $F(1, 91) = 61.05$, $p < .001$, partial $\eta^2 = .40$.

The interaction between catastrophizing and gender was not significant. There were also no significant two- or three-way interactions between time, catastrophizing, and gender (see Table 11).

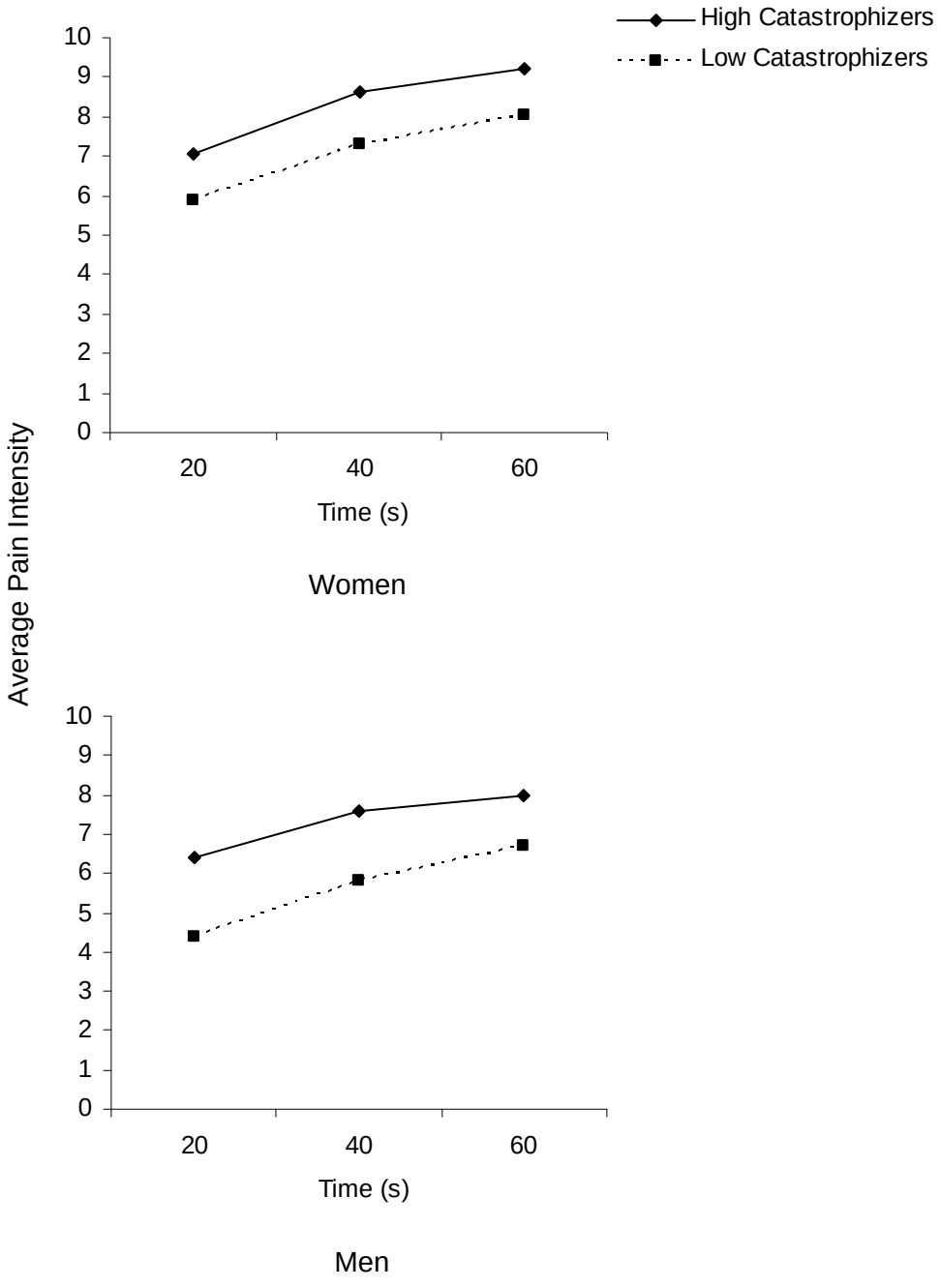

Figure 4. Pain intensity as a function of catastrophizing and gender over the pain induction period.

To determine whether the effects of the previous analysis existed when controlling for depression and anxiety, a 2 (Catastrophizing Group) x 2 (Gender) x 3 (Time) mixed-model ANCOVA was conducted using depression and anxiety as covariates. Results of the ANCOVA indicated that catastrophizing and gender were significantly related to pain intensity even while controlling for depression and anxiety (see Table 14). As before, pairwise comparisons revealed that at all three time points during the pain induction period high catastrophizers reported higher pain intensity than low catastrophizers (20 seconds: $F(1, 88) = 12.27$, $p = .001$, partial $\eta^2 = .12$; 40 seconds: $F(1, 88) = 18.84$, $p < .001$, partial η^2 .18; and 60 seconds: $F(1, 88) = 13.14$, $p < .001$, partial $\eta^2 = .13$) and women reported higher pain intensity than men (20 seconds: $F(1, 88) = 6.73$, $p = .01$, partial $\eta^2 = .07$; 40 seconds: $F(1, 88) = 12.47$, $p = .001$, partial η^2 .12; and 60 seconds: $F(1, 88) = 13.37$, $p < .001$, partial $\eta^2 = .13$).

Furthermore, results of the ANCOVA indicated that time still predicted pain intensity even while controlling for depression and anxiety (see Table 14). Helmert contrasts indicated that pain intensity reported at 20 seconds into pain induction period was significantly lower than pain intensity reported at later times, $F(1, 88) = 4.61$, $p < .04$, partial $\eta^2 = .05$. However, pain intensity reported at 40 seconds was no longer significantly different from pain intensity reported at 60 seconds, $F(1, 88) = 1.26$, $p = .26$, partial $\eta^2 = .01$.

Table 14

ANCOVA for Pain Intensity Over the Pain Induction Period

Source	*df*	*F*	Partial η^2	*p*
Between subjects				
CES-D	1	0.12	.001	.73
STAI (State Form)	1	1.76	.02	.19
STAI (Trait Form)	1	1.31	.02	.26
Catastrophizing	1	16.64	.16	< .001
Gender	1	11.93	.12	.001
Catastrophizing x Gender	1	1.03	.01	.31
Error	88			
Within subjects				
Time	1.28	3.81	.04	.04
Time x CES-D	1.28	2.19	.02	.14
Time x STAI (State Form)	1.28	0.20	.002	.72
Time x STAI (Trait Form)	1.28	0.54	.006	.50
Time x Catastrophizing	1.28	0.74	.008	.42
Time x Gender	1.28	0.45	.005	.55
Time x Catastrophizing x Gender	1.28	1.50	.02	.23
Error	112.94			

Note. CES-D = Center for Epidemiological Studies Depression Scale, STAI = State-Trait Anxiety Inventory. *df* for within subjects analyses were corrected using Greenhouse-Geisser correction due to violations of sphericity assumption.

Hypothesis 3

To investigate the effects of catastrophizing and gender on pain intensity during the pain recovery period, a 2 (Catastrophizing Group) x 2 (Gender) x 10 (Time) mixed-model ANOVA was conducted to evaluate the effects of catastrophizing and gender across the 10 minutes of the pain recovery period. As can be seen from Table 15, results of the ANOVA indicated a significant main effect of catastrophizing, a significant main effect of gender, and a significant main effect of time. The two-way interactions between catastrophizing and time and between gender and time were also significant. However the three-way interaction between catastrophizing, gender, and time was not significant. See Figure 5 for a depiction of the results.

Table 15

ANOVA for Pain Intensity Over the Pain Recovery Period

Source	*df*	*F*	Partial η^2	*p*
Between subjects				
Catastrophizing	1	10.96	.11	.001
Gender	1	22.39	.20	< .001
Catastrophizing x Gender	1	1.91	.02	.17
Error	91			
Within subjects				
Time	2.20	220.32	.71	< .001
Time x Catastrophizing	2.20	9.94	.10	< .001
Time x Gender	2.20	11.32	.11	< .001
Time x Catastrophizing x Gender	2.20	0.81	.10	.46
Error	199.90			

Note. df for within subjects analyses were corrected using Greenhouse-Geisser correction due to violations of sphericity assumption.

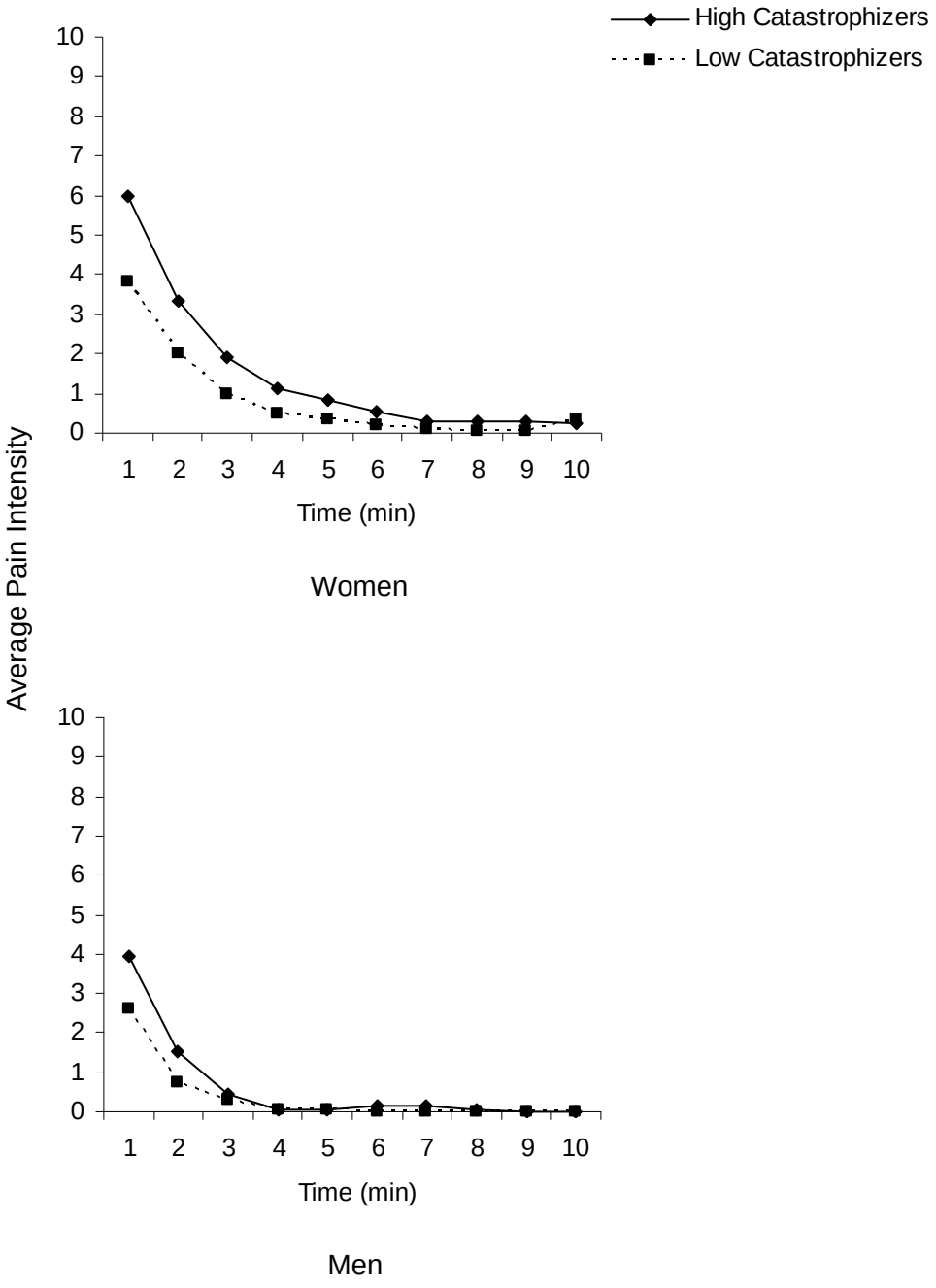

Figure 5. Pain intensity as a function of catastrophizing and gender over the pain recovery period.

To follow-up the significant two-way interaction between catastrophizing and time, pairwise comparisons between high and low catastrophizers at each time point were examined. Because 10 comparisons were made, the alpha was adjusted to .005 (i.e., .05/10) using a Bonferroni correction to correct for Type I error. As shown in Table 16, pairwise comparisons revealed that high catastrophizers reported higher pain intensity than low catastrophizers until 2 minutes after removing their arm from the cold pressor.

Table 16

Pairwise Comparisons of Pain Intensity Ratings Between High and Low Catastrophizers Over the Pain Recovery Period

Time	Level of Catastrophizing	*M*	*SD*	*F*	Partial η^2	*P*
1 minute	High	5.11	2.34	17.72	.16	< .001
	Low	3.22	1.94			
2 minutes	High	2.58	1.99	9.92	.10	.002
	Low	1.38	1.56			
3 minutes	High	1.27	1.45	5.24	.05	.02
	Low	0.62	1.03			
4 minutes	High	0.69	1.14	3.82	.04	.05
	Low	0.26	0.69			
5 minutes	High	0.49	0.97	2.36	.02	.13
	Low	0.20	0.54			
6 minutes	High	0.38	0.86	3.54	.04	.06
	Low	0.10	0.36			
7 minutes	High	0.24	0.61	4.42	.05	.04
	Low	0.04	0.20			
8 minutes	High	0.18	0.54	3.24	.03	.08
	Low	0.02	0.14			
9 minutes	High	0.16	0.52	2.31	.02	.13
	Low	0.02	0.14			
10 minutes	High	0.13	0.50	0.07	.001	.79
	Low	0.16	1.00			

Note. $df(1, 91)$ for all comparisons.

To follow up the significant two-way interaction between gender and time, pairwise comparisons were made between women and men for each time point. Again, significance was evaluated against an alpha of .005 to control for multiple comparisons. As shown in Table 17, pairwise comparisons indicated that women reported greater pain than men until 5 minutes after removing their arm from the cold pressor.

Table 17

Pairwise Comparisons of Pain Intensity Ratings Between Women and Men Over the Pain Recovery Period

Time	Gender	*M*	*SD*	*F*	Partial η^2	*P*
1 minute	Women	4.92	2.36	15.59	.15	< .001
	Men	3.18	1.93			
2 minutes	Women	2.69	2.05	20.82	.19	< .001
	Men	1.09	1.16			
3 minutes	Women	1.43	1.51	21.20	.19	< .001
	Men	0.34	0.53			
4 minutes	Women	0.82	1.18	19.28	.18	< .001
	Men	0.05	0.21			
5 minutes	Women	0.59	0.98	12.89	.12	.001
	Men	0.05	0.21			
6 minutes	Women	0.37	0.82	4.85	.05	.03
	Men	0.07	0.33			
7 minutes	Women	0.20	0.53	1.57	.02	.21
	Men	0.07	0.33			
8 minutes	Women	0.16	0.50	2.69	.03	.10
	Men	0.02	0.15			
9 minutes	Women	0.16	0.50	4.21	.04	.04
	Men	0.00	0.00			
10 minutes	Women	0.27	1.08	2.78	.03	.10
	Men	0.00	0.00			

Note. $df(1, 91)$ for all comparisons.

To determine whether these effects existed when controlling for depression and anxiety, a 2 (Catastrophizing Group) x 2 (Gender) x 10 (Time) mixed-model ANCOVA was conducted using depression and anxiety as covariates. As depicted in Table 18, results of the ANCOVA indicated that the main effects of gender and time remained significant when controlling for depression and anxiety, but the main effect of catastrophizing was no longer significant. However, the two-way interaction between time and catastrophizing remained significant, as did the two-way interaction between time and gender. The follow-up pairwise comparisons to the significant two-way interactions yielded similar results as for the ANOVA. As before, the alpha was adjusted to .005 to correct for multiple comparisons. Specifically, results indicated that high catastrophizers rated their pain marginally significantly higher than low catastrophizers for the first minute, but not thereafter, $F(1, 88) = 7.21$, $p = .009$, partial $\eta^2 = .08$, ps $> .12$ for all others. For the time by gender interaction, as with the ANOVA, follow-up comparisons indicated that women rated their pain higher than men until 5 minutes after the pain induction period Fs$(1, 88) > 12.66$, ps $\leq .001$; ps $> .03$ for all other comparisons.

Table 18

ANCOVA for Pain Intensity Over the Pain Recovery Period

Source	*df*	*F*	Partial η^2	*p*
Between subjects				
CES-D	1	1.62	.02	.21
STAI (State Form)	1	0.001	< .001	.97
STAI (Trait Form)	1	0.13	.001	.72
Catastrophizing	1	2.18	.02	.14
Gender	1	22.38	.20	< .001
Catastrophizing x Gender	1	1.96	.02	.17
Error	88			
Within subjects				
Time	2.16	7.56	.08	< .001
Time x CES-D	2.16	0.96	.01	.39
Time x STAI (State Form)	2.16	1.63	.02	.20
Time x STAI (Trait Form)	2.16	0.27	.003	.78
Time x Catastrophizing	2.16	4.53	.05	.01
Time x Gender	2.16	11.60	.12	< .001
Time x Catastrophizing x Gender	2.16	0.91	.01	.41
Error	189.78			

Note. CES-D = Center for Epidemiological Studies Depression Scale, STAI = State-Trait Anxiety Inventory. *df* for within subjects analyses were corrected using Greenhouse-Geisser correction due to violations of sphericity assumption.

Hypothesis 4

To investigate the relationship between catastrophizing, gender, and the retrospective report of pain as assessed with the SF-MPQ, a 2 (Catastrophizing Group) x 2 (Gender) between-subjects ANOVA was conducted with the SFMPQ Total Scale (PRI-Total) as the dependent variable. As shown in Table 19, there was a significant main effect of catastrophizing, such that high catastrophizers (M = 24.28, SD = 7.85) reported higher pain than low catastrophizers (M = 15.96, SD = 6.48). There was also a significant main effect of gender, such that women (M = 22.13, SD = 7.82) reported greater pain after task completion than men (M = 17.31, SD = 8.08). As with previous analyses, the

interaction between catastrophizing and gender was not significant. See Figure 6 for a depiction of the results.

Table 19

ANOVA for Short-Form McGill Pain Questionnaire Pain Rating Index – Total

Source	*df*	*F*	Partial η^2	*p*
Catastrophizing	1	32.04	.26	< .001
Gender	1	8.36	.08	.005
Catastrophizing x Gender	1	0.48	.005	.49
Error	91			

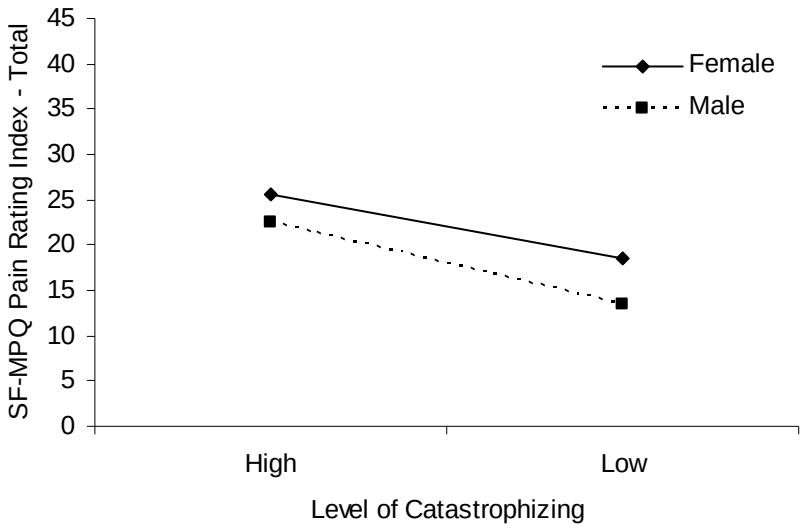

Figure 6. Short-Form McGill Pain Questionnaire Pain Rating Index - Total as a function of catastrophizing and gender.

To determine whether these effects existed when controlling for depression and anxiety, a 2 (Catastrophizing Group) x 2 (Gender) between-subjects ANCOVA was conducted using depression and anxiety as covariates. Results of the ANCOVA indicated significant main effects of catastrophizing (high catastrophizers > low catastrophizers) and gender (women > men) even when controlling for depression and anxiety, but the interaction between catastrophizing and gender was not significant (see Table 20).

Table 20

ANCOVA for Short-Form McGill Pain Questionnaire Pain Rating Index – Total

Source	*df*	*F*	Partial η^2	*p*
CES-D	1	1.75	.02	.19
STAI (State Form)	1	2.52	.03	.12
STAI (Trait Form)	1	3.04	.03	.08
Catastrophizing	1	19.70	.18	< .001
Gender	1	8.79	.09	.004
Catastrophizing x Gender	1	1.39	.02	.24
Error	88			

Note. CES-D = Center for Epidemiological Studies Depression Scale, STAI = State-Trait Anxiety Inventory.

A MANOVA was also conducted to evaluate whether there were different effects for sensory and affective dimensions of pain report. Catastrophizing and gender were the independent variables and sensory and affective dimensions of pain report from the SF-MPQ were the dependent variables. Results of the MANOVA indicated that catastrophizing and gender were significantly related to the two dimensions of pain report (see Table 21). The interaction between catastrophizing and gender was not significant.

Table 21

MANOVA for Short-Form McGill Pain Questionnaire Pain Rating Index Sensory and Affective Subscales

Source	*df*	*F*	Partial η^2	*p*
Catastrophizing	2	17.94	.28	< .001
Gender	2	7.09	.14	.001
Catastrophizing x Gender	2	0.49	.01	.62
Error	90			

Note. Dependent variables were the Short-Form McGill Pain Questionnaire sensory and affective subscales.

As shown in Table 22, univariate tests were conducted to follow-up the significant multivariate effects. Results indicated that for the sensory dimension of pain (see Figure 7), there was a significant effect of catastrophizing, such that high catastrophizers (M = 19.06, SD = 5.51) reported significantly higher sensory pain than low catastrophizers (M = 13.98, SD = 5.69). There was also a significant effect of gender, such that women (M = 18.41, SD = 5.80) reported significantly higher sensory pain than men (M = 14.05, SD = 5.71). However, with regard to the affective dimension of pain (see Figure 8), only catastrophizing had a significant effect, such that high catastrophizers (M = 5.22, SD = 3.21) reported significantly higher affective pain than low catastrophizers (M = 1.98, SD = 2.41). The main effect of gender on affective report of pain was not significant. None of the interactions were significant for either the sensory or affective dimensions of pain.

Table 22

Univariate Results for Short-Form McGill Pain Questionnaire Pain Rating Index Sensory and Affective Subscales

Source	DV	*df*	*F*	Partial η^2	*P*
Catastrophizing	Sensory Index	1	19.85	.18	< .001
	Affective Index	1	30.02	.25	< .001
Gender	Sensory Index	1	12.98	.12	.001
	Affective Index	1	0.12	.001	.73
Catastrophizing x Gender	Sensory Index	1	0.84	.01	.36
	Affective Index	1	0.00	< .001	1.00
Error		91			

Note. DV = dependent variable.

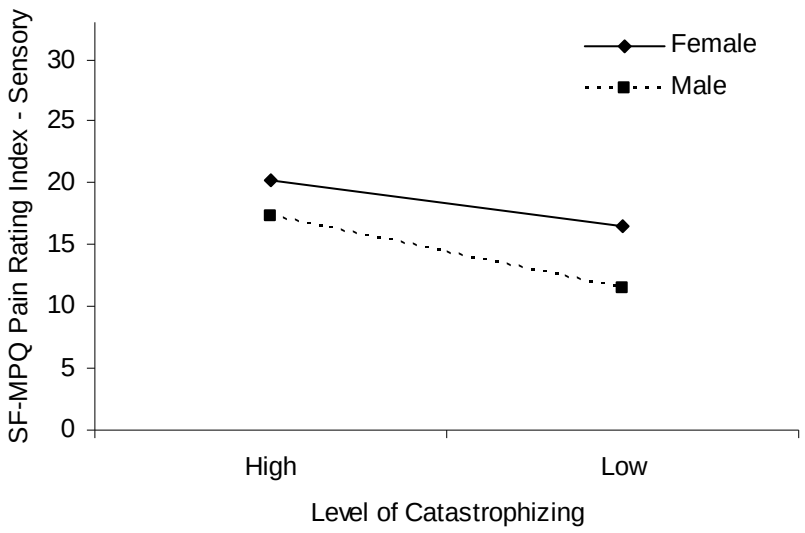

Figure 7. Short-Form McGill Pain Questionnaire Pain Rating Index - Sensory as a function of catastrophizing and gender.

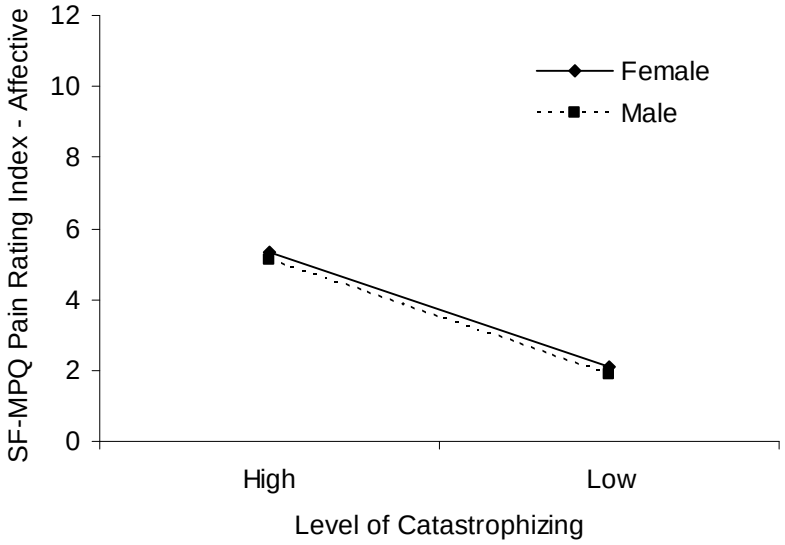

Figure 8. Short-Form McGill Pain Questionnaire Pain Rating Index - Affective as a function of catastrophizing and gender.

The results were followed up with a MANCOVA to determine whether the effects of catastrophizing and gender were significant while controlling for depression and anxiety. As with the MANOVA, catastrophizing and gender were the independent variables and sensory and affective dimensions of pain report from the SF-MPQ were the dependent variables. As with the previous analysis, results of the MANCOVA indicated that catastrophizing and gender were significantly related to the two dimensions of pain report (see Table 23). The interaction between catastrophizing and gender was not significant[3].

Table 23

MANCOVA for Short-Form McGill Pain Questionnaire Pain Rating Index Sensory and Affective Subscales

Source	*df*	*F*	Partial η^2	*p*
CES-D	2	1.22	.02	.30
STAI (State Form)	2	1.54	.03	.22
STAI (Trait Form)	2	1.99	.04	.14
Catastrophizing	2	10.51	.20	< .001
Gender	2	7.75	.15	.001
Catastrophizing x Gender	2	0.94	.02	.40
Error	87			

Note. CES-D = Center for Epidemiological Studies Depression Scale; STAI = State-Trait Anxiety Inventory.

[3] Note that follow-up univariate results of the MANCOVA yielded similar results to those for the MANOVA.

Hypothesis 5

HR. To investigate the relationship between catastrophizing, gender, and HR before, during, and after pain, a 2 (Catastrophizing Group) x 2 (Gender) x 3 (Time) mixed-model ANOVA was conducted[4].

As shown in Table 24, results of the ANOVA indicated a significant main effect of time. Examination of the contrasts revealed a significant quadratic effect of time, $F(1, 89) = 167.32$, $p < .001$, partial $\eta^2 = .65$. As can be seen in Figure 9, HR was higher during the pain induction period ($M = 91.92$, $SD = 14.40$) than before ($M = 80.06$, $SD = 10.63$) or afterwards ($M = 74.58$, $SD = 10.61$).

Table 24

ANOVA for Heart Rate Over Baseline, Pain Induction, and Pain Recovery Periods

Source	*df*	*F*	Partial η^2	*p*
Between subjects				
Catastrophizing	1	4.16	.04	.04
Gender	1	0.18	.002	.67
Catastrophizing x Gender	1	0.38	.004	.54
Error	89			
Within subjects				
Time	1.24	164.44	.65	< .001
Time x Catastrophizing	1.24	1.93	.02	.17
Time x Gender	1.24	0.25	.003	.67
Time x Catastrophizing x Gender	1.24	0.13	.001	.78
Error	110.77			

Note. df for within subjects analyses were corrected using Greenhouse-Geisser correction due to violations of sphericity assumption.

[4] Note that this analysis was also conducted with the reactivity and deactivation variables, which produced similar results.

Results of the ANOVA also indicated that there was a significant main effect of catastrophizing (refer to Table 24). A follow-up to this significant main effect indicated that high catastrophizers had higher pain during the pain induction period, but not during baseline or pain recovery periods (see Table 25). However, although there were no significant differences between catastrophizing during the baseline and pain recovery periods, as shown in Figure 9, there was a trend to this effect.

Table 25

Pairwise Comparisons of Heart Rate Between High and Low Catastrophizers Over the Baseline, Pain Induction, and Pain Recovery Periods

Time	Level of Catastrophizing	*M*	*SD*	*F*	Partial η^2	*P*
Baseline	High	82.02	10.43	2.81	.03	.10
	Low	78.36	10.61			
Pain Induction	High	95.52	15.85	5.14	.05	.03
	Low	88.82	12.36			
Pain Recovery	High	76.27	10.52	1.99	.02	.16
	Low	73.14	10.59			

Note. $df(1, 89)$ for all comparisons.

The main effect of gender was not significant, and neither were any two- or three-way interactions among time, catastrophizing, and gender (refer to Table 24).

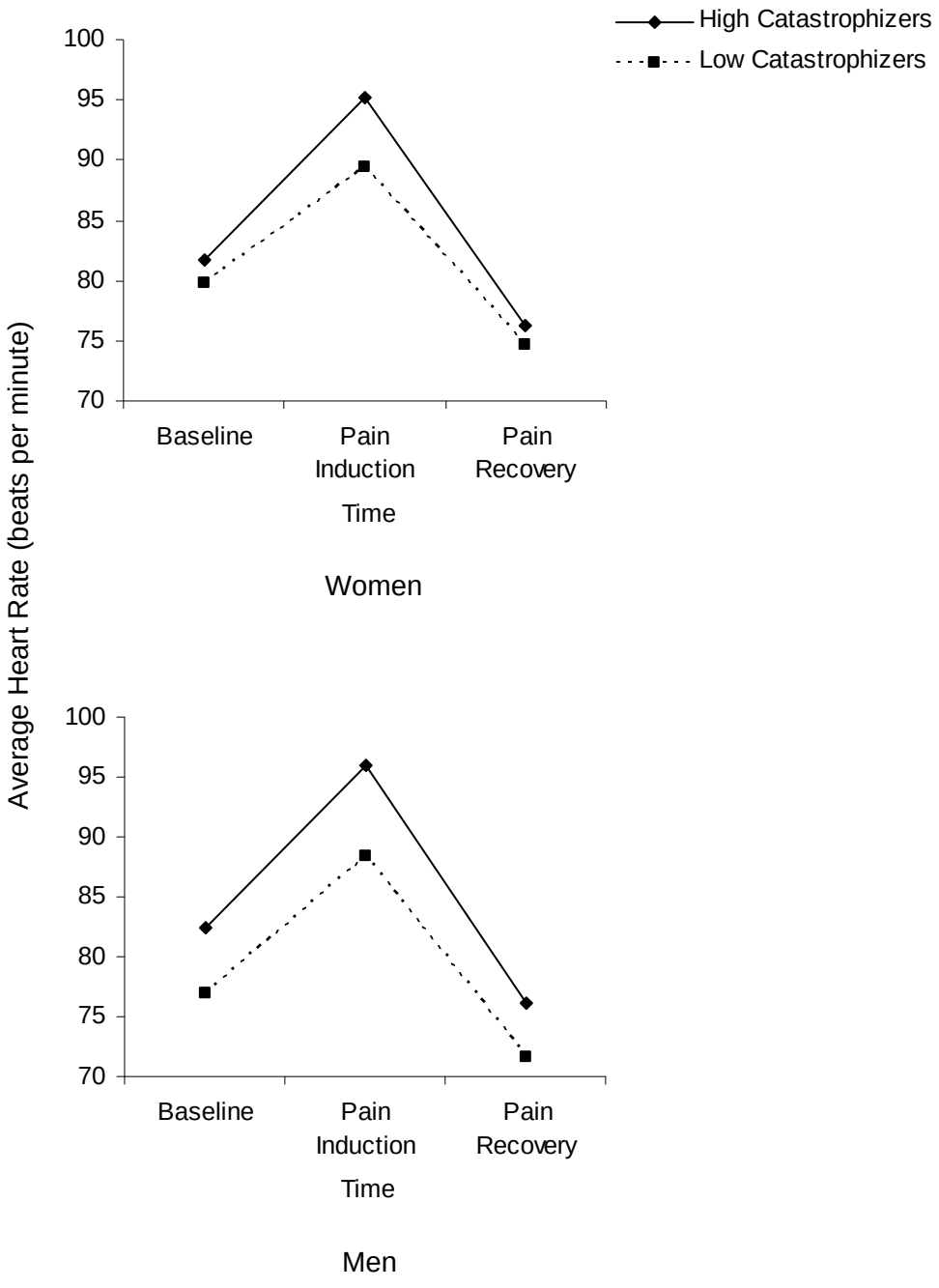

Figure 9. Heart rate as a function of catastrophizing and gender over baseline, pain induction, and pain recovery periods.

Because an effect of catastrophizing was found for HR during the pain induction period, a 2 (Catastrophizing Group) x 2 (Gender) x 3 (Time) mixed-model ANOVA was conducted as a follow-up to examine the HR over the 1-minute long pain induction period using 20-second intervals.

The main effect of time was not significant (see Table 26), however examination of the polynomial contrasts indicated a significant quadratic contrast, $F(1, 90) = 7.05$, $p = .01$, partial $\eta^2 = .07$ (refer to Figure 10).

Table 26

ANOVA for Heart Rate Over the Pain Induction Period

Source	*df*	*F*	Partial η^2	*p*
Between subjects				
Catastrophizing	1	5.59	.06	.02
Gender	1	0.02	< .001	.88
Catastrophizing x Gender	1	0.04	< .001	.84
Error	90			
Within subjects				
Time	1.40	1.24	.01	.28
Time x Catastrophizing	1.40	2.00	.02	.15
Time x Gender	1.40	0.11	.001	.83
Time x Catastrophizing x Gender	1.40	0.58	.006	.50
Error	126.25			

Note. df for within subjects analyses are corrected using Greenhouse-Geisser correction due to violations of sphericity assumption.

The ANOVA indicated a significant main effect of catastrophizing (refer to Table 26). Upon further examination, pairwise comparisons revealed that high catastrophizers

had higher HR than low catastrophizers for the time period from 20-40 seconds and 40-60 seconds, but not during the first 20-second interval (see Table 27). The main effect of gender was not significant and neither were any interactions (see Table 26).

Table 27

Pairwise Comparisons of Heart Rate Between High and Low Catastrophizers Over the Baseline, Pain Induction, and Pain Recovery Periods

Time	Level of Catastrophizing	*M*	*SD*	*F*	Partial η^2	*p*
0-20 seconds	High	94.51	15.50	3.29	.04	.07
	Low	89.30	12.58			
20-40 seconds	High	96.53	17.09	4.34	.05	.04
	Low	89.77	13.67			
40-60 seconds	High	96.35	17.21	7.61	.08	.01
	Low	87.39	13.58			

Note. $df(1, 90)$ for all comparisons.

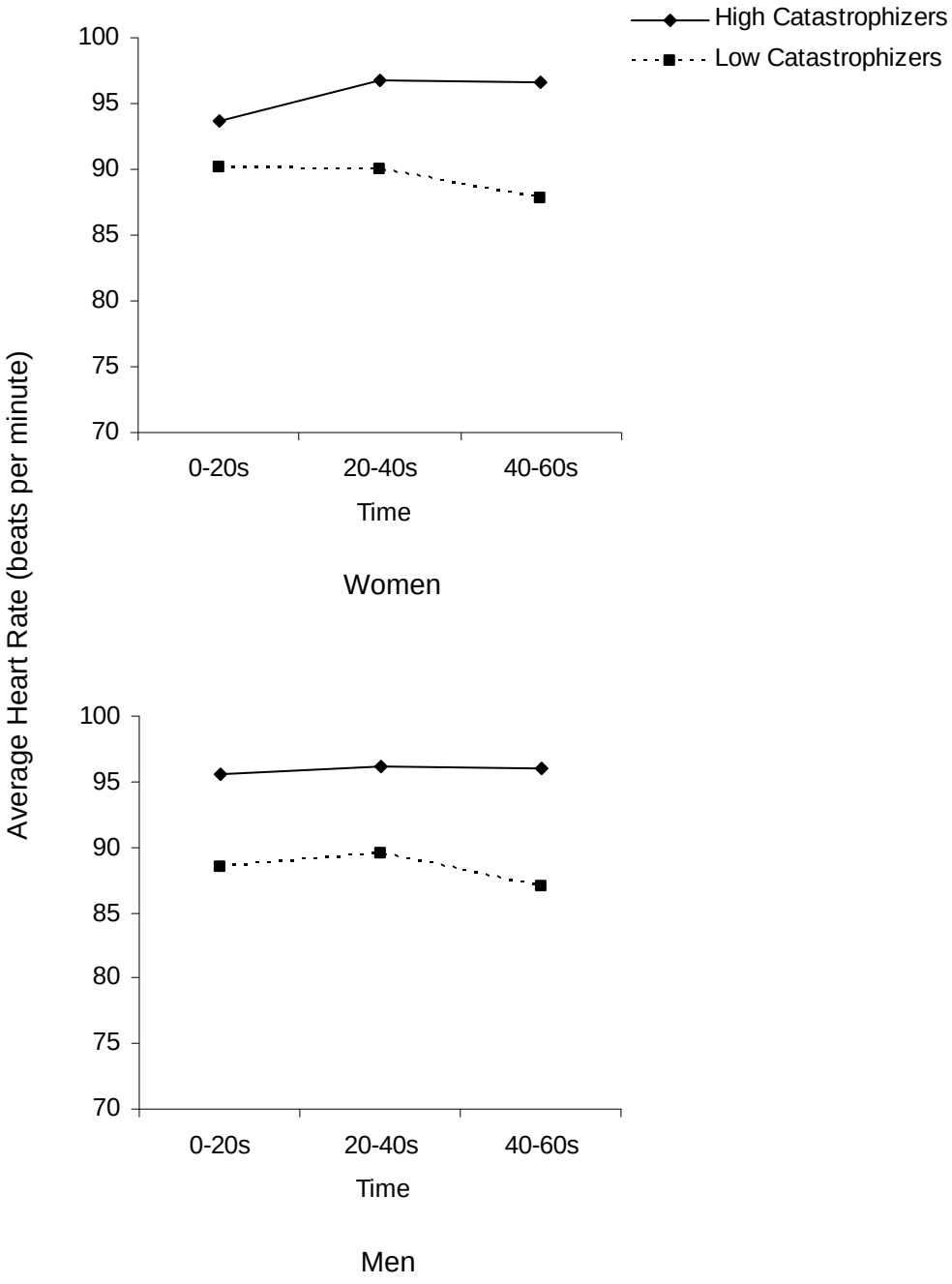

Figure 10. Heart rate as a function of catastrophizing and gender over the pain induction period.

Although there were no effects for the overall recovery period, a 2 (Catastrophizing Group) x 2 (Gender) x 10 (Time) mixed model ANOVA was conducted to examine effects of catastrophizing and gender on HR over the 10 minutes of the recovery period.

With the exception of a significant main effect of time, the results of the ANOVA were not significant (see Table 28). Although not significant, there appeared to be a trend such that high catastrophizers had higher HR throughout the recovery period (see Figure 11).

Table 28

ANOVA for Heart Rate Over the Pain Recovery Period

Source	*df*	*F*	Partial η^2	*P*
Between subjects				
Catastrophizing	1	1.70	.02	.20
Gender	1	0.34	.004	.56
Catastrophizing x Gender	1	0.57	.01	.45
Error	90			
Within subjects				
Time	2.56	13.00	.13	< .001
Time x Catastrophizing	2.56	1.36	.02	.26
Time x Gender	2.56	0.49	.005	.66
Time x Catastrophizing x Gender	2.56	0.49	.005	.66
Error	230.73			

Note. df for within subjects analyses are corrected using Greenhouse-Geisser correction due to violations of sphericity assumption.

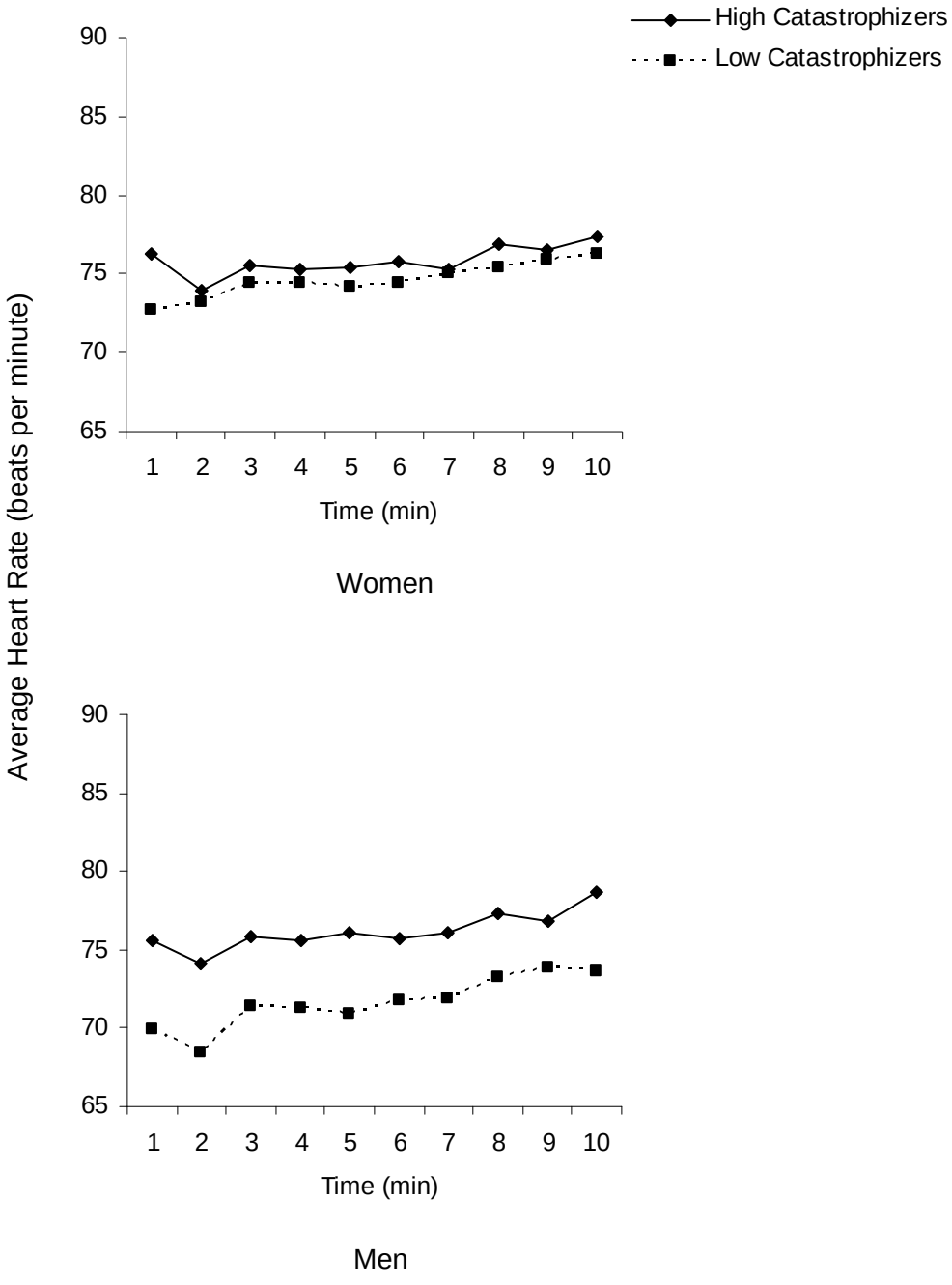

Figure 11. Heart rate as a function of catastrophizing and gender over the pain recovery period.

GSR. Similar to analyses conducted for the HR variables, a 2 (Catastrophizing Group) x 2 (Gender) x 3 (Time) mixed-model ANOVA was conducted to examine effects of catastrophizing and gender on GSR over baseline, pain induction, and pain recovery periods[5].

With the exception of a significant main effect of time, the results of this ANOVA were not significant (see Table 29). Examination of the contrasts revealed a significant quadratic effect of time, $F(1, 89) = 354.0$, $p < .001$, partial $\eta^2 = .80$. GSR was higher during the pain induction period ($M = 36.29$, $SD = 17.93$) than before ($M = 13.88$, $SD = 8.82$) or afterwards ($M = 18.56$, $SD = 11.25$). See Figure 12 for a depiction of the results.

Table 29

ANOVA for Galvanic Skin Response Over Baseline, Pain Induction, and Pain Recovery Periods

Source	*df*	*F*	Partial η^2	*p*
Between subjects				
Catastrophizing	1	0.03	< .001	.86
Gender	1	1.35	.01	.25
Catastrophizing x Gender	1	0.42	.005	.52
Error	89			
Within subjects				
Time	1.43	272.26	.75	< .001
Time x Catastrophizing	1.43	0.13	.002	.80
Time x Gender	1.43	1.21	.01	.29
Time x Catastrophizing x Gender	1.43	0.18	.002	.76
Error	127.53			

Note. df for within subjects analyses were corrected using Greenhouse-Geisser correction due to violations of sphericity assumption.

[5] Note that this analysis was also conducted with reactivity and deactivation variables, which produced similar results.

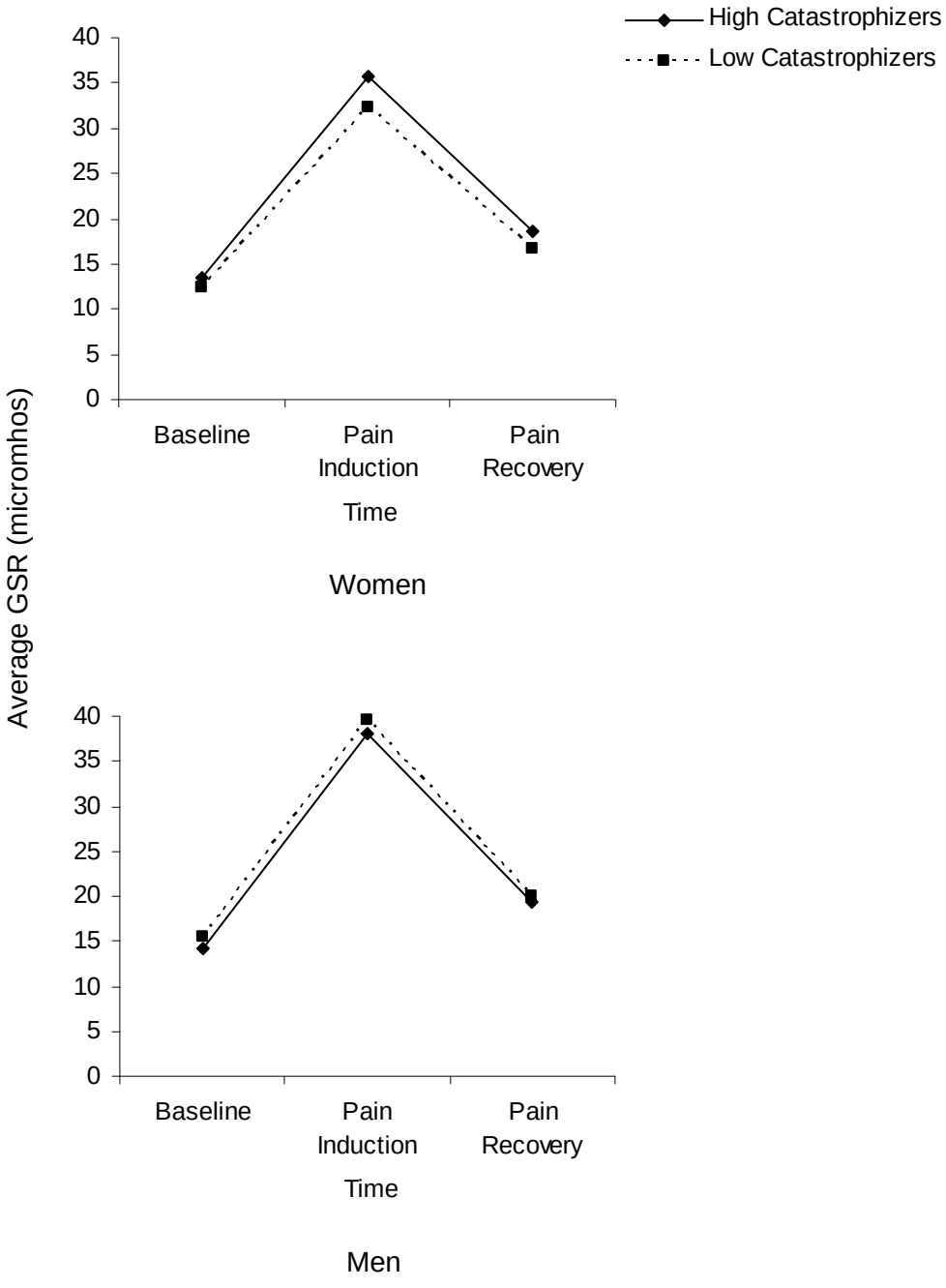

Figure 12. Galvanic skin response (GSR) as a function of catastrophizing and gender over the baseline, pain induction, and pain recovery periods.

A 2 (Catastrophizing Group) x 2 (Gender) x 3 (Time) mixed-model ANOVA was conducted to examine GSR over the 1-minute long pain induction period using 20-second intervals. Results of the ANOVA were not significant (see Table 30 and Figure 13).

Table 30

ANOVA for Galvanic Skin Response Over the Pain Induction Period

Source	*df*	*F*	Partial η^2	*p*
Between subjects				
Catastrophizing	1	0.03	< .001	.87
Gender	1	1.81	.02	.18
Catastrophizing x Gender	1	0.31	.003	.58
Error	90			
Within subjects				
Time	1.16	2.76	.03	.09
Time x Catastrophizing	1.16	0.28	.003	.63
Time x Gender	1.16	1.70	.02	.20
Time x Catastrophizing x Gender	1.16	0.68	.01	.43
Error	104.20			

Note. df for within subjects analyses are corrected using Greenhouse-Geisser correction due to violations of sphericity assumption.

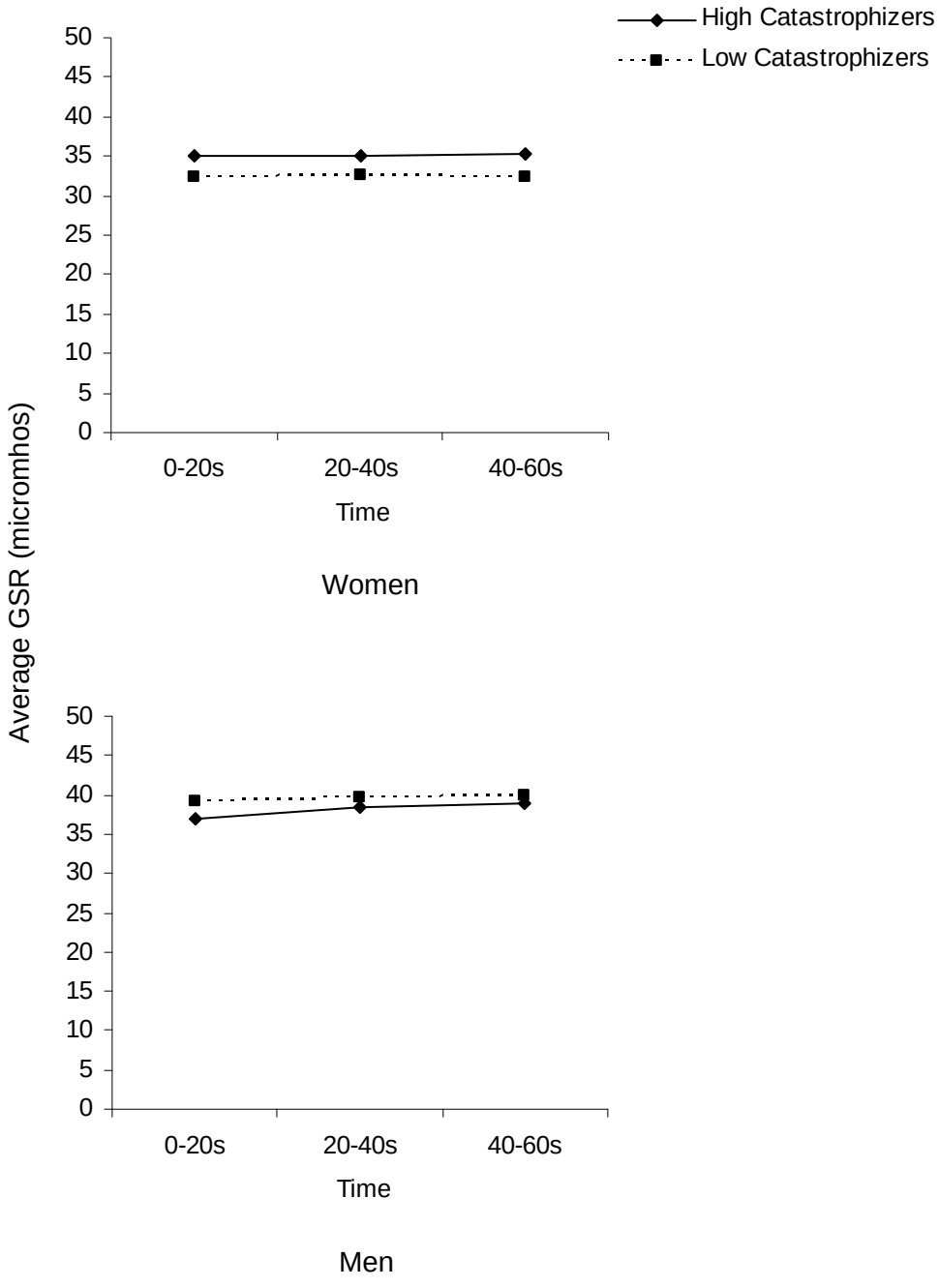

Figure 13. Galvanic skin response (GSR) as a function of catastrophizing and gender over the pain induction period.

A 2 (Catastrophizing Group) x 2 (Gender) x 10 (Time) mixed-model ANOVA was conducted to examine GSR over the 10-minute long pain recovery period using 1-minute long intervals. With the exception of a significant effect of time, which decreased linearly over time, $F(1, 90) = 56.75$, $p < .001$, partial $\eta^2 = .39$, results of the ANOVA were not significant (see Table 31 and Figure 14).

Table 31

ANOVA for Galvanic Skin Response Over the Pain Recovery Period

Source	*df*	*F*	Partial η^2	*p*
Between subjects				
Catastrophizing	1	0.04	< .001	.85
Gender	1	0.85	.01	.36
Catastrophizing x Gender	1	0.24	.003	.63
Error	90			
Within subjects				
Time	1.85	45.57	.34	< .001
Time x Catastrophizing	1.85	0.34	.004	.70
Time x Gender	1.85	0.09	.001	.90
Time x Catastrophizing x Gender	1.85	0.24	.003	.77
Error	166.55			

Note. df for within subjects analyses were corrected using Greenhouse-Geisser correction due to violations of sphericity assumption.

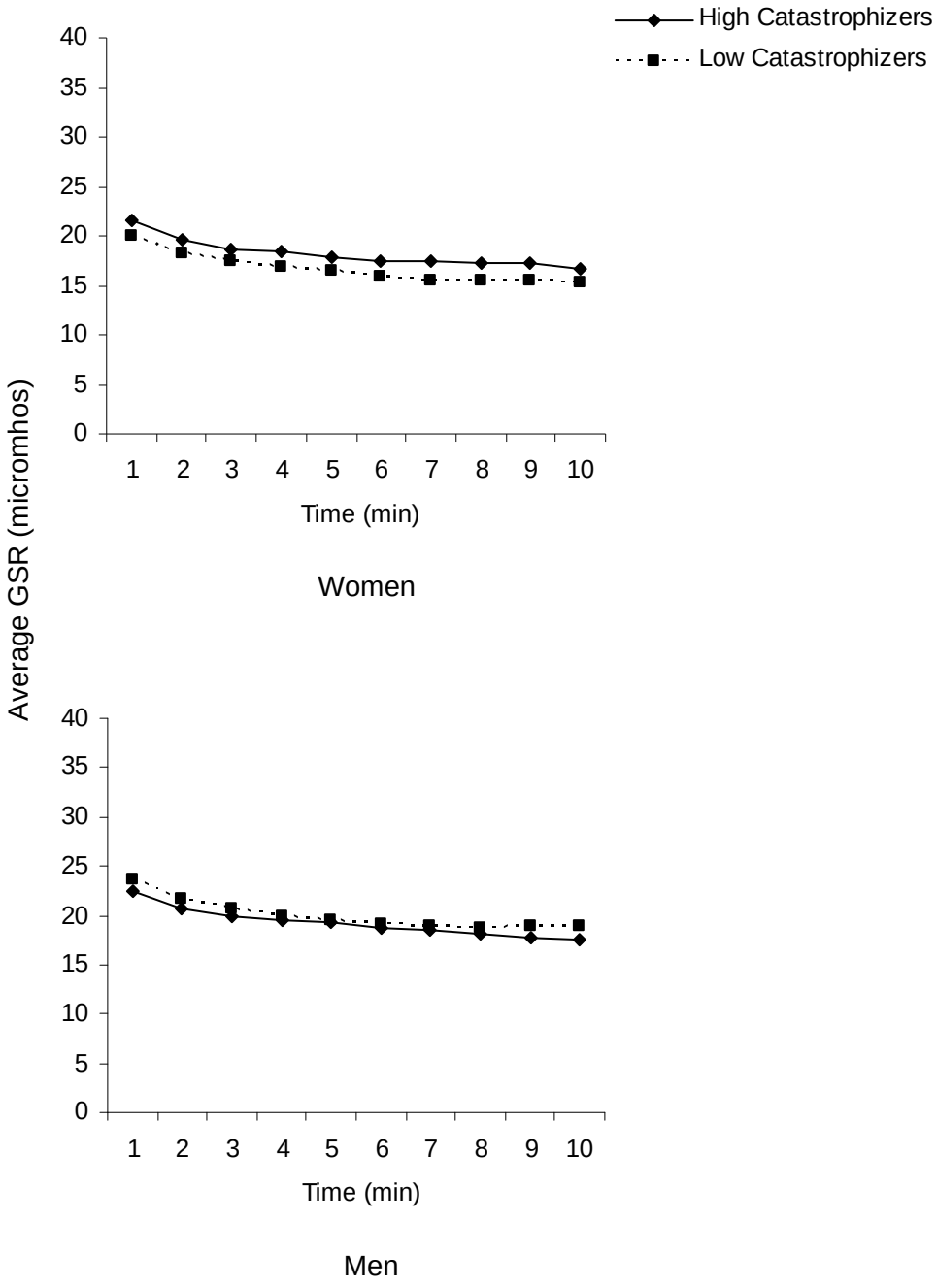

Figure 14. Galvanic skin response (GSR) as a function of catastrophizing and gender over the pain recovery period.

Hypothesis 6

To investigate the hypotheses that arousal would mediate the relationship between catastrophizing and pain report, mediation analyses were conducted. Catastrophizing was not significantly related to HR during the baseline period, to HR during the pain recovery period, or to GSR during any time period. Therefore, only two mediation analyses were conducted. The first analysis examined whether HR during the pain induction period mediated the relationship between catastrophizing and pain intensity during the pain induction period. The second analysis examined whether HR during the pain induction period mediated the relationship between catastrophizing and pain intensity during the pain recovery period.

To assess whether HR mediated the relationship between catastrophizing and pain report, two mediational regression analyses were conducted using Baron and Kenny's (1986) procedure. In the first step of the mediation analysis, a regression analysis was performed using catastrophizing as the independent variable and pain intensity during the appropriate period (i.e., pain induction or pain recovery) as the dependent variable. The second step of the analysis was to perform a regression analysis with catastrophizing as the independent variable and HR during the pain induction period as the dependent variable. The third and final step was to perform a regression analysis with catastrophizing and HR during the pain induction period as the independent variables and pain intensity during the appropriate period as the dependent variable and evaluate whether the effect of catastrophizing on pain report disappeared or decreased when the mediator was included in the model.

As shown in Table 32, for the first mediation analysis (i.e., examining whether HR during pain induction mediated the effect between catastrophizing and pain intensity during the pain induction period), results of the first step of the analysis indicated that catastrophizing was significantly related to pain intensity during the pain induction period (as expected given the results of the ANOVA conducted for hypothesis 2). Results of the second step indicated that catastrophizing was related to HR during pain induction (as expected given the results of the ANOVA conducted for hypothesis 5). The results of the third step of the analysis indicated that when catastrophizing and HR during the pain induction period were both included in the regression model, catastrophizing remained a significant predictor of pain intensity during the pain induction period. However, HR during the pain induction period was not a significant predictor of pain intensity during the pain induction period. The relationship between catastrophizing and pain intensity decreased slightly when HR was included as a variable in the analysis, thus a Sobel's test was conducted to determine whether it was a significant reduction. The results of the Sobel's test indicated that there was no effect of mediation, Sobel's test = 0.15, $p = .88$.

Table 32

Results of the Mediation Analysis of HR During Pain Induction as a Mediator of the Relationship Between Catastrophizing and Pain Intensity During Pain Induction

Step	IV	DV	*B*	β	*T*	*df*	*p*
Step 1	Catastrophizing	Pain Intensity	1.54	.40	4.18	93	< .001
Step 2	Catastrophizing	HR	6.95	.24	2.40	92	.02
Step 3	Catastrophizing	Pain Intensity	1.49	.38	3.87	91	< .001
	HR		0.002	.01	0.18	91	.86

Note. IV = independent variable, DV = dependent variable. Catastrophizing was entered as a dichotomous variable.

As shown in Table 33, for the second mediation analysis (i.e., examining whether HR during pain induction mediated the effect between catastrophizing and pain intensity during the pain recovery period), results of the first step of the analysis indicated that catastrophizing was significantly related to pain intensity during the pain recovery period (as expected given the results of the ANOVA conducted for hypothesis 2). As with the previous analysis, results of the second step of the analysis indicated that catastrophizing was related to HR during pain induction. The results of the third step of the analysis indicated that when catastrophizing and HR during the pain induction period were both included in the regression model, catastrophizing remained a significant predictor of pain intensity during the pain recovery period. However, HR during the pain induction period was not a significant predictor of pain intensity during the pain recovery period. A Sobel's test was conducted to determine whether this was significant mediation. The results of the Sobel's test indicated that there was no effect of mediation, Sobel's test = -0.33, $p = .74$.

Table 33

Results of the Mediation Analysis of HR During Pain Induction as a Mediator of the Relationship Between Catastrophizing and Pain Intensity During Pain Recovery

Step	IV	DV	*B*	β	*t*	*df*	*p*
Step 1	Catastrophizing	Pain Intensity	0.52	.34	3.43	93	.001
Step 2	Catastrophizing	HR	6.95	.24	2.40	92	.02
Step 3	Catastrophizing	Pain Intensity	0.53	.34	3.35	91	.001
	HR		-0.002	-.03	-0.33	91	.74

Note. IV = independent variable, DV = dependent variable. Catastrophizing was entered as a dichotomous variable.

Chapter 4: Discussion

The main purpose of the present study was to investigate whether individuals high and low in catastrophizing would manifest differential psychophysiological arousal patterns in an experimental laboratory pain setting. Expected differences in this study were formed from theory based on a cognitive appraisal model of catastrophizing. The model proposed in this study outlined potential associations between catastrophizing, pain, and arousal in the periods before, during, and after pain induction (see Figure 1). Based on the proposed model and the supportive literature, several hypotheses were specified and examined. What follows is a discussion of these hypotheses in the order presented in the results. Following discussion of the particular hypotheses, theoretical and clinical implications, study limitations, future research, and general conclusions are presented.

Associations among Pain Report and Arousal Variables (Hypothesis 1)

The first hypothesis suggested that the relationships outlined in the proposed study model would be significantly associated, with several associations specified. To summarize the associations, the hypotheses and respective outcomes are displayed in Table 34. This table indicates that several hypotheses were supported fully or in part.

Table 34

Hypotheses Regarding Associations among Pain Report and Arousal Variables and Outcomes

Hypothesis	Outcome
1a. Pain reported during the pain induction period would be positively associated with pain during the pain recovery period. Also, pain intensity and pain unpleasantness during the pain induction period would be positively associated as would pain intensity and pain unpleasantness during the pain recovery period.	Supported
1b. Arousal (i.e., HR, GSR) during the baseline period would be positively associated with arousal during the pain induction period. Also, arousal during pain induction would be positively associated with arousal during the pain recovery period. Finally, HR and GSR would be positively associated within the baseline, pain induction, and pain recovery periods.	Supported
1c. Pain reported during the pain induction period would be positively associated with arousal during pain induction. Likewise, pain reported during the pain recovery period would be positively associated with arousal during the pain recovery period.	Partially Supported
1d. Arousal during the baseline period would be positively associated with pain during the pain induction period. As well, arousal during the pain induction period would be positively associated with pain during the pain recovery period.	Not Supported
1e. Pain during the pain induction period would be positively associated with arousal during the pain recovery period	Not Supported

Hypothesis 1a. Results indicated that different measures of pain report (i.e., pain intensity and pain affect) were associated within the time periods. In fact, pain measures of intensity and unpleasantness were so highly associated that they were considered redundant, and thus, only the results for pain intensity were presented. These results are consistent with previous literature indicating that pain intensity and pain affect are associated. For example, in a study investigating physiological responses to acute cold pressor pain, Dixon et al. (2004) found that pain intensity and pain unpleasantness, as assessed using visual analogue scales, were significantly and positively associated at the moderate level.

It was interesting that pain intensity and pain unpleasantness showed such high correlations in the present study, as previous research has noted that although pain affect (i.e., the emotional response to pain) is positively associated with pain intensity (i.e., the amount the pain stimulus hurts), the two constructs are somewhat conceptually and empirically distinct (see Jensen & Karoly, 2001). Perhaps the use of a single-item measure of pain affect (i.e., pain unpleasantness) did not adequately measure the construct. More comprehensive measures of pain affect that include multiple descriptors of emotionality might more accurately measure the affective component of pain. For example, Jensen and Karoly noted that the SF-MPQ (or the full version of the MPQ), which contains a specific subscale of pain affect, may more accurately measure the affective component of pain. In the present study, the relationships among pain intensity, pain unpleasantness, and the SF-MPQ were also assessed. Pain intensity was strongly associated with the affective subscale of the SF-MPQ, but not so strongly correlated that the two constructs would be considered redundant. Furthermore, pain unpleasantness was

associated to a similar degree as pain intensity with the affective subscale of the SF-MPQ, which indicates that pain affect, as measured by the SF-MPQ, is different than pain unpleasantness rated on a NRS. Thus, as Jensen and Karoly (2001) noted, it may be that pain affect assessed using the SF-MPQ may be a more accurate measure of the affective component of pain than pain unpleasantness and should be considered in future research as such.

Results of the present study also indicated that pain intensity at each of the 20-second time points during the pain induction period were highly associated with one another. Similarly, pain intensity reports at each of the 1-minute time points during the pain recovery period were highly associated with one another. The one exception was that during the pain recovery period, the last pain intensity rating was not correlated with the first three ratings. This lack of a correlation has not previously been presented in the pain literature so there is no standard for comparison. However, it is speculated that the lack of association with the first ratings may be due to a different pain experience as one recovers from the cold pressor pain induction task. That is, by the end of the pain recovery period, most people reported no pain by the final rating. Specifically, 93.7% of the sample reported 0 on the scale ranging from 0 (*No Pain*) to 10 (*Extreme Pain*). Thus, because almost everyone reported no pain at the 10-minute time point of the recovery period, by the end of the recovery period there is no difference between those who were reporting higher pain versus those reporting lower pain at the beginning of the recovery period.

Correlational findings also indicated that pain report was associated across time periods (i.e., pain intensity and unpleasantness during pain induction were associated

with pain intensity and unpleasantness during pain recovery and retrospective reports of pain using the SF-MPQ). These findings supported the hypothesis that pain report at earlier time periods would be associated with pain report at later time periods. Thus, the amount of pain one experiences during an acute pain stimulus is related to the amount of pain experienced and recalled afterwards. These results indicate that those individuals who report heightened pain during an acute pain experience also report heightened pain following termination of the pain induction task. The results are consistent with Ito, Kurita, Ito, and Arao's (2002) clinical pain results, which showed that patients with burning mouth syndrome (i.e., burning discomfort or pain experienced in patients for whom dental or medical causes have been excluded) who reported higher pain when pain was induced with heat, cold, or mechanical stimuli, also reported higher pain for the period following pain induction.

Hypothesis 1b. As with pain data, arousal was found to be associated across time periods. Specifically, HR during the baseline period was associated with HR during the pain induction and recovery periods. Likewise, GSR during the baseline period was associated with GSR during the pain induction and recovery periods. Thus, the findings for arousal were similar to pain in that arousal at earlier periods was associated with arousal at later periods. Results suggest that the body's response to an impending pain stressor is associated with the amount of arousal one will experience during and then after pain. These findings are consistent with the proposed model and are also consistent with results indicating psychophysiological measures are associated at different time points. For example, Dixon et al. (2004) found that blood pressure before and after cold pressor pain induction were positively associated.

Contrary to the hypothesis was the finding that HR was not associated with GSR when using inter-individual correlations. This finding was unexpected because HR and GSR are both indexes of SNS activation (Flor, 2001). However, HR is an index of SNS activation *and* PNS activation (Flor). Thus, perhaps differences in PNS activation (i.e., the "rest and digest" component of the ANS) might explain why the two variables were not correlated.

Although it was expected that different measures of arousal would be associated, other studies have also shown no association between measures of HR and GSR (e.g., Lazarus et al., 1963). Apparently, there may be stimulus specificity in the arousal response that is elicited, where particular stressors elicit specific autonomic response profiles. For example, Lovallo (1975) noted that although GSR initially increases during exposure to the cold pressor task, it is the cardiovascular response that is the most prominent with exposures greater than one minute. Further, stimulus specificity might be used to partially explain the lack of correlations between different measures of autonomic arousal. Calculating the intra-individual correlation as opposed to inter-individual correlation is suggested to have greater validity in such comparisons and improves correlations between measures of arousal (Lazarus et al., 1963). Indeed, in the present study when the intra-individual correlation was used for the pain recovery period, HR and GSR were significantly associated, such that higher HR was associated with lower GSR. This significant relationship between these two variables is consistent with Lazarus et al.'s results that calculating intra-individual correlations improved the relationship observed between the measures of autonomic arousal, which is what one would expect given that both HR and GSR are measures of arousal.

With regard to the directionality of the relationship, although it may seem odd to have a negative association between two measures of SNS activation, the negative association is appropriate when considering that that the association occurred during the pain recovery period. From Figure 11, it is shown that HR declined for the first minute. However, for the remainder of the pain recovery period, HR increased slightly. In contrast, GSR consistently decreased throughout the recovery period (see Figure 14). These differences in directions of arousal during the recovery period may explain the negative association between HR and GSR. Due to the fact that no published research has documented an association between HR and GSR over a pain recovery period, the present data must be viewed cautiously.

Hypothesis 1c. Another component of the first hypothesis was that pain would be associated with arousal variables (i.e., HR and GSR). During the pain induction period, pain intensity was not associated with the raw HR variables. However, pain intensity ratings were consistently associated with the HR reactivity for the last 20-seconds of the pain induction period (i.e., the increase in HR from baseline to the HR for the last 20 seconds of the pain induction period). The finding that HR reactivity and pain intensity were related is consistent with previous research indicating that HR and subjective pain reports during cold pressor pain were related (e.g., Victor, Mainardi, & Shapiro, 1978). The lack of relationship between pain intensity and HR for the first 40 seconds of the pain induction period is consistent with findings from Dowling (1983). Dowling suggested that the lack of relationship might be due to tachycardia induced by immersing one's arm in cold water, as noted by Lovallo (1975). Although Dowling did not report HR for the period following 40 seconds of immersion, it seems likely that this period of

tachycardia might dissipate with increased exposure as participants become accustomed to the task, which could be the reason for the significant relationships with HR later on in the pain induction period observed in the present study.

During the recovery period there were no associations between pain report and HR with either the raw HR variables or the deactivation variables. Thus, there was little correspondence between pain and HR after the pain induction task. Regardless of what participants reported, their physiology did not correspond. Thus, while an individual may still report higher pain after they remove their arm, their physiology did not correspond to this report. This finding contradicts the hypothesis proposed based on the model. There is some research to support this non-significant finding. For example, Edwards and Fillingim (2005) found no association between retrospective reports of pain intensity from a CPT or pain tolerance (i.e., the amount of time the pain stimulus can be withstood) and changes in cardiovascular measures from pre-pain to post-pain.

It is suggested that this lack of relationship between HR and pain may have something to do with ANS system activity during the recovery period. When a stressor is introduced, the body reacts by activating the SNS; when the stressor is removed the body reacts by activating the PNS to re-establish homeostasis (Johnson et al., 1992). Thus, once the pain stimulus is removed, the body reacts to the removal of the pain stressor by activating the PNS. Although HR is a measure of both SNS and PNS activity, perhaps a measure of purely PNS activity may more accurately capture what occurs during the recovery period. While the activation of the PNS may result in decreased SNS activity, it may be that the PNS activity during the recovery period may show a stronger relationship with pain report.

With regard to GSR, there were no relationships between pain intensity and any of the GSR variables in either the pain induction or the pain recovery periods. These findings are contrary to those of Peters and Schmidt (1991), which showed a relationship between GSR and pain, such that patients with chronic low back pain had higher GSR reactions than control participants during pain induction. However, the findings of Peters and Schmidt may not generalize to the current sample because chronic pain was not examined. Importantly though, results regarding the relationship between GSR and pain intensity are mixed even within the chronic pain literature. For example, GSR and pain were studied in chronic low back pain patients with no association found between GSR and pain (Flor, Birbaumer, Schugens, & Lutzenberger; Flor, Turk, & Birbaumer, 1985). From the present results, it may be concluded that in acute pain induction tasks, as used in this study, perhaps HR might be a better indicator of arousal than GSR because HR correlates with pain intensity ratings. Such a result would also be consistent with findings indicating that the cardiovascular response is most prominent in cold pressor tasks (Lovallo, 1975).

Another possibility is that GSR may not be associated with report of pain intensity, but may instead be more associated with other measures of pain experience, such as pain threshold (i.e., the time at which the sensations are reported as painful) or pain tolerance (i.e., the amount of time one can withstand the pain experience). For example, Dowling (1983) found a relationship between GSR during cold water immersion and pain tolerance. These particular pain measures were not collected in the present study and thus these associations cannot be confirmed within this sample.

Importantly, although correlations were not observed between pain intensity at all times and the arousal variables, both arousal variables increased during the pain induction period from baseline and decreased during the recovery phase (as evidenced by the analyses for the fifth hypothesis which can be seen in Figures 9 and 12 for HR and GSR, respectively). This finding suggests that participants experienced arousal in response to the pain stressor in a manner consistent with the proposed model. Furthermore, this pattern of arousal response is consistent with previous literature indicating that pain elicits arousal-based physiological activation (e.g., al'Absi & Petersen, 2003; Dixon et al., 2004; Melzack, 1999).

Hypotheses 1d & 1e. Results of the present study also indicated that there were no significant correlations across time periods between pain report and arousal variables. Thus, psychophysiological arousal at earlier time points was not associated with pain at later time points. Likewise, pain at earlier time points (i.e., during the pain induction period) was not associated with arousal during later time points (i.e., during the pain recovery period). Given that the associations between pain and arousal within the same time point were small and/or absent, it is not surprising that across time periods arousal and pain were also not related.

In summary, the primary finding of the first set of correlational hypotheses was that pain and arousal variables are associated with themselves but not with one another. It is also worth mentioning that speculative associations were not supported in this study, such as the associations between pain and arousal during recovery or across periods. The following section reviews the relationship among catastrophizing, gender, and pain report during pain induction.

Catastrophizing, Gender, and Pain Report During Pain Induction (Hypothesis 2)

To summarize the results of the second hypothesis, the specific hypotheses and respective outcomes are displayed in Table 35.

Table 35

Hypotheses Regarding the Relationship between Catastrophizing, Gender, and Pain Report During the Pain Induction Period

Hypothesis	Outcome
2a. High catastrophizers would report greater pain intensity and unpleasantness during the pain induction period than low catastrophizers.	Supported
2b. Women would report higher pain intensity and pain unpleasantness during the pain induction period than men.	Supported
2c. Catastrophizing and gender would interact, such that for women, high catastrophizers would report greater pain than low catastrophizers, but this effect would not be as large for men.	Not Supported

Consistent with the second hypothesis, results of the present study replicated previous research, indicating a clear association between catastrophizing and heightened pain report (e.g., Edwards et al., 2006; France et al., 2002; Jacobsen & Butler, 1996; Smeets et al., 2006; Sullivan et al., 1995, 1998, 2000, 2001, 2004, Tripp et al., 2006). Specifically, the present study showed that high catastrophizers reported greater pain than low catastrophizers at all three time points during the pain induction period. This finding is consistent with the model presented in Figure 1, which proposed that catastrophizing

would be related to heightened pain report during the pain induction period. Thus, this finding supports previous literature which suggested that catastrophizing is an individual difference factor that manifests greater pain in a laboratory setting in healthy, pain-free participants (France et al., 2002; Sullivan et al., 1995, 2000).

Furthermore, these results also showed significant effects of gender, where women reported greater pain than men. The gender difference in this study is consistent with research showing that women report greater pain than men (e.g., Sullivan et al., 2000; Keogh & Herdenfeldt, 2002; Unruh, 1996). Importantly, this gender difference result is considered to be robust because the experiment was designed in a way that sought to eliminate differences in catastrophizing from gender. Previous studies suggested that gender differences might be confounded with catastrophizing because females tend to catastrophize more than men (e.g., Sullivan et al., 1995). Although some studies found that when controlling for catastrophizing, there was no longer a relationship between gender and pain report (e.g., Sullivan et al., 2000), this finding has not been consistent in the literature. For example, Edwards et al. (2004) replicated the effect for daily reported pain, but not for experimentally-induced pain. Thus, in the present study when pre-existing differences in catastrophizing were eliminated by selecting equal groups by their level of catastrophizing (i.e., top 3rd of each distribution), women still reported higher pain than men, providing support for studies which indicate that gender differences are an important consideration when determining pain report (e.g., Unruh, 1996).

There were no interactions between catastrophizing and gender for pain intensity during the pain induction period. That is, the effect of catastrophizing did not differ for

women and men (i.e., both female and male high catastrophizers reported higher pain than their low catastrophizing counterparts). Previous pain research has not directly reported interactions between catastrophizing and gender, typically because catastrophizing is often measured on a continuous scale rather than dichotomized, as was done in the present study to maximize group differences. However, because both catastrophizing and gender were examined, the interactions were also examined in the present study. The lack of an interaction suggests that catastrophizing is related to increased pain in both men and women.

Thus, consistent with the model and with previous literature, results indicated that for both men and women catastrophizing was associated with heightened pain report during the pain induction task. The next section reviews findings regarding pain report post-pain induction during the pain recovery period.

Catastrophizing, Gender, and Pain Report During Pain Recovery (Hypothesis 3)

To summarize the results of the third hypothesis, the specific hypotheses and respective outcomes are displayed in Table 36.

Table 36

Hypotheses Regarding the Relationship between Catastrophizing, Gender, and Pain Report During the Pain Recovery Period

Hypothesis	Outcome
3a. High catastrophizers would report greater pain intensity and unpleasantness during the pain recovery period than low catastrophizers.	Supported
3b. Women would report higher pain intensity and pain unpleasantness during the pain recovery period than men.	Supported
3c. Catastrophizing and gender would interact, such that for women, high catastrophizers would report greater pain than low catastrophizers, but this effect would not be as large for men.	Not Supported

What is novel with respect to the findings of pain report was that catastrophizing was associated with pain intensity after the pain task was completed. Pain report following a cold pressor task has not been previously reported in the catastrophizing literature. These results are consistent with previous literature demonstrating that catastrophizing is related to higher pain report during pain induction (e.g., France et al., 2002; Sullivan et al., 1995, 2001), but adds to the literature in suggesting that catastrophizers report experiencing greater pain even after an acute pain stimulus is terminated. Specifically, it was not until 2 minutes after the termination of the CPT that participants high and low in catastrophizing were reporting similar pain ratings. Thus, pain lasts longer for individuals higher in catastrophizing, but this time effect seems to be limited in nature. Clinically, this finding is consistent with surgery research indicating

that pre-operative catastrophizing was related to greater post-operative pain in post-surgical patients (Pavlin, Sullivan, Freund, & Roesen, 2005).

The finding that catastrophizing and heightened pain report in the recovery period were associated also supports the proposed model. This finding suggests that following a pain experience, catastrophizers may still experience negative ruminative or magnifying thoughts about their pain and interpret the situation as threatening (i.e., primary appraisals) or perhaps they feel helpless with respect to coping with the pain (i.e., secondary appraisals). This suggestion must be considered speculative because these cognitions were not assessed during the different study periods but remain plausible according to the proposed model.

There was also a significant effect of gender for pain intensity reported during the recovery period, such that females reported greater pain than men. This result replicated the findings of al'Absi and Petersen (2003), which showed that women rate pain higher in a recovery period following cold pressor pain induction than men. Thus, as with high catastrophizers, women rated pain higher even after the pain stimulus was removed. These results further indicate that gender differences are an important consideration in experimental pain. As with pain during the pain induction period, there was no interaction between catastrophizing and gender for pain in the recovery period. As before, it seems as though catastrophizing acts to increase pain similarly in males and females.

Thus, as with pain intensity during the pain induction period, results indicated that for both men and women, catastrophizing was associated with heightened pain report during the pain recovery period. These findings were consistent with previous literature (e.g., Edwards et al., 2006; France et al., 2002; Jacobsen & Butler, 1996; Smeets et al.,

2006; Sullivan et al., 1995, 1998, 2000, 2001, 2004, Tripp et al., 2006) as well as with the proposed model, which suggested that high catastrophizers should also report pain as more intense following the pain induction task. Effects of catastrophizing and gender on retrospective report of sensory and affective pain are discussed in the following section.

Catastrophizing, Gender, and Retrospective Pain Report (Hypothesis 4)

To summarize the results of the fourth hypothesis, the specific hypotheses and respective outcomes are displayed in Table 37.

Table 37

Hypotheses Regarding the Relationship between Catastrophizing, Gender, and Retrospective Pain Report

Hypothesis	Outcome
4a. High catastrophizers would report greater sensory pain and affective pain than low catastrophizers.	Supported
4b. Women would report greater sensory pain and affective pain than men.	Partially Supported
4c. Catastrophizing and gender would interact, such that for women, high catastrophizers would report greater sensory and affective pain than low catastrophizers, but this effect would not be as large for men.	Not Supported

Examining differences in retrospective pain report among catastrophizers was also a novel addition to the pain literature. Although the SF-MPQ has been used in experimental pain induction studies investigating gender effects (e.g., Keogh &

Herdenfeldt, 2002), it has not been used in the investigation of pain report differences in the catastrophizing literature. In the present study, the SF-MPQ was used to assess whether participants differed in pain ratings after the pain stimulus was terminated and 10 minutes had passed. Furthermore, the SF-MPQ was used to investigate the sensory and affective components of pain experience. Sensory pain refers to the physical sensations of pain, whereas affective pain refers to the emotional components of pain experience. Affective pain is suggested to be more complex and difficult to measure than sensory pain which was the rationale for including the SF-MPQ in the present study – a reliable and valid measure of affective pain (Jensen & Karoly, 2001).

Results of the present study indicated significant effects of catastrophizing for both the sensory and affective dimensions of pain, such that high catastrophizers reported significantly higher sensory and affective pain than low catastrophizers. With regard to the sensory dimension of pain, there was a significant effect of gender, such that women reported greater sensory pain than men. However, there were no gender differences in terms of affective pain report.

The results that there were catastrophizing group and gender differences in terms of sensory pain are consistent with previous literature indicating that high catastrophizers and women report greater pain intensity than low catastrophizers and men, respectively (e.g., Keogh & Herdenfeldt, 2002; Sullivan et al., 1995, 2000, 2001; Unruh, 1996). These results are also consistent with studies indicating that sensory pain is rated higher in females than men (e.g., al'Absi & Petersen, 2003; Keogh & Herdenfeldt, 2002). The finding that high catastrophizers rated their affective pain higher than low catastrophizers is consistent with the body of research indicating that catastrophizers have a more

negative interpretation of the pain experience. For example, Sullivan, Rodgers, et al. (2001) found that catastrophizing was associated with not only heightened pain, but also heightened emotional distress resulting from pain. Thus, catastrophizers rate pain stimuli as not only hurting more, but they also rate emotional components of pain as being worse as well.

It was interesting that for the affective dimension of pain there was an effect for catastrophizing, but not for gender. This lack of a gender effect with regard to the affective dimension of pain was somewhat surprising given previous findings indicating that women report greater pain and seek more care for their pain than men (Unruh, 1996). However, these findings replicated those of Keogh and Herdenfeldt (2002), which showed that women rated their sensory pain significantly higher than men, but there were no differences between men and women in their overall ratings of affective pain. Although the full version of the McGill Pain Questionnaire was used, al'Absi and Petersen (2003) also found no differences in affective pain between genders, but did find differences in sensory pain. Thus, although females rate their pain higher than men, it is only in the sensory aspect that this pain differs, not in terms of the affective description of pain. Thus, women may find pain more physically painful, but are not more emotionally affected by it. Indeed, Unruh et al. (1999) found that there were no gender differences in terms of emotional upset caused by pain in a community sample with pain classified as troublesome.

These results from retrospective reports of pain further provide support for the model. As with pain report collected during the pain induction period and during the recovery period, high catastrophizers (regardless of gender) reported greater pain even

after the pain stimulus was terminated when recalling pain from the pain stimulus. Furthermore, the finding that catastrophizers differed in affective report of pain indicates that high catastrophizers may be interpreting pain more negatively than low catastrophizers, which provides further support for an appraisal model of catastrophizing.

Catastrophizing, Gender, and Pain Report with Covariates

For all of the analyses regarding relationships between catastrophizing, gender, and pain report at different points described above, when the analyses were repeated while controlling for depressive symptoms and anxiety, the effects of catastrophizing and gender described above remained significant. These results indicated that catastrophizing and gender had effects on the variety of pain report measures even while controlling for psychological variables which are reliably associated with pain experience in acute and chronic pain samples (e.g., Asmundson et al., 1996; Banks & Kerns, 1996; McCraken et al., 1996; Robinson & Riley, 1999). This set of findings should be seen as a very conservative test of effects and it adds to the literature on the robust association between catastrophizing and pain. Indeed, these data are consistent with the research indicating that catastrophizing is a strong predictor of the pain experience (e.g., Sullivan, Thorn, et al., 2001; Tripp et al., 2006). These findings also provide support for those studies finding that gender is an important distinguishing factor in determining pain outcomes (e.g., Unruh, 1996) and that catastrophizing is conceptually differentiated from constructs of mood (e.g., Sullivan & D'Eon, 1990; Sullivan, Thorn et al., 2001).

Catastrophizing, Gender, and Arousal (Hypothesis 5)

To summarize the results of the fifth hypothesis, the specific hypotheses and respective outcomes are displayed in Table 38.

Table 38

Hypotheses Regarding the Relationship between Catastrophizing, Gender, and Arousal Across Baseline, Pain Induction, and Pain Recovery Periods

Hypothesis	Outcome
5a. High catastrophizers would have higher arousal than low catastrophizers across baseline, pain induction, and pain recovery.	Supported for HR
5b. Women would have higher arousal than men across baseline, pain induction, and pain recovery.	Not Supported
5c. Arousal would be higher during the pain induction period than during baseline or pain recovery.	Supported
5d. Catastrophizing and gender would interact, such that for women, high catastrophizers would display higher arousal than low catastrophizers, but this effect would be smaller for men.	Not Supported
5e. Catastrophizing and time would interact, such that high catastrophizers would have higher arousal during pain induction than baseline, but there would be no difference in arousal between pain induction and pain recovery periods. Low catastrophizers, in contrast, would have higher arousal during pain induction than during baseline, but there is expected to be a decrease in arousal during the pain recovery period.	Not Supported
5f. Females would have greater arousal during pain induction than during baseline; however, female arousal would not decrease as drastically as for males during the pain recovery period.	Not Supported
5g. Catastrophizing, gender, and time would interact, such that for low catastrophizing females, arousal would decrease slightly during the recovery period, but would not decrease for the high catastrophizing females. In contrast, for low catastrophizing males, arousal would decrease over time to a greater extent than for the low catastrophizing females, and would decrease only slightly for the high catastrophizing males.	Not Supported

Results of the main analysis of this study examined differences in arousal for high and low catastrophizers over the baseline, pain induction, and pain recovery periods, showing significant main effects of time for HR and GSR. That is, HR and GSR increased in response to pain induction and decreased during the pain recovery period. These results are consistent with research suggesting that pain activates the stress response (e.g., Melzack, 1999) and also those studies which showed that pain induction increased psychophysiological reactivity (e.g., al'Absi & Petersen, 2003; Dixon et al., 2004; Donaldson et al., 2003).

Results also indicated that high catastrophizers had significantly higher HR than low catastrophizers during the pain induction period, but not during the baseline or pain recovery periods. Although not significant, there was a trend such that high catastrophizers had higher HR during the baseline and recovery periods as well (see Figure 9). Furthermore, upon examining HR over the pain induction period, high catastrophizers had higher HRs for the final 40 seconds of the 60-second pain induction period, but did not differ significantly during the first 20-second interval, although there was a trend to this effect (see Figure 10). Running counter to the study hypotheses, none of the other HR by catastrophizing effects were significant. With regard to GSR, there was no effect of catastrophizing nor were there any other significant results, with the exception of the significant main effect of time discussed above. However, as mentioned previously, the lack of findings for GSR may be because HR is a better measure of arousal when dealing with cold pressor pain induction (Lovallo, 1975). Thus, for the remainder of the discussion pertaining to the findings from this hypothesis, only HR will be discussed.

Although catastrophizing was associated with greater HR during the pain induction period, catastrophizing was not related to HR during the baseline period or the pain recovery period, which contradicts the proposed model (see Figure 1). It was initially hypothesized that high catastrophizers would exhibit higher arousal during the baseline and recovery periods. Although differences do appear to exist in arousal between high and low catastrophizers during the baseline and recovery periods, these effects were not significant.

One possibility for the lack of differences in baseline arousal in high and low catastrophizers concerns the timing of arousal for catastrophizers. In particular, it may be that catastrophizing cognitions can be somewhat dormant or counteracted by other cognitive mechanisms until pain is actually experienced by the individual. This suggestion does not run counter to the appraisal model of catastrophizing, but rather suggests that there may be other defensive cognitive activity at work during the threat of impending pain designed to protect the individual against catastrophic thinking and negative emotional responses until pain becomes an undeniable focus of attention. In other words, it is not until the threat of pain becomes imminent that the task is appraised as threatening and anticipatory stress responses are manifested.

Indeed, Sullivan, Rodgers, et al. (2001) reported that catastrophizers significantly underestimated pain in the period leading up to an impending experimental acute pain situation. Sullivan, Rodgers, et al. suggested that perhaps catastrophizers find the process of negatively appraising the upcoming task as affectively taxing, so they actively use defensive processes, such as denial, to reduce their catastrophizing in regard to upcoming experimental pain. Furthermore, Waxman, Tripp, Smith, Davidson, & Hsieh (2005)

showed that catastrophizers underestimated upcoming experimental cold pressor task pain, similar to the findings reported by Sullivan, Rodgers, et al. However, when examining patients just prior to orthopaedic knee surgery, the catastrophizing effect of underestimation was not found between the catastrophizers and non-catastrophizers for postoperative pain. Thus, when the threat of pain was "real" or ecologically valid, underestimation of upcoming pain was nullified.

The fact that only when the threat of pain is imminent that catastrophizing might result in accurate anticipation of the threat leads to a discussion of the null finding for baseline arousal between high and low catastrophizers found in the present study. Specifically, the null finding may exist because the experimental pain situation was potentially not appraised as threatening. Factors of the experimental design and procedures of the present study that may have contributed to a lack of threat regarding the impending pain task are described below.

Firstly, participants were asked to wait for the sensors to become accustomed to measuring their body, according to the equipment manufacturer recommendations, for approximately 8-10 minutes before the baseline period was recorded. During this period participants were seated directly beside the cold pressor apparatus. Thus, participants may have become acclimatized to having the cold pressor apparatus beside them. Of course, this effect could potentially work in the opposite direction for some and create greater distress as participants sit by the apparatus contemplating the forthcoming pain task, but the present data do not suggest this to be the case.

Secondly, although participants were informed that they would be participating in a pain induction task involving placing their arm into cold water, the instructions about

placing their arm into the cold water had not yet been emphasized. Thus, participants may not have viewed the task as threatening because they might not have been thinking about the pain task. The instructions also stated that the task is a safe methodology to induce controlled discomfort in a lab setting, which may have further contributed to the lack of threatening nature about the task. Indeed, there has been previous concern that the laboratory paradigms used to study pain may fail to adequately mimic the conditions under which people must endure pain (e.g., Edens & Gil, 1995; Pen & Fisher, 1994). Further, Sullivan et al. (2000) specifically noted that the nature of experimental pain studies might result in less threat than clinical pain experiences.

In addition, participants were asked to follow instructions to relax and breathe normally during the baseline period. The regulation of breathing was used to obtain a standardized baseline for other cardiac analyses. The difficulty with this part of the procedure may be that relaxation may have dampened physiological arousal. In fact, many anti-anxiety treatments use relaxation strategies that heavily emphasize diaphragmatic breathing to reduce physiological arousal that is commonly seen across panic and other anxiety disorders (e.g., Conrad & Roth, 2007; Jacobs, 2001). Thus, the combination of these study features may have lead to a dampening of the arousal response during baseline. Perhaps the relax command and breathing along with the low physical threat described in the consent combined to create low arousal through reduced negative anticipation of upcoming discomfort on part of the catastrophizers. The data do indicate a clear trend between arousal and catastrophizing in baseline, but no significant effect.

In summary, the lack of a significant catastrophizing by arousal effect in the baseline period may be influenced by the lack of threat imposed by the study design and the instructions and the documented tendency for catastrophizers to underestimate upcoming pain. Based on the preceding discussion, future research should consider examining whether increased threat during the baseline period might correspond with higher arousal. To more accurately test whether catastrophizers would exhibit arousal before a task, it may be necessary to create a situation whereby catastrophizers might be expected to appraise the impending situation as threatening. For example, having a period where anticipation of the pain stimulus is created regarding the impending pain experience might elicit greater differences in arousal between high and low catastrophizers. Future research regarding this point will be discussed in more detail in the pertinent section.

The results of the present study also did not show differences between high and low catastrophizers in HR during the recovery period, as was initially specified in the model. Examining the recovery period data, it appeared that overall HR remained higher for high catastrophizers than for low catastrophizers throughout the recovery period even though this effect was not significant (see Figure 11). This nonsignificant finding contradicts what was proposed based on the model. Furthermore, the finding is not consistent with the pain report findings of the present study, which indicated that catastrophizing was related to pain intensity reported during the recovery period.

The finding that catastrophizing did show a relationship to HR during the pain recovery is contrary to those from Edwards and Fillingim (2005), which showed that catastrophizing was associated with the change in cardiac contractility from baseline to

post-pain induction[6]. There are two possibilities for the discrepancies seen between the findings from their study and the present study: 1) Edwards and Fillingim used the Coping Strategies Questionnaire (CSQ; Rosenstiel & Keefe, 1983) to assess catastrophizing, which might have different relationships with physiology than the PCS because the PCS assesses rumination and magnification components in addition to the helplessness component that the CSQ solely assesses (Edwards et al., 2004), and 2) Edwards and Fillingim used other measures of cardiovascular activity, which did not include HR. These differences between the present study and the previous findings might have accounted for the differences in results seen with regard to the relationship between catastrophizing and physiology during the recovery period.

As with the baseline period, one possible explanation for the lack of difference seen between high and low catastrophizers in HR during the recovery period is that the amplitude of these effects may be due to the lack of threat value of the experimental pain induction paradigm (Edens & Gil, 1995; Pen & Fisher, 1994; Sullivan et al., 2000). Furthermore, once participants remove their arm from the water, sensations associated with the task dissipate readily (Edens & Gil, 1995). This rapid recovery may contribute to the lack of threat value associated with the task during the recovery period and hence, no significant differences in psychophysiological arousal between high and low catastrophizers. However, because there was a trend suggesting that high catastrophizers had higher arousal during the recovery period, it is suggested that if the threat value of the stimulus was changed, that this might result in greater differences in arousal after pain induction. For example, future studies may examine psychophysiological arousal in

[6] It should be noted that Edwards and Fillingim (2005) did not examined physiology during acute pain.

response to other acute pain situations (e.g., clinical pain), where outcomes of pain are uncertain, and may therefore result in greater threat appraisals, and resulting arousal.

Another possibility for the lack of a significant difference in HR between high and low catastrophizers during the recovery period is that during the recovery period the body is attempting to re-establish homeostasis. As mentioned previously, perhaps using a measure of purely PNS activity may show a better effect during the recovery period as once the stressor is removed, the body activates the PNS to re-establish homeostasis (Johnson et al., 1992). Although SNS activation may decrease over the pain recovery period, PNS activation may better indicate differences between catastrophizers. Differences in PNS activation may be used to explain differences in pain report during the recovery period between high and low catastrophizers. For example, perhaps in high catastrophizers the PNS may not be as active following a pain stressor as in the low catastrophizers. This would result in a decreased ability to inhibit SNS activation during the recovery period, which may manifest as higher activation. Indeed, this trend towards heightened activation during the recovery period was observed in the present study. Research has demonstrated that perseverative cognitions (i.e., thoughts that are similar in nature to catastrophizing) are related to lower PNS activation (e.g., Brosschot et al., 2006), thus this may be an avenue for future research to explore.

One of the other findings from the arousal analyses was that gender did not show any significant relation to psychophysiological arousal during the baseline, pain induction, or the recovery periods. This finding is contrary to what was hypothesized based on previous studies that showed gender effects in psychophysiological arousal (e.g., al'Absi & Petersen, 2003; Dixon et al., 2004). However, these previous studies

showed gender differences for blood pressure, such that males had higher blood pressure than females, which is related to lower pain intensity (al'Absi & Peterson; Dixon et al.). With regard to HR, gender differences were not found (Al'Absi & Petersen; Fillingim & Maxiner, 1996). Others have noted that when pain is encountered women experience an increase in HR, whereas men have an increase in blood pressure, but this finding is not reliably demonstrated (e.g., Dixon et al.). Thus, findings regarding gender differences in psychophysiological arousal are mixed.

In summary, catastrophizing and gender were both related to pain report with high catastrophizers reporting greater pain than low catastrophizers and females reporting greater pain than males. However, only the high catastrophizers had higher arousal in response to pain induction. Thus, it may be that gender effects in the pain literature may exist for different reasons than those from catastrophizing. Due to the suggested lack of differences in appraisals between women and men (Unruh, 1999), psychophysiological activity may not differ between genders in this pain paradigm.

Mediation Models (Hypothesis 6)

To summarize the results of the sixth hypothesis, the specific hypotheses and respective outcomes are displayed in Table 39.

Table 39

Hypotheses Regarding the Mediation Analyses

Hypothesis	Outcome
6a. Arousal in the baseline phase would fully mediate the relationship between catastrophizing and pain during the pain induction period.	Not Supported
6b. Arousal in the pain induction period would partially mediate the relationship between catastrophizing and pain during the pain induction period.	Not Supported
6c. Arousal in the pain induction period would partially mediate the relationship between catastrophizing and pain in the pain recovery period.	Not Supported
6d. Arousal in the pain recovery period would partially mediate the relationship between catastrophizing and pain during the pain induction period.	Not Supported

The exploratory mediation models were not significant. Firstly, because there were no relationships between catastrophizing and arousal during pain induction and pain recovery periods, only two of the original four models were conducted. The first model showed that arousal during pain induction did not mediate the relationship between catastrophizing and pain report during pain induction. The second model showed that arousal during pain induction did not mediate the relationship between catastrophizing and pain report during pain induction. Thus, contrary to the predictions based on the model, arousal does not mediate the relationship between catastrophizing and acute pain. Perhaps arousal may mediate relationships between catastrophizing and pain in chronic

pain situations where prolonged arousal may result in long-term disruptions to homeostasis and then lead to a disease state (e.g., Melzack, 1999; Salpolsky, 2007). This relationship may not transfer to laboratory acute pain situations where the arousal disrupts homeostasis for only a short period of time.

Theoretical Implications

This study has numerous theoretical implications for the relationship between catastrophizing and pain report. By studying psychophysiological arousal, it has been shown that individuals high in catastrophizing exhibit greater arousal in response to pain than individuals low in catastrophizing. Furthermore, there seems to be evidence for a trend suggesting that individuals high in catastrophizing had higher HR before and after pain as well. Thus, individuals high in catastrophizing not only report differences in subjective pain, but also manifest differences in psychophysiological arousal. Although the results do not directly support all aspects of the model proposed in Figure 1, the results of this study provide an important starting point for investigating catastrophizing as an appraisal process.

The results of this study may also have implications for theories regarding the development of chronic pain disorders. For example, Melzack (1999) suggested that prolonged activation of the stress response system may be related to the development of chronic pain disorders through muscle, bone, and neural degeneration. Further, Turk and Flor (1999) suggested that persistent arousal of the SNS may initiate and exacerbate pain episodes in chronic pain patients. In addition, Brosschot et al. (2006) proposed that perseverative cognitions lead to prolonged activation of the stress response, which in turn leads to chronic disease. Indeed, Brosschot et al. noted that prolonged activation is

associated with a number of negative health outcomes (e.g., cardiovascular disease). The results of the present study showed that catastrophizers had a heightened stress response to a pain situation. Thus, catastrophizers may have higher reactions to clinical pain situations, exhibiting higher arousal during pain and perhaps following with painful flare-ups or other acute pain episodes. In the present study, although catastrophizers did not show a significantly higher response following pain induction, there did appear to be a trend to such an effect. In clinical pain situations, catastrophizers may be more likely to have a heightened stress response following removal of the pain stimulus as they might be worrying about lasting effects and engaging in maladaptive secondary appraisal processes, such as feeling helpless to cope with their situation. Based on the evidence indicating that lasting stress is associated with disease (e.g., Brosschot et al., 2006; Melzack, 1999; Salpolsky, 2007), and if catastrophizing does indeed create higher stress not only during but also after a stressor, then in the face of chronic pain onset, where pain is persistent, it may be that catastrophizers continue on in a heightened state of arousal, thus implicating catastrophizing in the development and maintenance of chronic pain disorders (e.g., Norton & Asmundson, 2003; Sullivan, Thorn, et al., 2001; Vlaeyen, Kole-Snijders, Boeren, & Van Eek, 1995).

Clinical Implications

Catastrophizers have higher physiological activation during pain and this seems to last into the recovery period. This result could have implications for how catastrophizers deal with pain over time. As alluded to in the theoretical implications, if high catastrophizers sustain serious injury with pain, not only would they have higher arousal in response to the pain that occurred during the injury, but if the pain continues then they

may also have increased psychophysiological arousal for as a long as the pain persists. Thus, intervening with methods to reduce the stress response when faced with pain stressor may be of benefit to catastrophizers.

One treatment implication of this study would be to examine biofeedback when performing acute pain procedures on those who are high in catastrophizing. Biofeedback is a procedure that involves providing physiological feedback through which the patient is taught to alter their physiological responses (Arena & Blanchard, 2002). Because catastrophizing is associated with greater psychophysiological arousal in response to pain stressors, teaching catastrophizers to control and perhaps lower their psychophysiological responses to pain stressors may be of benefit. Indeed, studies have shown success with biofeedback in acute pain. For example, Victor et al. (1978) found that completing biofeedback training during a primary CPT resulted in decreased HR and subjective reports of pain during a second CPT. Biofeedback has also been used in the treatment of chronic pain disorders. For example, electromyographic (EMG) and thermal feedback have been used in the treatment of tension headaches, migraine headaches, and chronic low back pain (e.g., Arena & Blanchard, 2002). Whether or not reducing the level of arousal in catastrophizers specifically is of any benefit to pain reduction will have to await further research.

Cognitive-behavioural therapy (CBT) is another common method used to treat pain (Keefe, 1996). The present results provided partial support for the proposed model suggesting that catastrophizers manifest greater physiological arousal during an acute pain task. It is speculated that the mechanism of action in this association is that the appraisals made by the individual in pain are that they cannot manage or tolerate their

pain. Thus, the situation is viewed as a threat to the individual on multiple levels. Perhaps interventions incorporating a cognitive component aimed at reducing catastrophic thoughts and associated appraisals might be of benefit in the reduction of pain and the arousal associated with pain. Thorn, Sullivan, & Boothby (2002) developed a version of CBT for chronic pain patients in which they specifically targeted catastrophic thoughts. Others have applied a variation of this catastrophizing reduction intervention to acute pain situations (e.g., Ng, 2006). Investigating the application of these therapeutic techniques in clinical settings while measuring subjective pain report as well as psychophysiological arousal would be useful in the treatment of pain.

Relaxation techniques are commonly incorporated in cognitive-behavioural therapies for pain (e.g., Keefe, 1996; Thorn, 2004; Turk, 2002). Although mainly used in the treatment of chronic pain, the present results suggest that relaxation therapies may also be of benefit to those high in catastrophizing when faced with an acute pain stressor. Teaching patients prone to experiencing acute pain to cope with their pain through relaxation techniques may be helpful in reducing the amount of stress and pain associated with acute pain injuries. Indeed, relaxation treatments have been successfully used in anxiety and depressive disorders for several years, showing that when relaxation techniques are employed, stress is reduced (Jacobs, 2001).

An additional clinical implication is that the model proposed by the present study could be used by clinicians as a training aid in the delivery of CBT to help patients develop a meta-cognitive perspective on how catastrophizing may affect their body, which is a difficult task for many patients to grasp (Turk, 2002). That is, the model proposed in the present study may be helpful to illustrate to patients that their thinking

can impact directly how they experience pain – both their psychological *and* physiological reactions to the pain experience.

Limitations

Some limitations should be considered when interpreting the results of the present study. First, the present study employed the use of experimental pain induction. Although the cold pressor task is thought to be a valid and reliable measure of pain and is used widely throughout the pain literature (e.g., Edens & Gil, 1995), there has been some critique of its ecological validity. For example, Pen and Fisher (1994) noted that experimental pain induction procedures are limited in that participants are informed that the procedure is safe and participants can terminate the task at any point. Thus, these results may not generalize to clinical pain populations undergoing actual pain processes, where outcomes of acute pain are uncertain (e.g., surgery, injury) (Edens & Gil, 1995; Pen & Fisher, 1994).

A second potential limitation to the present study was the generalizability of the sample. The sample consisted of young, educated, healthy, participants. This sample may therefore not generalize to the general population or to clinical populations. Furthermore, the sample was predominantly Caucasian. Pain functions differently for different ethnic groups (Davidhizar & Giger, 2004; Hsieh, 2006). For example, Hsieh (2006) found that Chinese participants displayed lower pain tolerance and reported higher pain than European Canadians. Another study found that African American participants reported greater pain, had a lower pain tolerance, and exhibited higher cardiovascular reactivity in response to a variety of laboratory-induced acute pain induction tasks than Caucasian

participants (Cianfrini, 2005). Thus, the present results may not generalize to other ethnicities.

Another potential limitation of the present study was that catastrophizing was not assessed during the study (i.e., catastrophizing was only assessed before the study commenced). Due to the literature showing that catastrophizing may change depending on the situation (e.g., Dixon et al., 2004; Thorn et al., 2004; Turner & Aaron, 2001), those who were identified pre-study as low catastrophizers could theoretically have started to catastrophize during the study more so than would be expected based on their initial catastrophizing score. However, in this study only participants who scored within the accepted ranges of high or low catastrophizing at two time points were included in the study. Therefore, it is unlikely that any transient catastrophizers were included in the study. If they had been included, there would have been little way of controlling for this, as it was not feasible to obtain catastrophizing scores during pain due to the collection of other measures. However, due to evidence indicating that catastrophizing may act as both a situational and dispositional characteristic (Thorn et al., Turner & Aaron), measuring catastrophizing directly before or during pain induction (e.g., Dixon et al.) may have provided a better score of catastrophizing in the period surrounding the CPT.

During the study, participants placed their left hand in the water regardless of whether they were right- or left-handed because changing the equipment set-up between each participant was not feasible. Previous pain research typically uses the non-dominant hand in the cold pressor, whereas physiological research typically uses the non-dominant hand to attach the GSR sensors. However, other studies that have used psychophysiological recording during cold water immersion pain induction tasks have

used the dominant hand for the psychophysiological sensors (e.g., Dowling, 1983). Although the present study was slightly different regarding this procedure, only 10.5% of the sample was left-handed and did not fit with the procedures used in previous studies.

Another potential limitation of the study is that physiology was not recorded during the period in which participants were instructed on when they would be immersing their arm in the cold pressor. In retrospect, with regard to seeing physiological differences as a result of differences in appraisals, it would have been ideal to have these measures during this period of direct anticipation of the task, as differences in physiology between high and low catastrophizers during that anticipatory period may have been more likely to be seen.

Finally, all participants were tested with the same female experimenter. Recent studies have shown interactions between subject gender and experimenter gender with regard to subjective pain report, such that males report greater pain to female experimenters, but females reported similar degrees of pain to male and female experimenters (e.g., Aslaksen et al., 2007). However, there was no such effect for HR or GSR. Therefore, having only a female experimenter may have created greater pain report in males. However, actions were taken to ensure that there were minimal experimenter effects. For example, the experimenter was in a different room from the participant during all tasks, with the exception of when she was delivering instructions to participants. In addition, a script was followed during all contact with participants to ensure that the experimenter did not treat participants differently.

Future Research

Further research is needed to help clarify the relationship between catastrophizing and appraisals. In particular, studies manipulating the threat value of the pain stimulus would be useful to ascertain psychophysiological differences in catastrophizing before and after pain induction to inform the appraisal model of catastrophizing. Indeed, studies have shown that appraisals and psychophysiological responses can be changed based on altering instructions (Tomaka, Blascovich, Kibler, & Ernst, 1997). A future study might manipulate the instructions given to participants and measure their psychophysiological responses to those instructions. In one condition, the threat could be minimized by saying that most people can complete the task. Another condition could be neutral, giving similar instructions to what were given in the present study. A third high-threat condition could tell participants that it is a very painful task, and some participants have a really difficult time keeping their arm in for the duration. Furthermore, asking participants about the appraisals they are making would be useful to determine how they are appraising the situation (e.g., Unruh, 1999). Measuring arousal in response to varying threats and asking participants about the appraisals of the situation in vivo may help to better investigate how catastrophizing acts in increasing pain.

A main focus for future research would be to investigate other indicators of stress response – both psychological and physiological. For example, some studies have used Likert-type scales measuring perceived stress (e.g., Tomaka et al., 1993). For physiological measures, it would be interesting to investigate cortisol or immune system functioning. For example, recent research has shown relationships between catastrophizing and heightened levels of interleukin, a signalling protein involved in

immune system function (e.g., Edwards, 2008). Measuring different forms of stress responses may enable researchers to better determine how catastrophizing acts in response to pain. In relation to this, examining whether catastrophizing is related to a measure of purely PNS activity would also be useful, as HR is a measure of both SNS and PNS activation. Respiratory sinus arrhythmia (RSA; i.e., the variability of the heart rate for each respiratory cycle) is a measure of vagal tone (i.e., a measure of parasympathetic control of the heart) (Rottenberg et al., 2002). RSA thus provides a measure of PNS activity. Previous research has shown that RSA is related to perseverative cognitions, which are similar in nature to catastrophic thoughts (Brosschot et al., 2005, 2006). Examining RSA would allow for the determination of whether catastrophizing affects solely PNS functioning.

Furthermore, although the cold pressor task is widely used in the pain literature (e.g., Edens & Gil, 1995), other measures of pain induction are used as well, which future investigations may consider. For example, mechanical pressure, ischemic pain (i.e., blood flow interruption using isotonic or isometric exercises), thermal pain, and electrical stimulation pain have all been used in the pain literature as methods of acute pain induction (Edens & Gil, 1995). These other types of pain induction may elicit different psychophysiological responses than the cardiovascular response, which the cold pressor task most prominently activates (Lovallo et al., 1975), which may be useful with regard to studying differences in catastrophizing and arousal. In addition, given research indicating that the cardiovascular response is most prominent in exposures to the CPT of greater than one minute (Lovallo, 1975), perhaps increasing the amount of time in the cold pressor may attenuate differences among high and low catastrophizers.

Future research may also address the theoretical and clinical implications outlined above. For example, investigating whether there is a relationship between catastrophizing and prolonged stress following an acute injury might provide perspective on models of chronic pain. It would also be interesting to study some of the clinical applications of this study. For example, studying biofeedback or cognitive therapies specifically targeting catastrophizing and assessing the effectiveness of these types of programs would provide a useful starting point in treating this individual difference factor which is so highly associated with pain.

Finally, another direction for future research would be to consider additional covariates that were not examined in the present study. For example, personality factors, such as locus of control, may be associated with the degree to which one catastrophizes and hence experiences stress and pain. Indeed, one study showed that people who cope with their pain by catastrophizing scored higher on a chance locus of control orientation (i.e., the perception that outcomes are controlled by fate or luck) (Crisson & Keefe, 1988). Other studies have shown that an internal locus of control (i.e., the perception that one has control over outcomes) mediated the effectiveness of CBT programs in reducing depression and pain behaviour in patients with chronic low back pain (Spinhoven et al., 2004). Socio-demographic factors, such as psychiatric illnesses and abuse history, may also be useful to examine in the context of pain, considering the high comorbidity between depression and anxiety and chronic pain disorders (e.g., McWilliams, Cox, & Enns, 2003) and the relationship between abuse history and greater reports of pain (e.g., Fillingim, Wilkinson, & Powell, 1999). Lastly, medication use is another factor that was not examined presently, but would be an important factor to consider in future studies

(e.g., Dixon et al., 2004). Pain medications, psychiatric medications, and cardiovascular medications, would all be especially important to consider as they may affect one's arousal and pain responses.

Conclusions

Catastrophizing was related to greater pain report during pain induction, during pain recovery, and in retrospective reports of sensory and affective pain. Not only was catastrophizing related to greater pain report, but it was also related to higher HR during pain induction. This indicates that in response to pain, individuals high in catastrophizing exhibit a greater physiological arousal response (i.e., stress response). Furthermore, there appeared to be trends suggesting that catastrophizing may also be associated with arousal before and after pain induction. Gender was also related to greater pain report, such that women reported greater pain than men during pain induction, during pain recovery, and in retrospective reports of sensory pain. There were no gender differences in terms of affective pain report, nor were there any gender differences in terms of physiological arousal.

These results provide partial support for an appraisal model of catastrophizing, and may be useful in informing models implicating catastrophizing in the development and maintenance of chronic pain. These results also have important clinical implications regarding treatments aimed at reducing psychophysiological arousal associated with catastrophizing. Future research is needed to clarify the relationship between catastrophizing and appraisals by examining arousal before and after pain while manipulating the threat value of the pain stimulus.

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Appendix A

DEMOGRAPHICS QUESTIONNAIRE

INSTRUCTIONS: Please indicate your responses in the space provided.

1. Gender:

a) Male _____ Female _____

b) If female….approximately how many days ago was your last period?

_____

2. What is your age? _____

3. What is your ethnicity?

4. Please indicate your dominant hand (i.e., the hand you use to write with or do most things with).

Right _____ Left _____

5. Please check off any individual below that has chronic pain or chronic illness (e.g., diabetes, arthritis, Chrohn's disease, etc.). If you need more space, please feel free to write on the back of this sheet.

	Person	**Medical Issue**
	Father	
	Mother	
	Sister	
	Brother	
	Other Family Member (please specify relationship: _____)	

6. Please list any previous pain experiences you may have had in the previous 6 months and estimate the duration and intensity (how much it hurt) of your pain (e.g., broken bones, serious muscle sprains, or illnesses causing pain). If you need additional space, please write on the back of this sheet.

Pain Type / situation	Intensity of pain (0 'no pain' – 10 "worst possible pain")	Duration (approximately how many days – use 1 day if between 1-24 hours duration)

7. Have you ever had a surgery or been hospitalized for a significant injury? If so, please describe how long were you hospitalized and describe your injury. (If you need more space please write on the back of this sheet).

Appendix B

PCS

INSTRUCTIONS: Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery.

We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you have these thoughts and feeling when you are experiencing pain.

0 – Not at all
1 – To a slight degree
2 – To a moderate degree
3 – To a great degree
4 – All the time

When I'm in pain…

1. I worry all the time about whether the pain will end……. ☐
2. I feel I can't go on…….. ☐
3. It's terrible and I think it's never going to get any better….. ☐
4. It's awful and I feel that it overwhelms me…… ☐
5. I feel I can't stand it any more…… ☐
6. I become afraid that the pain may get worse…….. ☐
7. I think of other painful experiences…… ☐
8. I anxiously want the pain to go away……. ☐
9. I can't seem to keep it out of my mind……... ☐
10. I keep thinking about how much it hurts…… ☐
11. I keep thinking about how badly I want the pain to stop….. ☐
12. There is nothing I can do to reduce the intensity of the pain. ☐
13. I wonder whether something serious may happen……. ☐

Appendix C

SF-MPQ

Think of you're the pain you just experienced. Rate how much the following words describe your pain. Indicate the severity of each pain experience word by shading the circle under "None", "Mild", "Moderate", "Severe".

	None	**Mild**	**Moderate**	**Severe**
Throbbing	O_0	O_1	O_2	O_3
Shooting	O_0	O_1	O_2	O_3
Stabbing	O_0	O_1	O_2	O_3
Sharp	O_0	O_1	O_2	O_3
Cramping	O_0	O_1	O_2	O_3
Gnawing	O_0	O_1	O_2	O_3
Hot-Burning	O_0	O_1	O_2	O_3
Aching	O_0	O_1	O_2	O_3
Heavy	O_0	O_1	O_2	O_3
Tender	O_0	O_1	O_2	O_3
Splitting	O_0	O_1	O_2	O_3
Tiring-Exhausting	O_0	O_1	O_2	O_3
Sickening	O_0	O_1	O_2	O_3
Fearful	O_0	O_1	O_2	O_3
Punishing-Cruel	O_0	O_1	O_2	O_3

Appendix D

CES-D

INSTRUCTIONS: Check the statement that best describes how often you felt or behaved this way, during the past week.

		Rarely or none of the time (Less than 1 day)	Some or little of the time (1-2 days)	Occasionally or a moderate amount of time (3-4 days)	Most or all of the time (5-7 days)
1.	I was bothered by things that usually don't bother me.	❑	❑	❑	❑
2.	I did not feel like eating; my appetite was poor.	❑	❑	❑	❑
3.	I felt that I could not shake off the blues even with help from my family or friends.	❑	❑	❑	❑
4.	I felt that I was just as good as other people.	❑	❑	❑	❑
5.	I had trouble keeping my mind on what I was doing.	❑	❑	❑	❑
6.	I felt depressed.	❑	❑	❑	❑
7.	I felt that everything I did was an effort.	❑	❑	❑	❑
8.	I felt hopeful about the future.	❑	❑	❑	❑
9.	I thought my life had been a failure.	❑	❑	❑	❑

10.	I felt fearful.	❑	❑	❑	❑
11.	My sleep was restless.	❑	❑	❑	❑
12.	I was happy.	❑	❑	❑	❑
13.	I talked less than usual.	❑	❑	❑	❑
14.	I felt lonely.	❑	❑	❑	❑
15.	People were unfriendly.	❑	❑	❑	❑
16.	I enjoyed life.	❑	❑	❑	❑
17.	I had crying spells.	❑	❑	❑	❑
18.	I felt sad.	❑	❑	❑	❑
19.	I felt that people disliked me.	❑	❑	❑	❑
20.	I could not get “going”.	❑	❑	❑	❑

Appendix E

STAI
PART 1

INSTRUCTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel ***right now,*** that is, ***at this moment***. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

(1) Not at all; (2) Somewhat; (3) Moderately so; (4) Very much so

Right now, at this moment…

1.	I feel calm……..	1	2	3	4
2.	I feel secure……	1	2	3	4
3.	I am tense……..	1	2	3	4
4.	I feel strained…….	1	2	3	4
5.	I am at ease……	1	2	3	4
6.	I feel upset…….…….	1	2	3	4
7.	I am presently worrying over possible misfortunes.……..	1	2	3	4
8.	I feel satisfied…….	1	2	3	4
9.	I feel frightened……..	1	2	3	4
10.	I feel comfortable……...	1	2	3	4
11.	I feel self-confident……	1	2	3	4
12.	I feel nervous…….	1	2	3	4
13.	I am jittery…….	1	2	3	4
14.	I feel indecisive…….....	1	2	3	4
15.	I am relaxed……...	1	2	3	4

16. I feel content…….. 1 2 3 4

17. I am worried……..... 1 2 3 4

18. I feel confused…… 1 2 3 4

19. I feel steady……..... 1 2 3 4

20. I feel pleasant…… 1 2 3 4

STAI
PART 2

INSTRUCTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you generally feel.

(1) Not at all; (2) Somewhat; (3) Moderately so; (4) Very much so

Generally…

1.	I feel pleasant…….	1	2	3	4
2.	I feel nervous and restless……..	1	2	3	4
3.	I feel satisfied with myself…….	1	2	3	4
4.	I wish I was as happy as others seem to be……	1	2	3	4
5.	I feel like a failure……..	1	2	3	4
6.	I feel rested……	1	2	3	4
7.	I am calm cool and collected…….	1	2	3	4
8.	I feel that difficulties are piling up so that I cannot overcome them…….	1	2	3	4
9.	I worry too much over something that doesn't really matter.....	1	2	3	4
10.	I am happy…….	1	2	3	4
11.	I have disturbing thoughts…….	1	2	3	4
12.	I lack self-confidence……	1	2	3	4
13.	I feel secure……	1	2	3	4
14.	I make decisions easily……..	1	2	3	4
15.	I feel inadequate……	1	2	3	4
16.	I am content……	1	2	3	4

17. Some unimportant thought runs through my mind and bothers me…….. 1 2 3 4

18. I take disappointments so keenly that I can't put them out of my mind……... 1 2 3 4

19. I am a steady person……... 1 2 3 4

20. I get in a state of turmoil as I think over my recent concerns & interests…….. 1 2 3 4

Appendix F

Pre-Study Letter of Information
Cold Pressor Pain and Psychophysiological Activation

This research is being conducted by Tara Haley, who is a Master's student working with Dr. Dean Tripp, Assistant Professor, of the Departments of Psychology, Anesthesiology, and Urology at Queen's University in Kingston, Ontario.

The purpose of filling out this questionnaire is to determine your eligibility for a study investigating coping and psychophyiological activation involved in pain perception in an acute pain induction task. In the screening for the study you will be asked to complete a short questionnaire.

We estimate that it will take approximately 5 minutes to complete this task. There are no known physical, psychological, economic or social risks associated with this experiment. This research has been cleared by the Queen's University Research Ethics Board.

Your participation is completely voluntary. Although it be would be greatly appreciated if you would answer all material as frankly as possible, you should not feel obliged to answer any material that you find objectionable or that makes you feel uncomfortable. You may also withdraw at any time with no effect on your standing in school.

We will keep your responses confidential. We will store the data from the questionnaires and the videos in a locked filing cabinet and on computer data files containing no personal identifiers until the raw data is no longer needed, at which time it will be destroyed. Only experimenters in the Pain Lab will have access to this area. To help us ensure confidentiality, please do not put your name on any of the research study answer sheets. The data may also be presented in professional psychological journals, or at scientific conferences, but any such presentations will be of general findings and will never violate confidentiality. Should you be interested, you are entitled to a copy of the findings.

In the event that you have any complaints, concerns, or questions about this research, please feel free to contact Tara Haley (2th2@qlink.queensu.ca), Dr. Dean Tripp (533-6955, trippd@post.queensu.ca), the Head of the Department of Psychology (533-2492), or Dr. Albert Clark, Chair of the Queen's University Health Sciences and Affiliated Teaching Hospitals Research Ethics Board at (613) 533-6081.

Again, thank you. Your interest in participating in this research study is greatly appreciated.

Dr. Dean Tripp
Assistant Professor

Tara Haley
Master's Student

Appendix G

Pre-Study Consent Form
Cold Pressor Pain and Psychophysiological Activation

Name (please print clearly): _____

1. I have read the Letter of Information and have had any questions answered to my satisfaction.

2. I understand that I will be participating in a screening for the study called "Cold Pressor Pain and Psychophysiological Activation". I understand that this means that I will be asked to complete a questionnaire pertaining to coping and that the results from this questionnaire will be used to determine my eligibility to participate in another study pertaining to coping, physiological reactivity and pain. I also understand that I will not receive compensation for participating in the screening.

3. I understand that my participation in this study is voluntary and I may withdraw at any time.

4. I understand that every effort will be made to maintain the confidentiality of the data now and in the future. I understand that the data will be stored in a locked filing cabinet and on computer data files containing no personal identifiers until the raw data is no longer needed, at which time it will be destroyed. Only experimenters in the Pain Lab will have access to this area. I also understand that the data may also be presented in professional psychological journals, or at scientific conferences, but any such presentations will be of general findings and will never violate confidentiality.

5. I am aware that if I have any questions, concerns, or complaints, I may contact Tara Haley (2th2@qlink.queensu.ca), Dr. Dean Tripp (533-6955, trippd@post.queensu.ca), the Head of the Department of Psychology (533-2492), or Dr. Albert Clark, Chair of the Queen's University Health Sciences and Affiliated Teaching Hospitals Research Ethics Board at (613) 533-6081.

6. I have read the above statements and freely consent to participate in this research:

Signature: _____ Date: _____

Appendix H

Pre-Study Debriefing Form
Cold Pressor Pain and Psychophysiological Activation

Pain is very prevalent, debilitating, and costly to both the person experiencing the pain and to society. Thus, it is useful to study the variables associated with pain and how those variables might interact with one another.

The information in the questionnaire you just filled out gives us information on how you cope with pain. The responses you provided will enable us to determine whether you are eligible to participate in a future study investigating psychophysiological activation (e.g., heart rate) and pain.

Your participation in this study is greatly appreciated. If participating in this research has caused to feel distressed, and you would like to talk to someone about your thoughts, please contact one of the following:

Queen's Student Counseling Service	613-533-2506
TALK Distress & Information Line	613-544-1771
Canadian Mental Health Association	613-549-7027

If you have any complaints, concerns or questions about this research please feel free to contact Tara Haley (2th2@qlink.queensu.ca), Dr. Dean Tripp (613-533-6955, trippd@post.queensu.ca), the Head of the Department of Psychology (613-533-2492). If you have any concerns about your rights as a research subject, please contact Dr. Albert Clark, Chair of the Queen's University Health Sciences and Affiliated Teaching Hospitals Research Ethics Board at (613) 533-6081.

Again, thanks very much for participating in this study!

Appendix I

EXAMPLE SCRIPT

All text in tables is dialogue

In Hallway outside Craine 208:

1. Know the participant's name
2. Meet participant in front of Craine 208
(front foyer of Humphrey Hall if doors are locked)

3. Ask participant if she needs to use the washroom

We don't want to unhook the sensors during the experiment
Do you need to go to the washroom?

4. Ask the participant to take their boots off outside Craine 208

In Observation Room:

Just so you are aware, there is a video camera in the other room, so that I can see what you are doing, that way I'll be able to tell when you are done completing the questionnaires. The video will not be recorded except for during the experiment. Also, just so that I don't startle you, I will knock every time I enter the room.

1. Participant reads information sheet and signs consent form
2. Say you'll come back in 2 minutes
3. Knock and enter the room when they're finished
3. Give questionnaires in random order: Demographics, CES-D, and STAI
4. Say you'll be back in 10 minutes
5. Knock and enter the room when they're finished

In Observation Room:

**<u>Ask the participant to remove sweaters, watches and hats and to spit gum out into the garbage.</u>

1. Respiration (has to be first)
    a. Place belt around lower rib cage on mid-area of chest (for females, just below the breasts)
    b. Make sure sensor buckle is in front of the participant

c. Make sure the sensor is upside down and the wire goes over the participant's shoulder
d. Have participant exhale completely and secure belt so that it feels snug, but two fingers should be able to fit under the belt

First sensor to go on
Arms up
Around lower rib cage
Big breath out
Snug but not uncomfortable
Is that alright?

2. Heart Rate (ECG)

e. Swipe area with alcohol
f. Attach electrode conductance patches to target site (1 patch 2 inches below collar bone on right side of chest, the other patch on the side between the lower ribcage and hip bone)
g. Attach red electrode to participant's left side (participant can attach the electrode)
h. Attach white electrode to participant's right side (*white and right rhyme)
i. drape the leads over shoulders toward back of chair

Now heart rate
First sticker under right collarbone
Rubbing alcohol
Place sticker
Explain where other sticker will go *
They rub alcohol
They put sticker on
Now I'll hook up the sensors

*** if participant really does not want to expose side/flank then put the other sticker on the left side two inches under their collarbone (same as the right side)**

3. Galvanic Skin Response

j. Locate top of the 3rd and 4th finger on palm side of non-dominant hand (top of 3 separations of your finger)
k. use a gently abrasive hand wash to rub the middle and ring fingers on his or her non-dominant hand
l. Swipe area with alcohol
m. Squeeze conducting gel into both electrodes- enough so there are no air bubbles, not so much that it will seep out from electrode hole
n. Place electrodes and fasten so they will not slide around, but do not cut off circulation
o. Hand should be placed lightly on chair and not moved for duration of testing

Last sensors
Left-handed or right?
Rub with gritty handwash
Rubbing alcohol
Conducting gel
Bands should feel snug but should not cut off circulation

4. Check to make sure that the sensors are recording properly

"I am just going to go into the other room to ensure that the sensors are recording properly. I will be back in just a moment, so just sit there and relax."

Go check to ensure that everything is working in the other room. Fix anything that needs to be fixed.

Paced Breathing

In Observation Room:

"Ok, everything is working fine, so now we'll get started".

1.Move the monitor in front of the participant
2. Ask the participant to synchronize his or her breathing with a rising and falling bar which is shown on the monitor for 2 minutes
3. The participant should inhale as the bar goes up and exhale as the bar goes down

"Ok, so during this next task, you are going to be timing your breathing with this bar.

Please don't move around in the chair or move your arms because if you do it will screw up the data.

So, if you just move your arms a little right now, and look at the monitor beside you- you'll see how it messes up the data. (SHOW THEM). Do you see that?

Ok, so as you can see on the monitor, there's a bar moving up and down.

For this task you will breathe continuously in when you see the bar go up and breathe continuously out as you see the bar go down.

Try to time your breathing with the bar's movement.

Ok, let's practice. So breathe in…and out…(Do this a few times) Great!
(*Make sure they are doing this properly)

Again, please stay still in your chair and relax as much as possible- ok?

You keep breathing with the bar and I'll leave the room and I'll come back in 2 minutes –don't worry about the time.

Do you have any questions?

Ok. Keep breathing with the bar and I'll be back in 2 minutes."

4. Leave observation room and close door
5. Refer to instructions for how to record the data. Once two minutes of data have been recorded return to room.

<u>Baseline</u>

In Observation Room:

1. Instruct the participant to sit quietly and motionless (if possible) in the room for 3 minutes

"Ok that was great! For this next task we don't need the monitor, so I'll just move it out of the way.

For this task the sensors will be recording while you are calm and alone. So please just relax and breathe normally.

Again, please don't move around in the chair.

Ok, so now I'm going to leave the room for 3 minutes and when the time is up, I'll knock on the door and then come back in - you don't have to worry about the time.

Do you have any questions?

Ok, sit there and relax, and I'll see you in 3 minutes."

2. Turn off the monitor, leave observation room and close door
3. Go to the control room, refer to instructions on recording the data. Once three minutes is up, return to the observation room.
4. Knock on the door and enter.

Cold Pressor Task

In Observation Room:

1. Knock and enter the room with a clipboard in hand
2. Instruct the participant about how to perform the cold pressor task.

This is the cold pressor machine – it is filled with cold water. When I tell you to begin, put your hand on the bottom, with your fingers spread, so that your fingertips are touching the bottom of the tank. *(Demonstrate with own hand how to do it in without putting your arm in the water)*.

Once you place your arm in the water, I'd like you to keep it in for 60 seconds (1 minute) when I will ask you to remove your arm. You may feel some physical discomfort during the task and if it becomes overwhelming you can remove your arm, but it is important that you keep it in until I ask you to remove it.

Just so that I know I'm explaining this correctly, could you repeat for me in your own words what I'd like you to do.

If they don't understand, try to clarify.

Remember, you can take your arm out of the water if the physical discomfort becomes overwhelming, but please keep it in until I ask you to remove it.

Ok, so after you put your hand in the water, there will be two things for you to do. The first thing for you to do is to rate how much pain you feel on the scale from 0 to 10 in front of you whenever I say "report". Zero means no pain at all, and ten means extreme pain. The second thing for you to do is to rate how unpleasant the pain feels on the scale from 0 to 10 in front of you whenever I say "report".

Just to be clear I'll repeat this again….*repeat above paragraph*

Again, to make sure that I've explained the procedure correctly, could you briefly repeat to me what you have to do from beginning to end?

Have them say the procedure in their own words. If they don't remember a part, re-explain it, as described above. If they understand everything, then continue.

During the task we ask that you keep your eyes straight ahead and look at the two scales in front of you for the entire time. Try to stay as still as possible.
I will be in the other room, and over a microphone, instructions on when to put your hand in the water, report, and remove your arm will be played. When you are told to remove your arm, please remove your arm from the cold presor and place your arm on this towel in front of you. Please try not to move as much as possible. You will be asked to sit still for 10 minutes and just relax and breathe normally during this time.

Do you have any questions?

OK, in a moment I will tell you to put your hand in the water by saying "begin". Don't forget to rate your pain and pain unpleasantness when I say "report".

Once again, please don't move around in your chair and keep your eyes on the scales in front of you.

3. Leave the room and close the door.
4. Play the tape-recorded instructions into the microphone. Refer to instructions on recording data.
5. When the time is up you will return to the observation room.

Conclusion

In Observation Room:

1. Knock and enter the room

"Ok, that was good! Now for the last part of the study."

2. Ask the participant to complete the SF-MPQ (tell them you will come get them in 5 minutes)
3. Leave the room and close the door
4. Return after the participant is finished the questionnaire
3. Ask the participant to fill out the final consent

4. If the participant wishes for his or her data to be deleted, the experimenters will delete the data at this time
5. Debrief the participant, provide contact numbers for psychological help
6. If the participant is choosing subject pool, make sure you credit their account. However, if the participant is to be paid, give the participant $10 (whether the data was kept or deleted)
    a. Participants must sign that they received the money
7. Ask the participant if he or she has any further questions
8. Walk the participant outside to where you met them

After Participant Leaves:

In Control Room:

1. Make sure that you have saved all the data and closed all computer programs
2. Make sure doors are locked

In Observation Room

1. Make sure that all equipment is turned off
2. Make sure doors are locked

Appendix J

Letter of Information
Cold Pressor Pain and Psychophysiological Activation

This research is being conducted by Tara Haley, who is a Master's student working with Dr. Dean Tripp, Assistant Professor, of the Departments of Psychology, Anesthesiology, and Urology at Queen's University in Kingston, Ontario.

This study is being conducted to investigate coping and psychophysiological activation involved in pain perception in an acute pain induction task. In this study, you will be asked to fill out a demographics form and a series of questionnaires. We then ask you to participate in a cold pressor pain induction task, where you will place your hand in cold water (approximately 1^{o}C-3 ^{o}C) for approximately 1 minute and verbally rate your pain experience, while your psychophysiological responses are recorded. For this study, 3 measures of your physiological state will be recorded: (1) skin conductance: the small changes in the amount of moisture or perspiration on the surface of two fingers, (2) heart rate: the rhythmic pattern of your heart's electrical impulses that cause it to contract and pump blood throughout your body, and (3) respiration rate: the pace and depth of each inhale and exhale. To record these measures, we will need to affix sensors in the following manner: (1) The two middle fingers on your left hand will need to be cleaned and the sensor location will be gently wiped once or twice with an exfoliant gel to remove any excess skin (this is important for clean measurements). A small dab of gel will be placed on the sensor before it is secured on to each finger by means of a Velcro strap firmly enough to keep it in place but not tight. (2) Two small sensors about the size of a Loonie will be placed just beneath each collarbone to measure heart activity. (3) A stretchy fabric strap will be looped around your upper torso, just beneath the heart sensors and under your armpits, to measure respiration. The strap will fit snugly but not tightly and is not uncomfortable. After a very short period, people generally forget that these sensors are there. Every effort will be made to make sure you are comfortable both as the sensors are being placed on your body and during the experiment.

You will be recorded using a video recorder which will be used to ensure that you are performing the task correctly while the experimenter is not in the room. With your permission, your actions may later be coded by authorized judges from the Pain Lab to be used in future studies.

We estimate that it will take approximately 45 minutes to complete these tasks. There are no known physical, psychological, economic or social risks associated with this experiment. There will be induced pain through the immersion of your hand in cold water, but this activity DOES NOT cause any permanent tissue damage and the sensation will be begin to dissipate as soon as you remove your hand from the cold water. This research has been cleared by the Queen's University Research Ethics Board.

Your participation is completely voluntary. Although it be would be greatly appreciated if you would answer all material as frankly as possible, you should not feel obliged to answer any material that you find objectionable or that makes you feel uncomfortable. You may also withdraw at any time without any consequences or penalty. We will indicate that you have earned 1 extra credit for Psychology 100 or we will pay you $10 in exchange for your participation.

We will keep your responses confidential. We will store the data from the questionnaires and the videos in a locked filing cabinet and on computer data files containing no personal identifiers

until the raw data is no longer needed, at which time it will be destroyed. Only experimenters in the Pain Lab will have access to this area. To help us ensure confidentiality, please do not put your name on any of the research study answer sheets. The data may also be presented in professional psychological journals, or at scientific conferences, but any such presentations will be of general findings and will never violate confidentiality. Should you be interested, you are entitled to a copy of the findings.

In the event that you have any complaints, concerns, or questions about this research, please feel free to contact Tara Haley (2th2@qlink.queensu.ca), Dr. Dean Tripp (613-533-6955, trippd@post.queensu.ca), the Head of the Department of Psychology (613-533-2492). If you have any concerns about your rights as a research subject, please contact Dr. Albert Clark, Chair of the Queen's University Health Sciences and Affiliated Teaching Hospitals Research Ethics Board at (613) 533-6081.

Again, thank you. Your interest in participating in this research study is greatly appreciated.

Dr. Dean Tripp
Assistant Professor

Tara Haley
Master's Student

This portion will be detached and kept by the experimenters.

Name: _____ Date: _____

Please indicate which option you would prefer by initialing on the appropriate line:

____ I would like to receive 2 subject pool credits (i.e., 1% towards my final grade in Psyc 100)

____ I would like to receive $10

Appendix K

Consent Form
Cold Pressor Pain and Psychophysiological Activation

Name (please print clearly): _____

1. I have read the Letter of Information and have had any questions answered to my satisfaction.

2. I understand that I will be participating in the study called "Cold Pressor Pain and Psychophysiological Activation". I understand that this means that I will be asked to complete a demographics questionnaire and a series of measures pertaining to coping. I also understand that I will be asked to place my hand in cold water and verbally rate my pain experience, for the purpose of investigating coping mechanisms used in the pain experience. In addition, I understand that my psychophysiological responses (i.e., heart rate, breathing rate, and skin conductance) will be recorded). Furthermore, I understand that I will be videotaped during the study, and I will indicate below what I would like done with the videotaped data. Lastly, I understand that I am not to participate if I have any history of frostbite, hypertension, cardiovascular problems, chronic pain, or if I have previously participated in a cold pressor task this year.

3. I understand that my participation in this study is voluntary and I may withdraw at any time.

4. I understand that every effort will be made to maintain the confidentiality of the data now and in the future. I understand that the data will be stored in a locked filing cabinet and on computer data files containing no personal identifiers until the raw data is no longer needed, at which time it will be destroyed. Only experimenters in the Pain Lab will have access to this area. I also understand that the data may also be presented in professional psychological journals, or at scientific conferences, but any such presentations will be of general findings and will never violate confidentiality.

5. Please **initial** any of the following options that apply (you will be presented with these options again at the end of the study and will be allowed to change them at that time):

   ____ I will allow the information collected in this study, including videotaped images of myself to be used for public display only for research purposes
   ____ I will allow Dr. Dean Tripp to keep the information collected in this study, including videotaped images of myself, so that they can conduct further analysis of this information
   ____ I do not want videotaped images of myself to be viewed by anyone but researchers associated with data analysis in this particular study
   ____ I do not want videotaped images of myself used in future investigations, please destroy them

I am aware that if I have any questions, concerns, or complaints, I may contact Tara Haley (2th2@qlink.queensu.ca), Dr. Dean Tripp (613-533-6955, trippd@post.queensu.ca), the Head of the Department of Psychology (613-533-2492). If I have any concerns about my rights as a research subject, I understand I can contact Dr. Albert Clark, Chair of the Queen's University Health Sciences and Affiliated Teaching Hospitals Research Ethics Board at (613) 533-6081.

I have read the above statements and freely consent to participate in this research:

Signature: _____ Date: _____

Appendix L

Debriefing Form
Cold Pressor Pain and Psychophysiological Activation

Pain is very prevalent, debilitating, and costly to both the person experiencing the pain and to society. Thus, it is useful to study the variables associated with pain and how those variables might interact with one another.

Pain catastrophizing is defined as "a negative mental set brought to bear during the pain experience" (Sullivan et al., 2001, p. 53). Catastrophizing is related to greater acute pain. For example, catastrophizing is related to acute pain in clinical populations (e.g., post-operative patients; Jacobsen & Butler, 1996) as well as in healthy individuals undergoing pain induction tasks, such as the pain induction task in which you just participated (e.g., Sullivan et al., 1995).

Neural imaging studies indicate that individuals who are high and low catastrophizers differ in the brain areas that are activated during pain (Gracely et al., 2004). Although interesting, such studies do not investigate the "stress response" of participants. Thus, in this study, we are interested in exploring the relationship between degree of catastrophizing and psychophysiological activation (i.e., heart rate, breathing rate, and skin conductance) before, during, and after an acute pain induction task (i.e., the cold pressor task) and whether the effects are different for men and women.

If you are interested in this area of research you may wish to read the following references:

Gracely, R. H., Geisser, M. E., Giesecke, T., Grant, M. A. B., Petzke, F., Williams, D. A, et al. (2004). Pain catastrophizing and neural responses to pain among persons with fibromyalgia. *Brain, 127,* 835-843.

Sullivan, M. J. L., Thorn, B., Haythornthwaite, J. A., Keefe, F., Martin, M., Bradley, L. A., et al. (2001). Theoretical perspectives on the relation between catastrophizing and pain. *The Clinical Journal of Pain, 17,* 52-64.

Your participation in this study is greatly appreciated. If participating in this research has caused you to feel distressed, and you would like to talk to someone about your thoughts, please contact one of the following:

Queen's Student Counseling Service	613-533-2506
TALK Distress & Information Line	613-544-1771
Canadian Mental Health Association	613-549-7027

If you have any complaints, concerns or questions about this research please feel free to contact Tara Haley (2th2@qlink.queensu.ca), Dr. Dean Tripp (613-533-6955, trippd@post.queensu.ca), the Head of the Department of Psychology (613-533-2492). If you have any concerns about your rights as a research subject, please contact Dr. Albert Clark, Chair of the Queen's University Health Sciences and Affiliated Teaching Hospitals Research Ethics Board at (613) 533-6081.

Again, thanks very much for participating in this study!

This portion will be detached and kept by the experimenters.

Name: _____ Date: _____

Please re-indicate how you would like us to use your videotaped data in the future (if you change your mind about these options in the future please contact either Tara Haley or Dr. Dean Tripp and we will change your options).

Please **initial** any of the following options that apply (you will be presented with these options again at the end of the study and will be allowed to change them at that time):

____ I will allow the information collected in this study, including videotaped images of myself to be used for public display only for research purposes
____ I will allow Dr. Dean Tripp to keep the information collected in this study, including videotaped images of myself, so that they can conduct further analysis of this information
____ I do not want videotaped images of myself to be viewed by anyone but researchers associated with data analysis in this particular study
____ I do not want videotaped images of myself used in future investigations