PSYCHOSOCIAL MEDIATORS AND MODERATORS OF THE

CATASTROPHIZING-PAIN RELATIONSHIP

IN INTERSTITIAL CYSTITIS/BLADDER PAIN SYNDROME (IC/BPS)

by

Abi Muere

A thesis submitted to the Graduate Department in Psychology

in conformity with the requirements for the

Degree of Master of Science

Queen’s University

Kingston, Ontario, Canada

(October, 2015)

Copyright © Abi Muere, 2015
Abstract

Interstitial Cystitis/Bladder Pain Syndrome (IC/BPS) is a urologic chronic pelvic pain syndrome without consensus on its etiology or treatment. Present biomedical outcomes for IC/BPS remain poor. There is growing recognition within the medical field of the significant role psychosocial factors play in the chronic pain experience. Catastrophizing, behavioural coping strategies, and depressive symptomology are all noted for their negative impact on chronic pelvic pain outcomes. In particular, the relationship between catastrophizing and pain, long recognized in other chronic pain populations, has recently been highlighted in IC/BPS research. The primary aim of this study was to identify the conditional processes through which catastrophizing predicts pain. Using the transactional model of stress and coping and the fear of re-injury model as theoretical frameworks, a moderated multiple-mediation model was tested which predicted that illness-focused and wellness-focused behavioural coping strategies mediate the relationship between catastrophizing and pain, and that these mediating effects were stronger among individuals with higher levels of depressive symptomology. Female patients diagnosed with IC/BPS (N=341; 49.77 ± 14.49 years) were recruited from tertiary care urology clinics in Canada, USA, Taiwan, Denmark, and India. Participants completed questionnaires measuring levels of pain, catastrophizing, behavioural coping strategy usage, and depressive symptomology. Illness-focused behavioural coping strategies were found to mediate the relationship between catastrophizing and pain (both sensory and affective). Additionally, depressive symptomology moderated the mediating effect of illness-focused behavioural coping on the relationship between catastrophizing and affective pain. The relationships between pain, catastrophizing, behavioural coping strategies, and depressive symptomology are discussed in
relation to the extant chronic pain literature, along with clinical implications, limitations, and areas for future research.
Acknowledgements

First and foremost, I would like to thank Dr. Dean Tripp for his continual guidance on this thesis and with all my work in the Pain Research Lab. Thank you for the generosity of your time and the support you have provided me over these past two years. Our insightful conversations on chronic pain continually remind me to stay curious and inspire me to raise the standards I hold as a researcher. I look forward to continuing our work together during my doctoral studies. Additionally, I would like to thank the Pain Research Lab, especially Laura Katz, Adrijana Krsmanovic, and Hayley Yurgan, for the support and camaraderie they have provided me.

I would like to thank my current committee members Dr. Caroline Pukall and Dr. Linda Booij, as well as Dr. Jill Jacobson for their feedback and guidance on my Master’s thesis. Thank you also to OGS and CIHR for their financial support of my Master’s research.

Finally, I would like to thank my family and friends for the unconditional love and the infinite amount of joy they give me as I work towards a career in clinical psychology.
Table of Contents

Abstract.................................................................................................................................ii
Acknowledgements..............................................................................................................iv
List of Tables ........................................................................................................................viii
List of Figures .......................................................................................................................ix
List of Abbreviations ..........................................................................................................xi
Chapter 1 Introduction .......................................................................................................1
  Understanding Chronic Pain and Its Impact ......................................................................1
    Acute versus chronic pain ...............................................................................................1
    Multidimensional conceptualization of pain and its management. ................................2
    Prevalence and impact of chronic pain. ........................................................................3
Chapter 2 Methods .............................................................................................................22
  Participants.........................................................................................................................22
  Measures ............................................................................................................................24
    Demographics. ................................................................................................................24
    IC/BPS symptoms and problems. ..................................................................................24
    Pain .................................................................................................................................25
    Pain catastrophizing. ......................................................................................................25
List of Tables

Table 1. Sample Demographics (N=341) ................................................................. 23
Table 2. Differences in Quantitative Characteristics Among North American Participants and International Participants ................................................................................................................................. 35
Table 3. Differences in Qualitative Characteristics Among North American Participants and International Participants ................................................................................................................................. 36
Table 4. Mean, Standard Deviation, and Questionnaire Score Range of Questionnaires ................................................................................................................................. 37
Table 5. Comparison of Observed Eigenvalues and Randomly Generated Eigenvalue for the SF-MPQ 39
Table 6. Factor Loadings for the SF-MPQ .............................................................................................................................................................................................................................................................................. 40
Table 7. Comparison of Observed Eigenvalues and Randomly Generated Eigenvalue for the PCS .............................................................................................................................................................................................................................................................................. 42
Table 8. Factor Loadings for the PCS .............................................................................................................................................................................................................................................................................. 43
Table 9. Comparison of Observed Eigenvalues and Randomly Generated Eigenvalue for the B-CPCI .............................................................................................................................................................................................................................................................................. 45
Table 10. Factor Loadings for the B-CPCI .............................................................................................................................................................................................................................................................................. 46
List of Figures

Figure 1. Categories of visceral pain. Gastrointestinal (GI), Obstetrics and Gynecology (OB GYN), and Urologic Chronic Pelvic Pain Syndromes (UCPPS). Inflammatory Bowel Disease (IBD) and Irritable Bowel Syndrome (IBS) fall under the GI category. Interstitial Cystitis/Bladder Pain Syndrome (IC/BPS) and Chronic Prostatitis/Chronic Pelvic Pain Syndrome (CP/CPPS) fall under the UCPPS category. .......................... 5

Figure 2. The path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between catastrophizing and pain. .......................................................................................................................... 19

Figure 3. The path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between catastrophizing and pain. Depressive symptomology is predicted to moderate (Mo) the relationship between illness-focused coping and pain, and the relationship between wellness-focused coping and pain.......................................................... 20

Figure 4. The alternate path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between pain and catastrophizing. Depressive symptomology is predicted to moderate (Mo) the relationship between illness-focused coping and catastrophizing, and the relationship between wellness-focused coping and catastrophizing................................. 21

Figure 5. Scree plot depicting eigenvalues across number of potential factors of the Short Form – McGill Pain Questionnaire (SF-MPQ). .............................................................................................................. 38

Figure 6. Scree plot depicting eigenvalues across number of potential factors of the Pain Catastrophizing Scale (PCS). ................................................................................................................................. 41

Figure 7. Scree plot depicting eigenvalues across number of potential factors of the Brief Chronic Pain Coping Inventory (B-CPCI). ............................................................................................................ 44

Figure 8. The path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between catastrophizing and sensory pain. ................................................. 48

Figure 9. The path model depicts illness-focused coping as a mediator (Me) of the relationship between catastrophizing and sensory pain. Depressive symptomology is predicted to moderate (Mo) the relationship between illness-focused coping and sensory pain................................................................. 49

Figure 10. The alternate path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between sensory pain and catastrophizing. .................................................. 50

Figure 11. The alternate path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between sensory pain and catastrophizing. Depressive symptomology is predicted to moderate (Mo) the relationship between illness-focused coping and catastrophizing, and the relationship between wellness-focused coping and catastrophizing .................................................. 51
Figure 12. The path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between catastrophizing and affective pain. .......................................................... 52

Figure 13. The path model depicts illness-focused coping as a mediator (Me) of the relationship between catastrophizing and affective pain. Depressive symptomology is predicted to moderate (Mo) the relationship between illness-focused coping and affective pain. .......................................................... 53

Figure 14. The alternate path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between affective pain and catastrophizing. .......................................................... 55
List of Abbreviations

B-CPCI: Brief Chronic Pain Coping Inventory
CBT: Cognitive-behavioural therapy
CES-D: Centre for Epidemiologic Studies Depression Scale
CI: Confidence interval
CP/CPPS: Chronic Prostatitis/Chronic Pelvic Pain Syndrome
CPA: Conditional process analysis
EFA: Exploratory factor analysis
GI: Gastrointestinal
IBD: Inflammatory Bowel Disease
IBS: Irritable Bowel Syndrome
IC/BPS: Interstitial Cystitis/Bladder Pain Syndrome
ICPI: Interstitial Cystitis Problem Index
ICSI: Interstitial Cystitis Symptom Index
LLCI: Lower limit of the confidence interval
Me: Mediator
Mo: Moderator
OB GYN: Obstetrics and gynecological disorders
QoL: Quality of life
PCS: Pain Catastrophizing Scale
SEM: Structural equation modeling
SF-MPQ: Short Form – McGill Pain Questionnaire
UCPPS: Urological chronic pelvic pain syndromes
Chapter 1

Introduction

Understanding Chronic Pain and Its Impact

Pain has been commonly defined as an “unpleasant sensory and emotional experience associated with actual or potential tissue damage” (Turk & Okifuji, 2010, pg. 14). It is clear from this definition that pain is much more than a simple physical sensation. When describing the qualities of pain, descriptors of pain can be divided into sensory and affective components. Sensory pain refers to specific physical sensations associated with pain and is described in terms of temporal, spatial, pressure, thermal, and other physical qualities. Sensory pain relates to the location and intensity of pain. In contrast, affective pain is the degree of emotional arousal caused by sensory pain. It relates to the aversiveness or unpleasantness of the pain experience. Affective pain is described in terms of tension and fear experienced when pain is viewed as cruel, frightening, or punishing (Jensen & Karoly, 2011; Melzack, 1987).

Acute versus chronic pain.

Pain can also be categorized as acute or chronic. Acute pain is common and refers to pain lasting for a relatively limited amount of time and resulting from injury of body tissues (e.g., stubbing a toe, muscle strains, appendicitis, or uncomplicated fractured bone). Acute pain serves as an important biological response to injury and disease and acts as an impetus to seek health care. Additionally, it tends to alleviate following pharmacological treatment (Turk & Okifuji, 2010). In contrast, chronic pain refers to pain extending for a long period of time that is likely elicited by both pathogenetic factors and factors separate from the originating cause (e.g.,
psychological factors). Chronic pain is differentiated by acute pain based on duration. The exact duration varies, though three months and six months are the most common chronological markers. Additionally, chronic pain is often resistant or not responsive to pharmacological treatment (Turk & Okifuji, 2010).

**Multidimensional conceptualization of pain and its management.**

Disease and its symptoms, including pain, have been historically conceptualized under the biomedical model of disease. This model assumes disease to be fully accounted for by biological variables, with no consideration of psychological, social, or behavioural factors of illness (Engel, 1977; Gatchel, Peng, Peters, Fuchs, & Turk, 2007). However, a biomedical model applied to the understanding and treatment of pain is incomplete, as is illustrated by our current incomplete understanding of the causes and treatment of chronic pain. Rather, today’s pain models suggest that psychosocial factors can make a significant contribution to illness beyond that of biological factors. In contrast to the biomedical model, pain-related outcomes are conceptualized by the biopsychosocial model as falling under three dimensions: biological, psychological, and social factors (Turk, Swanson, & Wilson, 2010). Psychological and social factors are often combined and conceptualized as psychosocial factors.

Pain management goals have expanded in parallel, and likely as a result of, the broadening conceptualization of pain. Prior to the present multidimensional conceptualization of pain, pain was treated through analgesic drugs, surgery, and rest. Present pain management programs’ strategies can be categorized as pharmacological (e.g., non-opioid analgesics, antidepressants), physical (e.g., surgery, acupuncture), and cognitive-behavioural (e.g., progressive muscle relaxation, cognitive restructuring). Furthermore, while historically the management of
pain aimed to eliminate all pain, present programs aim to modify pain perception, increase functioning, decrease distress, and improve coping ability (Marks, Murray, Evans, & Estacio, 2011).

**Prevalence and impact of chronic pain.**

Chronic pain is unfortunately common, costly, and associated with tremendous decreases in quality of life (QoL). In North America, pain is the most common reason for seeking health care for almost eight out of 10 patient visits to the emergency department (Marks et al., 2011; Todd et al., 2007). Chronic pain prevalence rates range from 20-37% in the Canadian adult population (Moulin, Clark, Speechley, & Morley-Forster, 2002; Schopflocher, Taenzer, & Jovey, 2011; Tripp, VanDenKerkhof, & McAlister, 2006). In the United States, the annual economic cost associated with chronic pain has been estimated to be between $560-635 billion (Relieving pain in America: a blueprint for transforming prevention, care, education, and research, 2011).

The impact of chronic pain should not be considered solely in economic terms. Chronic pain has a significant impact on patients’ personal and vocational lives. Chronic pain is associated with a lower QoL than other chronic diseases, including advanced coronary heart disease and diabetes (Choiniere et al., 2010). Chronic pain conditions are associated with reduced QoL, comorbid psychological disorders, and increased risk of suicide (Ilgen et al., 2013; Relieving pain in America: a blueprint for transforming prevention, care, education, and research, 2011). Research into causes and therapeutic targets that are useful in treating and managing chronic pain are essential to progressive healthcare.
Visceral Chronic Pain and Interstitial Cystitis/Bladder Pain Syndrome

In past medical writing, the visceral area of the body (i.e., the soft internal organs of the body, especially those contained within the abdominal and thoracic cavities) was considered to be insensitive to pain. However, research now suggests that visceral pain is the most common type of disease-related pain and is one of the most frequent reasons why individuals seek medical care for pain (Cervero & Laird, 1999). Visceral pain is associated with diminished QoL and lost workplace productivity (Halder & Locke III, 2009). Visceral pain has five key clinical characteristics: (1) it does not originate from all viscera (e.g., lung, liver); (2) it is not always linked to injury to the viscera; (3) it is a diffuse and poorly defined sensation; (4) it is referred to other areas in the body; and (5) it is accompanied by motor and autonomic reflexes (e.g., nausea, vomiting) (Cervero & Laird, 1999).

As shown in Figure 1, visceral pain can be divided into three areas: gastrointestinal (GI) pain, obstetrics and gynecological disorders (OB GYN), and urological chronic pelvic pain syndromes (UCPPS). The GI branch can be further divided into Inflammatory Bowel Disease (IBD) and Irritable Bowel Syndrome (IBS). Conditions such as endometriosis fall under the OB GYN branch. The UCPPS branch can be further divided into two distinct syndromes, Interstitial Cystitis/Bladder Pain Syndrome (IC/BPS) and Chronic Prostatitis/Chronic Pelvic Pain Syndrome (CP/CPPS). However, it should be noted that Figure 1 is a simplification of the different visceral pain conditions. In fact, there is a significant amount of overlap between visceral pain conditions in terms of co-morbidities and symptoms.

There are several areas of visceral pain research that remain mostly unexamined in visceral pain; in particular, the role of psychosocial symptoms and their influence on patient experience has become a novel and warranted area of investigation.
Figure 1. Categories of visceral pain. Gastrointestinal (GI), Obstetrics and Gynecology (OB GYN), and Urologic Chronic Pelvic Pain Syndromes (UCPPS). Inflammatory Bowel Disease (IBD) and Irritable Bowel Syndrome (IBS) fall under the GI category. Interstitial Cystitis/Bladder Pain Syndrome (IC/BPS) and Chronic Prostatitis/Chronic Pelvic Pain Syndrome (CP/CPPS) fall under the UCPPS category.

The present study focuses on IC/BPS. IC/BPS is a chronic pelvic pain syndrome characterized by cycling pain localized to the bladder and urologic symptoms of urgency, frequent voiding, dysuria (painful voiding), nocturia (frequent nighttime voiding), and incontinence (Nickel, Shoskes, & Irvine-Bird, 2009). Although men are also diagnosed with IC/BPS, female-to-male ratios are reported to be 5:1 or greater (Clemens et al., 2005; Nickel, Teichman, Gregoire, Clark, & Downey, 2005). A U.S. population-based study estimated an IC/BPS point prevalence rate between 2.7% to 6.5%, translating to 3.3 million to 7.9 million affected American adults (Berry et al., 2011). The IC/BPS prevalence rate among Canadian urology clinic outpatients has been estimated at 2.8% (Nickel et al., 2005). However, Clemens and colleagues (2005) have strongly suggested that IC/BPS may be under-diagnosed based on their study assessing the prevalence of IC/BPS symptoms in the general population.
Interstitial Cystitis/Bladder Pain Syndrome etiology.

Unfortunately for healthcare providers and patients alike, IC/BPS is a disease with no present consensus on its etiology. Landmark studies that have conducted histological examination of the bladder have found biomarkers of inflammation, such as mast cells and perineural inflammatory infiltrates (Fall, Johansson, & Vahlne, 1985; Larsen et al., 1982), while other researchers have proposed defects in the tissue lining the bladder (Parsons, Lilly, & Stein, 1991). Genetic factors have also been considered, with studies finding prevalence of IC/BPS to be 17 times higher among first degree relatives of individuals with IC/BPS than in the general population (Warren, Jackson, Langenberg, Meyers, & Xu, 2004). Other proposed causes of IC/BPS include autoimmune mechanisms and/or infections as potential triggers (for brief reviews on current theories, see Davis, Brady, & Creagh, 2014; Vij, Srikrishna, & Cardozo, 2012). Studies delineating the contributions of inflammation, structural defects, genetics, and infection in the etiology of IC/BPS remains at the forefront of urological research.

Interstitial Cystitis/Bladder Pain Syndrome treatment.

As with its etiology, there is also no universal agreement on the standard treatment of IC/BPS. Treatment of IC/BPS varies widely, with over 180 different types of therapies being reported (Rovner et al., 2000). Current treatment strategies can be classified into the following forms: conservative (including behavioural modification), oral medical therapy, intravesical drug instillation (i.e., administration of drugs directly into the bladder), neuromodulation of the sacral nerve, and surgical therapy (e.g., bladder augmentation cystoplasty) (Vij et al., 2012). Unfortunately, several systematic reviews of pharmacological treatments found many therapies to be ineffective, of limited efficacy, or to have inconclusive effects (Dimitrakov et al., 2007;
Peters, 2012). Furthermore, the more invasive treatments (e.g., intravesical drug instillation, neuromodulation, and surgery) are costly, painful, and have higher complication rates (e.g., risk of infection) (Davis et al., 2014; Vij et al., 2012).

**Interstitial Cystitis/Bladder Pain Syndrome and the Biopsychosocial Model**

It is essential to understand IC/BPS through a biopsychosocial model in which psychosocial factors are considered. Several key psychosocial risk factors are important in understanding the IC/BPS experience, with catastrophizing, behavioural coping strategies, and depressive symptomology all associated with patient outcomes (Nickel et al., 2008; Nickel et al., 2010; Tripp, Nickel, et al., 2006; Tripp et al., 2012). Urological researchers and clinicians alike advocate the use of a “UPOINT” classification system that places patients into one or more of the following domains based on their clinical symptom phenotype: Urological, Psychosocial, Organ specific, Infection, Neurological/systemic, and Tenderness, which forms the UPOINT mnemonic. The UPOINT system has been regarded as somewhat successful in classifying the heterogeneous IC/BPS patient population and as an aid in guiding treatment (Nickel et al., 2009; Shoskes, Nickel, Rackley, & Pontari, 2009). Each domain is associated with specific symptoms and suggestions for therapy. The psychosocial domain includes patients with clinical depression, social problems, or identifiable maladaptive coping mechanisms, with an emphasis on pain catastrophizing. This novel inclusion of a psychosocial domain in the urological literature highlights the growing recognition within the medical field for the role of psychosocial factors in the development and maintenance of pain associated with IC/BPS.
**Catastrophizing.**

Catastrophizing is a prominent cognitive factor associated with chronic pelvic pain. Pain catastrophizing is a negative pain appraisal process that has been defined as an “exaggerated negative orientation” towards actual or anticipated pain experiences (Sullivan, Bishop, & Pivik, 1995, p. 524). Sullivan et al. (1995) have suggested that this catastrophizing can be viewed as a construct comprised of three associated but not redundant dimensions: rumination, magnification, and helplessness. Rumination involves focusing on the threat value of a painful experience, while magnification involves exaggerating this threat value (Sullivan et al., 1995). Helplessness involves evaluating one’s ability to effectively deal with the pain (Sullivan et al., 1995).

There is a robust association between catastrophizing and patient outcome, which has been demonstrated in different experimental designs and across various pain populations. Studies have reported significant positive associations between catastrophizing and induced pain (Sullivan et al., 1995), dental pain (Sullivan & Neish, 1999), chronic neck pain (Thompson, Urmston, Oldham, & Woby, 2010), chronic musculoskeletal pain (Benyon, Muller, Hill, & Mallen, 2013), chronic prostatitis/chronic pelvic pain (Tripp, Nickel, et al., 2006), and pain in children with physical disabilities (Engel, Wilson, Tran, Jensen, & Ciol, 2013). Additionally, catastrophizing and disability have been positively associated among individuals suffering from spinal cord injury-related pain, whiplash associated disorder, fibromyalgia (Borsbo, Gerdle, & Peolsson, 2010), chronic back pain (Sullivan, Stanish, Waite, Sullivan, & Tripp, 1998), and chronic musculoskeletal pain (Benyon et al., 2013). Compared to healthy controls, individuals with IC/BPS report higher levels of catastrophizing, with catastrophizing strongly correlated with stress, anxiety, and depressive symptomology (Nickel et al., 2010) and more areas of pain in the
body (Tripp et al., 2012). In conclusion, catastrophizing has been associated with greater distress and pain in many patient groups, including IC/BPS.

**Coping.**

Another important factor in examining the cognitive and behavioural responses associated with patient outcomes is coping. Coping theory has highlighted that behavioural strategies are important in managing stress (Lazarus & Folkman, 1984). In pain research, behavioural coping strategies are viewed as essential mechanisms in understanding patient pain. For the past two decades, pain researchers have been conceptualizing and describing three distinct behavioural coping categories: wellness-focused (e.g., task persistence), illness-focused (e.g., guarding), and neutral coping (e.g., seeking social support) (Jensen, Turner, Romano, & Strom, 1995).

Research on the effects of behavioural illness-focused coping strategies has been intriguing but somewhat inconsistent. Behavioural illness-focused coping strategies have been found to be significantly associated with depressive symptomology and greater pain interference among a sample of veterans with a variety of chronic pain diagnoses (Tan, Teo, Anderson, & Jensen, 2011). Greater use of pain-contingent rest and asking for assistance (two specific types of illness-focused coping strategies) were associated with greater pain interference among participants with chronic low back pain (Finan, Burns, Jensen, Nielson, & Kerns, 2012). Greater use of guarding, another behavioural illness-focused coping strategy, significantly predicted pain interference among older adults with chronic pain (Chan, Hadjistavropoulos, Carleton, & Hadjistavropoulos, 2012) and has been associated with physical disability and depressive symptoms among individuals suffering from chronic pain in various primary sites of pain.
Further, in a study comparing chronic pain patients with lower versus higher levels of activity, individuals who reported lower levels of activity also reported significantly greater pain and distress pre-treatment (McMorrow et al., 2010). Behavioural illness-focused coping strategies predicted slower rates of recovery from short-term neck pain and disability, while no such association was found for more active/wellness-focused coping strategies (Carroll, Ferrari, Cassidy, & Cote, 2014).

Within the area of visceral pain research, several studies using a CP/CPPS population have found associations between behavioural illness-focused coping strategies and negative patient outcomes. In a study examining prediction of CP/CPPS patient QoL, greater pain contingent resting was the most robust predictor of poorer physical QoL functioning (Nickel et al., 2008). Furthermore, behavioural illness-focused coping strategies were found to mediate the relationship between pain and QoL (both physical and mental) (Krsmanovic et al., 2014). Pain contingent rest was also shown to be the strongest predictor of patient reported disability in a CP/CPPS population (Tripp et al., 2006), suggesting that behavioural coping may be important for CP/CPPS adjustment, as with other pain conditions. In sum, use of behavioural illness-focused coping strategies has been consistently associated with lower indices of patient outcome (e.g., pain interference, disability, lower QoL).

The results examining the effects of behavioural wellness-focused coping strategies are less conclusive. Studies such as that of Hadjistavropoulos and colleagues (1999) have found greater use of behavioural wellness-focused coping strategies to be associated with less interference in daily tasks and activities. As well, greater use of task persistence, a specific behavioural wellness-focused coping strategy in which patients continue with their daily activities in spite of pain, was associated with less pain interference among chronic low back
pain patients and fewer depressive symptoms (Finan et al., 2012). Task persistence has also been found to predict continued employment among adults suffering from chronic pain (Karoly, Ruehlman, & Okun, 2013). However, in a study with older adults suffering from chronic pain, greater use of coping self-statements, a behavioural wellness-focused coping strategy in which an individual thinks of a coping statement (e.g., “The pain will get better”), was found to be a positive predictor of pain interference (Chan et al., 2012). Similarly, Tan and colleagues (2011) found that greater levels of adaptive (i.e., wellness-focused) coping strategies, such as exercising/stretching and pacing, were associated with higher levels of pain intensity among veterans with mixed chronic pain diagnoses. Finally, others have suggested that neither behavioural illness-focused coping nor wellness-focused coping strategies were associated with improvement after treatment (Jensen, Turner, & Romano, 1994) or showed little ability to predict changes in pain intensity (Weijenborg, Ter Kuile, Gopie, & Spinhoven, 2009).

Thus, in contrast to behavioural illness-focused coping strategies, the use of behavioural wellness-focused coping strategies have been less conclusive, with some studies reporting associations with positive patient outcome (e.g., continued employment) and others reporting associations with lower indices of patient outcome (e.g., pain interference). Behavioural wellness-focused pain coping strategies have yet to be examined in IC/BPS, though a recent study found no association between behavioural wellness-focused coping and mental QoL among a CP/CPPS sample (Krsmanovic et al., 2014). Furthermore, behavioural wellness-focused coping did not mediate the relationship between pain and physical QoL.
Depression.

Pain provokes an emotional response. Greater levels of pain increase the likelihood of experiencing high anxiety, irritability, and/or agitation. These feelings are suggested as a natural response to pain and, as pain reduces, it is expected that this stress response is also likely to subside. However, in cases of chronic pain, wherein pain does not tend to remit or does not remit completely, patients will often report feeling persistent anxiousness and stress, which, over time, can manifest in emotional difficulties often associated with depression (Marks et al., 2011).

Although high rates of generalized anxiety disorder, post-traumatic stress disorder, and substance use have been reported among individuals suffering from chronic pain, major depression is described as the most common psychological disorder associated with chronic pain (Demyttenaere et al., 2007; Holmes, Christelis, & Arnold, 2012). Population-based studies suggest that 35% of individuals who reported experiencing chronic pain have comorbid depression (Miller & Cano, 2009). A national Canadian study estimated the prevalence of depression among individuals diagnosed with a chronic pain condition to be 11.3%, compared to 5.3% among pain-free individuals. Furthermore, women reported higher rates of comorbid chronic pain and depression than men (Munce & Stewart, 2007).

Women with IC/BPS were found to report more depressive symptoms than healthy female controls (Nickel et al., 2010). Among female patients with IC/BPS, those who reported higher levels of depressive symptoms also endorsed more body pain sites (Tripp et al., 2012). In a clinical study it was found that women with IC/BPS who had a recent history of depression or engaged in catastrophizing reported higher levels of pain (Nickel et al., 2009). Rabin and colleagues reported that greater levels of depressive symptoms among women with IC/BPS was
associated with decreased self-efficacy for coping with pain (Rabin, O'Leary, Neighbors, & Whitmore, 2000).

Importantly, several pain studies have indicated that depression may have significant moderating effects on pain; that is, the relationships between pain and psychosocial factors of interest are stronger among individuals suffering from depression. For example, clinical depression has been reported to moderate the relationship between ethnic differences and pain related to health problems (Hernandez & Sachs-Ericsson, 2006). Hispanic participants reported more pain compared to Caucasian participants and this difference was greater among clinically depressed participants. Further, amongst female adolescents suffering from recurrent headaches, the relationship between stress and head pain was stronger among participants with higher levels of depressive symptomology (Bjorling, 2009). In a separate study sampling male patients with chronic low back pain, Weickgenant and colleagues (1993) found that participants suffering from concurrent chronic pain and depression were more likely to engage in passive behavioural coping behaviours (i.e., avoidance and wishful thinking) than non-depressed patients and matched healthy controls. Finally, in a study examining guided self-instruction for individuals with chronic fatigue syndrome, a condition characterized by fatigue as well as pain and headaches, depressive symptomology and activity avoidance were both found to moderate treatment response (Tummers, Knoop, van Dam, & Bleijenberg, 2013). Participants with higher levels of depressive symptomology and a greater tendency to avoid activity benefited less from intervention and showed less clinical improvement. The study’s investigators note the importance of identifying moderators of pain outcomes in order to enhance treatment efficiency. In summary, patients suffering from various pain conditions who also had comorbid depression
or higher levels of depressive symptomology showed lower indices on patient outcome than patients without depression (e.g., greater pain, more passive coping, less improvement).

Theoretical Models to Understand the Role of Psychosocial Variables

The biopsychosocial model of pain advocates the importance of biological and psychosocial factors, but this study incorporated other theoretical models in order to better understand how psychosocial factors interact on the outcome of pain. Two models often used to explain pain outcomes and processes are the transactional model of stress and coping (Lazarus & Folkman, 1984) and the fear of re-injury model (Lethem, Slade, Troup, & Bentley, 1983).

Transactional model of stress and coping.

According to Lazarus and Folkman’s (1984) transactional model of stress and coping, the ways in which an individual appraises a stressful situation will strongly influence their coping process and emotional reaction. Catastrophizing is considered an appraisal of a pain experience. It is suggested that individuals engage in two related types of appraisals: primary and secondary. Primary appraisals are initial evaluations that categorize a situation as irrelevant, benign/positive, or stressful based on its significance to the individual’s well-being. Situations that are evaluated as threatening or involving potential harm or loss can evoke fear, anxiety, and anger. In contrast, stressful situations which are viewed as challenges, and not as a threat, produce feelings of excitement and eagerness. Following recognition of a threatening situation (i.e., primary appraisal), a secondary appraisal is suggested to occur in which the individual evaluates which coping options are available, the likelihood of success for each coping option, and the likelihood that they can apply the coping strategy effectively (i.e., self-efficacy). In individuals suffering
from chronic pain, who have likely been treated by a medical system that provides no cure and, in some patient reports, little hope and support for adaptation, patients can appraise the threat associated with chronic pain from a position of despair. Thus, secondary appraisals, such as pain helplessness, which is one of the dimensions of pain catastrophizing, may diminish the likelihood of employing active or behavioural wellness-focused coping strategies. Consequently, these patients may be more likely to employ strategies associated with poorer outcomes (i.e., behavioural illness-focused coping strategies).

**Fear of re-injury model.**

While the transactional model of stress and coping examines the relationship between appraisals (e.g., catastrophizing) and use of behavioural coping strategies, the fear of re-injury model focuses on the effect of enacted behavioural coping strategies on pain outcomes. According to the fear of re-injury model (Lethem et al., 1983), individuals vary in their degree of fear of pain and their coping response to this fear. Confrontation and avoidance are the two extreme responses to fear of pain or re-injury and these two responses lead to substantially different pain outcomes (Lethem et al., 1983). An individual suffering from chronic pain who adopts a confrontational behavioural coping style is more likely to engage in adaptive (wellness-focused) coping strategies, such as undertaking an increasing range of their former physical and social activities, which results in decreased pain. In contrast, individuals who adopt an avoidance-oriented (illness-focused) coping style are more likely to avoid circumstances which can lead to re-exposure to pain and thus reduce their physical and social activities. This inactivity can have both physical and psychological sequelae, including deconditioning, increased pain perception, and enhanced fear of pain. Thus, the fear of re-injury model is best viewed as a
recursive model of fear, avoidance, increased pain, and increased fear. Catastrophizing and depressed mood play significant roles in creating a vicious cycle of pain, fear, and avoidance. Catastrophizing thoughts are suggested to lead to anxiousness, dread of physical activities and movement, and, eventually, despair. Catastrophizing and depression have been suggested as critical variables of interest in poor pain outcomes (Leeuw et al., 2007; Wideman et al., 2013). These pain models suggest that appraisals of pain can impact how people react and cope with pain, but as noted in the models, they may have a recursive tendency.

**Present Research**

The basic association between pain catastrophizing and pain has been found in numerous studies. What no past studies have examined are the potential variables that may drive the relationship between catastrophizing and pain. The present study conducted an in-depth examination of the clinical relationship between catastrophizing and pain within an IC/BPS population by evaluating process variables (i.e., mediating variables) in the relationship. Based on the literature reviewed, it is suggested that behavioural coping strategies could have important implications for catastrophizing and pain. Using the transactional model of stress and coping (Lazarus & Folkman, 1984) and the fear of re-injury model (Lethem et al., 1983) as a theoretical framework, a pathway model was proposed in which the relationship between catastrophizing and pain was mediated by behavioural illness-focused coping and wellness-focused coping strategies. In other words, behavioural coping strategies were predicted to play a mechanistic role in the observed relationship between catastrophizing and pain.

Hayes (2013) has stated that while no model can ever be complete or accurate in describing human behaviour, mediation models that ignore the possibility of conditional effects
reduce the complexity of the process it is attempting to model. Moderated mediation models, or conditional process analyses (CPA), are conducted when the overarching research goal is to understand the conditional nature of a mechanism (i.e., a mediator) that links one factor to another (Hayes, 2013). A moderated mediation model tests for a conditional indirect effect in which the indirect effect of the mediator differs in direction and/or strength as a function of a moderator. In other words, moderated mediation models examine the contexts under which the indirect or mechanistic effect of the mediator strengthen, weaken, or even disappear. With this in mind, the present study aimed to extend the aforementioned multiple mediation model by examining a contextual factor which may impact the strength of the mechanistic effects of the two types of behavioural coping strategies. Based on past literature on chronic pain, depressive symptomology was predicted to moderate the mediating role of behavioural coping strategies. That is, the mediating effect of behavioural coping strategies on the relationship between catastrophizing and pain was expected to be stronger among individuals with greater levels of depressive symptomology.

The overarching aim of the present study was to identify the conditional mechanistic effects that drive the relationship between catastrophizing and pain among individuals with IC/BPS. In order to achieve this aim, data analysis was broken down into four steps: (1) Exploratory Factor Analysis; (2) Mediation Analysis (Process Analysis); (3) Moderated Mediation Analysis (CPA); (4) Examination of Alternate Models.

**Step 1: Exploratory factor analysis.**

The purpose of this exploratory factor analysis (EFA) was to use the factors derived from the EFA in the hypothesized mediation, moderated mediation, and alternate models (Steps 2-4).
Furthermore, it was necessary to conduct an EFA for each measure instead of using previously reported factor structures given the inconsistency in past chronic pain research in terms of final factor solutions reported, EFA techniques used, and sample characteristics (Davidson, Tripp, Fabrigar, & Davidson, 2008).

The present study examined the factor structure of several validated measures of pain, catastrophizing, and behavioural coping. The examined measures were the Short Form – McGill Pain Questionnaire (SF-MPQ; Melzack, 1987), the Pain Catastrophizing Scale (PCS; Sullivan et al., 1995), and the Brief Chronic Pain Coping Inventory (B-CPCI; Jensen, Keefe, Lefebvre, Romano, & Turner, 2003).

The factor structure of the Centre for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) was not examined. Although previous research has found the CES-D to have a four-factor structure (positive affect, negative/depressed affect, somatic symptoms, and interpersonal problems) (Radloff, 1977), other studies have reported optimal CES-D cut-off scores for identifying clinically meaningful levels of depressive symptomology among chronic pain populations using the CES-D total scores rather than subscales or factor scores (Geisser, Roth, & Robinson, 1997). Thus, it was decided a priori that depression would be conceptualized as a unitary construct and the total CES-D score would be used as an index of depressive symptomology.

**Step 2: Mediation analysis (process analysis).**

The purpose of Step 2 was to examine the mediating roles of the behavioural coping strategies in the relationship between catastrophizing and pain. Based on a theoretical framework provided by the transactional model of stress and coping (Lazarus & Folkman, 1984) and fear of
re-injury model (Lethem et al., 1983), a multiple-mediation model was hypothesized (see Figure 2 for the hypothesized multiple mediation model diagram). It was predicted that behavioural illness-focused coping (i.e., an avoidant coping style) and wellness-focused coping (i.e., a confrontational coping style) would each separately mediate the relationship between catastrophizing and pain. Specifically, higher levels of catastrophizing would lead to greater use of behavioural illness-focused coping strategies which would result in greater levels of pain. In contrast, higher levels of catastrophizing would lead to less use of behavioural wellness-focused coping resulting in greater levels of pain.

Figure 2. The path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between catastrophizing and pain.

**Step 3: Moderated mediation analysis (CPA).**

As recommended by Hayes (2013), Step 3 extended the multiple mediation model proposed in Step 2 by examining a potential contextual factor that could affect the hypothesized mediation. Given past clinical literature on the moderating effect of depression in chronic pain outcomes, it was predicted that depressive symptomology would moderate the indirect (mediating) effects of the two behavioural coping strategies. That is, the differing effects of behavioural illness-focused coping and wellness-focused coping would be more pronounced.
among participants suffering from greater levels of depressive symptoms (see Figure 3 for the hypothesized moderated mediation model).

![Diagram](attachment://diagram.png)

*Figure 3.* The path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between catastrophizing and pain. Depressive symptomology is predicted to moderate (Mo) the relationship between illness-focused coping and pain, and the relationship between wellness-focused coping and pain.

**Step 4: Alternate models.**

The study also evaluated an alternate model in which pain predicted catastrophizing (i.e., the predictor and outcome variables are switched; see Figure 4). Given the study’s cross-sectional design, an alternate model must be considered in which pain predicts catastrophizing, as opposed to the study’s hypothesis that catastrophizing predicts pain. It is plausible that individuals with IC/BPS who suffer from higher levels of pain will have a greater tendency towards catastrophizing. Thus, an alternate model was proposed in which the direction of the model was reversed: pain predicts catastrophizing and, in turn, behavioural coping strategies mediate this relationship. For this alternate model, the moderating role of depressive symptomology was also examined.
Figure 4. The alternate path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between pain and catastrophizing. Depressive symptomology is predicted to moderate (Mo) the relationship between illness-focused coping and catastrophizing, and the relationship between wellness-focused coping and catastrophizing.
Chapter 2

Methods

Participants

A total of 341 women participated in this study (Canada, \( n=157 \); United States, \( n=136 \); Taiwan, \( n=27 \); Denmark, \( n=15 \); India, \( n=6 \)). In order to be eligible for the study, participants had to be diagnosed with IC/BPS by an attending urologist according to the criteria outlined by the National Institute of Diabetes, Digestive and Kidney Diseases (Hanno, Landis, Matthews-Cook, Kusek, & Nyberg, 1999), over the age of 18 years, and able to read and understand English (excepting Taiwanese participants who had to be able to read and understand Mandarin). The mean age of the entire sample was 49.77 (SD=14.49 years, range: 20 – 83 years). The majority of participants had some college/university education or higher (72.4%), with 19.4% reporting a high school level of education and 8.2% reporting having less than a high school level of education. One participant did not indicate her level of education. With respect to ethnicity, the large majority of participants were Caucasian (86.6%). Asian participants made up 8.0% of the sample and an additional 5.4% of participants reported being black, Latino, Native American, or another unspecified ethnicity. Four participants did not indicate their ethnicity. Approximately half of the sample reported having some level of employment (50.4%; full-time, part-time, student, or homemaker), while the other half reported being unemployed, retired, or on disability (49.6%). Two participants did not indicate their employment status. The majority of participants were married (67.2%). For complete demographic information, including demographics by country, please see Table 1.
Table 1.

*Sample Demographics (N=341)*

<table>
<thead>
<tr>
<th></th>
<th>Total Sample (N=341)</th>
<th>Canada (n=157)</th>
<th>US (n=136)</th>
<th>Taiwan (n=27)</th>
<th>Denmark (n=15)</th>
<th>India (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>49.77 ± 14.49</td>
<td>50.98 ± 14.74</td>
<td>48.81 ± 14.62</td>
<td>46.22 ± 13.55</td>
<td>54.93 ± 10.76</td>
<td>42.67 ± 12.80</td>
</tr>
<tr>
<td>Range</td>
<td>20-83</td>
<td>20-83</td>
<td>21-81</td>
<td>24-69</td>
<td>33-68</td>
<td>22-60</td>
</tr>
<tr>
<td><strong>Education N (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than high school</td>
<td>28 (8.2%)</td>
<td>9 (5.8%)</td>
<td>4 (2.9%)</td>
<td>11 (40.7%)</td>
<td>3 (20.0%)</td>
<td>1 (16.7%)</td>
</tr>
<tr>
<td>High school</td>
<td>66 (19.4%)</td>
<td>40 (25.6%)</td>
<td>16 (11.8%)</td>
<td>8 (29.6%)</td>
<td>2 (13.3%)</td>
<td>0</td>
</tr>
<tr>
<td>Some university/college</td>
<td>91 (26.8%)</td>
<td>45 (28.8%)</td>
<td>41 (30.1%)</td>
<td>1 (3.7%)</td>
<td>2 (13.3%)</td>
<td>2 (33.3%)</td>
</tr>
<tr>
<td>University/college</td>
<td>108 (31.8%)</td>
<td>51 (32.7%)</td>
<td>43 (31.6%)</td>
<td>6 (22.2%)</td>
<td>6 (40.0%)</td>
<td>2 (33.3%)</td>
</tr>
<tr>
<td>Advanced degree</td>
<td>47 (13.8%)</td>
<td>11 (7.1%)</td>
<td>32 (23.5%)</td>
<td>1 (3.7%)</td>
<td>2 (13.3%)</td>
<td>1 (16.7%)</td>
</tr>
<tr>
<td>(graduate/professional)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Ethnicity N (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>292 (86.6%)</td>
<td>155 (98.7%)</td>
<td>122 (89.7%)</td>
<td>0</td>
<td>15 (100%)</td>
<td>0</td>
</tr>
<tr>
<td>Other</td>
<td>45 (13.4%)</td>
<td>2 (1.2%)</td>
<td>14 (10.2%)</td>
<td>24 (100%)</td>
<td>0</td>
<td>5 (100%)</td>
</tr>
<tr>
<td><strong>Employment N (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>171 (50.4%)</td>
<td>70 (44.6%)</td>
<td>73 (54.1%)</td>
<td>14 (53.8%)</td>
<td>8 (53.3%)</td>
<td>6 (100%)</td>
</tr>
<tr>
<td>Not working (unemployed, retired, disabled)</td>
<td>168 (49.6%)</td>
<td>87 (55.4%)</td>
<td>62 (45.9%)</td>
<td>12 (46.2%)</td>
<td>7 (46.7)</td>
<td>0</td>
</tr>
<tr>
<td>Married N (%)</td>
<td>229 (67.2%)</td>
<td>96 (61.1%)</td>
<td>96 (70.6%)</td>
<td>23 (85.2%)</td>
<td>8 (53.3%)</td>
<td>6 (100%)</td>
</tr>
<tr>
<td><strong>Time since diagnosis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>6.43 ± 5.94</td>
<td>7.40 ± 6.62</td>
<td>5.77 ± 4.92</td>
<td>2.58 ± 5.04</td>
<td>8.50 ± 6.01</td>
<td>6.25 ± 3.22</td>
</tr>
</tbody>
</table>
Measures

Demographics.

Demographic questions included age, education, ethnicity, employment status, and marital status.

IC/BPS symptoms and problems.

The Interstitial Cystitis Symptom Index and the Interstitial Cystitis Problem Index (ICSI and ICPI; O'Leary, Sant, Fowler, Whitmore, & Spolarich-Kroll, 1997) are brief, self-report measures of IC/BPS symptoms and their impact on patients’ lives. The ICSI is a four-item measure of the severity of urinary and pain symptoms experienced over the past 30 days. For the three urinary-related items, respondents indicate severity using a six-point scale ranging from 0 (Not at all [None]) to 5 (Almost always [5 or more times]). For the single pain-related item, individuals indicate pain severity using a five-point scale ranging from 0 (Not at all) to 5 (Usually); the authors intentionally excluded 1 as a response option. Thus, the ICSI total score ranges from 0 to 20, with higher scores indicating greater severity of IC/BPS symptoms. The ICPI is a four-item measure that assesses the extent to which frequent urination, nocturia, urgency, and pain are problematic. Respondents indicate how problematic each symptom is using a five-point scale ranging from 0 (No problem) to 4 (Big problem). The ICPI total score has a range from 0 to 16, with higher scores indicating greater severity of IC-BPS-related problems. For the present study, the ICSI and ICPI were used as indicators of IC/BPS symptoms and IC/BPS problems, respectively. Analysis of internal consistency found both the ICSI
(Cronbach’s alpha = .72) and ICPI (Cronbach’s alpha = .78) to have acceptable reliability in the present sample, according to guidelines on internal consistency (Nunally & Bernstein, 1994).

Pain.

The Short Form – McGill Pain Questionnaire (SF-MPQ; Melzack, 1987) is a self-report measure of current pain quality. It consists of 15 single-word descriptors of pain; 11 items represent components of sensory pain quality (e.g., “stabbing”, “throbboning”), and four items are descriptors of affective pain quality (e.g., “fearful”, “punishing-cruel”). For each descriptor, individuals indicate the degree to which the word describes the severity of their present pain using a four-point scale ranging from 0 (None) to 3 (Severe). Thus, the SF-MPQ total score has a range of 0 to 45, with higher scores indicating more severe pain. In addition to a total score, scores for the two subscales of sensory pain and affective pain can be calculated. Previous research on the SF-MPQ has confirmed the proposed two-factor structure (Wright, Asmundson, & McCreary, 2001). In this study, Cronbach’s alpha for all 15 items of the SF-MPQ was .90, indicating good reliability (Nunally & Bernstein, 1994).

Pain catastrophizing.

The Pain Catastrophizing Scale (PCS; Sullivan et al., 1995) is a measure of an individual’s negative appraisal process of a pain experience. The PCS consists of 13 statements describing a specific thought or feeling (e.g., “I worry all the time whether the pain will end”). For each statement, the individual indicates the degree to which they experienced the specified thought or feeling during a past pain experience. Each item is rated on a five-point scale from 0 (Not at all) to 4 (All the time). The PCS yields a total score that can range from 0 to 52, with
higher scores indicating greater pain catastrophizing. Additionally, three subscale scores can be calculated, which assess three interrelated dimensions of catastrophizing: magnification, rumination, and helplessness. Previous factor analysis studies have replicated the proposed three-factor structure (Van Damme, Crombez, Bijttebier, Goubert, & Van Houdenhove, 2002). In this study, Cronbach’s alpha for the PCS was .95, indicating good reliability (Nunally & Bernstein, 1994).

**Coping behaviours.**

The Brief Chronic Pain Coping Inventory (B-CPCI; Jensen et al., 2003) is a shortened version of the full length CPCI, a measure of behavioural coping strategies used over the past week by individuals suffering from chronic pain. Each item describes a specific behavioural coping strategy. The full version of the CPCI consists of 65 items and has eight coping subscales (Jensen et al., 1995). Each subscale assesses a different type of behavioural coping strategy: guarding, resting, asking for assistance, relaxation, task persistence, exercise/stretching, seeking social support, and coping self-statements. The B-CPCI retains the original eight subscale structure. It is comprised of 16 items (i.e., two items for each subscale). As with the full length CPCI, the items are scored on an 8-point scale, ranging from 0 to 7, indicating the number of days the individual used the specified coping strategy during the past week. In addition to a total score, which can range from 0 to 112, three measures of patient adjustment to chronic pain can be calculated: illness-focused coping, wellness-focused coping, and other coping. Previous factor-analytic research has demonstrated an eight-factor model consistent with that proposed by the developers of the CPCI (Davidson et al., 2008; Hadjistavropoulos et al., 1999). In this study,
reliability analysis of all 16 items found the B-CPCI to meet guidelines of acceptable internal consistency, Cronbach’s alpha = .83 (Nunally & Bernstein, 1994).

**Depressive symptoms.**

The Centre for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) is a short self-report measure of current depressive symptomology. It is a 20-item scale in which the individual rates the frequency of each symptom over the past week. Each item is scored on a four-point scale ranging from 0 (Rarely or none of the time [less than one day]) to 3 (Most or all of the time [5-7 days]). Total scores range from 0 to 60, with higher scores indicating greater severity of depressive symptomology. An optimal cut-off score of 27 has been recommended to discriminate between depressed and non-depressed chronic pain patients (Geisser et al., 1997). Previous research has found the CES-D to have a four-factor structure: positive affect, negative/depressed affect, somatic symptoms, and interpersonal problems (Radloff, 1977). In this study, the CES-D met guidelines of acceptable internal consistency, Cronbach’s alpha = .93 (Nunally & Bernstein, 1994).

**Procedure**

The present study examined existing data from two IC/BPS datasets, one from a study conducted in 2009 and the other from a study in 2010. Participants who had completed 80% or more of each of the present study’s measures were included in the present study’s sample. Combined, the data consists of 341 women who had been diagnosed with IC/BPS and were engaged in outpatient treatment at a tertiary care clinical center in Canada, the United States, Taiwan, Denmark, and India. The Canadian sites of recruitment were the IC/BPS clinics at the
Kingston General Hospital (Kingston, Ontario), Hotel Dieu Hospital (Kingston, Ontario), and the Sunnybrook Health Sciences Centre (Toronto, Ontario). American participants were recruited from the IC/BPS clinics at the University of Rochester Medical Center (Rochester, New York), Hofstra University School of Medicine (New Hyde Park, New York), the Loyola University Medical Center (Maywood, Illinois), the University of Washington (Seattle, Washington), the University of Tennessee (Knoxville, Tennessee), and Temple University (Philadelphia, Pennsylvania). Taiwanese participants were recruited from the Taipei Veterans General Hospital. Danish participants were recruited from the IC/BPS clinic at the University of Copenhagen (Herlev, Denmark). Indian participants were recruited from the IC/BPS clinic at Jivraj Mehta Hospital (Ahmedabad, India). All study centers received REB ethics clearances. All participants were approached individually following a urology outpatient clinic appointment by clinic staff. They were provided basic information about the study. Individuals who indicated interest in participating were given a full briefing of the study and were given a questionnaire package to be completed and mailed back to the Psychology Pain Research Lab at Queen’s University. The questionnaire package consisted of a letter of information, consent form, postage-paid return envelope, and the measures described previously, as well as several other measures that were not the focus of this present study.

North American, Danish, and Indian participants completed questionnaires in English. Taiwanese participants completed questionnaires in Mandarin because the urologist believed he had few patients able to read and understand English. Mandarin translated versions of each questionnaire were created using the back-translation procedure (Brislin, 1970). Back-translation is a well-known and widely-used procedure in cross-cultural psychological research (Cha, Kim, & Erlen, 2007). Questionnaires were first translated into Mandarin by bilingual research
assistants. Next, these questionnaires were back-translated (i.e., translated back into English) by an independent, professional translator to check for consistency of meaning and verify the quality of the original translation. The bilingual research assistants checked the questionnaires a final time to ensure the translations were error-free. Back-translation is utilized to improve the reliability and validity of research conducted in different languages.
Chapter 3

Data Analysis

Step 1: Exploratory Factor Analysis (EFA)

Fitting procedure.

An EFA was conducted in order to identify the underlying constructs (factors) of the study’s measures (i.e., the SF-MPQ, PCS, and B-CPCI). The developers of each questionnaire have proposed underlying factors, which are captured in subscale totals. For example, Melzack (1987) has proposed that the SF-MPQ, a measure of current pain, has two underlying factors: sensory pain and affective pain. The PCS is proposed to have three underlying factors: magnification, rumination, and helplessness (Sullivan et al., 1995). As the initial goal of this study is to examine the factor structures of each measure within the specific context of the study’s IC/BPS sample, principal axis factoring was used as the fitting procedure instead of the maximum likelihood procedure. Principal axis factoring is the ideal fitting procedure when the goal of factor analysis is descriptive and focused on the study sample. In contrast, the maximum likelihood procedure is more appropriate when the goal is to generalize the factor structure beyond the study sample.

Factor extraction.

Two extraction methods were employed to determine the number of factors to retain: the scree plot and parallel analysis (Tabachnick & Fidell, 2007). A scree plot is a graph of the eigenvalues (Y-axis) across the number of potential factors (X-axis) (Warner, 2013). An eigenvalue represents the common variance accounted for by a specific factor. For each measure,
the scree plot will be examined for a point of inflection, or a substantial drop, in the eigenvalue curve. The number of factors to the left of the point of inflection indicates the number of factors that should be retained. As the decision of what constitutes a point of inflection or a substantial drop is subjective, it is possible that the scree plot will indicate multiple potential factor structures that should be analyzed. As such, a second extraction method, parallel analysis, will also be used in tandem.

Parallel analysis is an extraction method that generates eigenvalues from random data with an equal sample size and equal number of measured variables. The eigenvalues from the study data and the random data are plotted on the same graph (eigenvalues as the Y-axis, number of factors as the X-axis), resulting in two intersecting lines. The number of factors to the left of the point of intersection indicates the number of factors that should be retained.

For both methods of extraction, if more than one factor is suggested, factor rotation was used to select the best factor structure. Orthogonal rotation and oblique rotation are the two fundamental types of factor rotation. Orthogonal rotations constrain factors so that they are uncorrelated. In contrast, oblique rotation permits factors to be correlated or uncorrelated. As factors are often correlated in psychological research, an oblique rotation (direct oblimin rotation) was used instead of an orthogonal rotation.

**Interpretation.**

After examining all the potential factor structures, the best model was chosen based on the convergence of results from the two extraction methods, interpretability of the retained factors, and the number of items that loaded substantially on at least one of the factors.
Steps 2-4: Mediation Analysis, Moderated Mediation Analysis, Alternate Models

The multiple mediation model proposed in Step 2, the moderated multiple-mediation model proposed in Step 3, and the alternate model described in Step 4 were tested using Hayes’s (2013) PROCESS macro. A total of 76 models can be tested using Hayes’s (2013) PROCESS macro, ranging from the simplest single mediation model to complex moderated multiple-mediation models. Hayes’s (2013) PROCESS macro will be used instead of the traditional causal steps approach towards mediation (Muller, Judd, & Yzerbyt, 2005) or Sobel’s test (Sobel, 1982) following several recent criticisms. Muller and colleagues’ (2005) causal steps approach for moderated mediation incorporates Baron and Kenny’s (1986) steps for establishing mediation. However, Hayes (2013) has argued that the Baron and Kenny (1986) approach should be abandoned for several reasons. Firstly, the Baron and Kenny (1986) approach does not quantify the indirect effect of the potential mediator, which is the ostensible goal of the approach. Furthermore, this approach does not provide a statistical test for the indirect effect (i.e., the mediating effect). Rather, this approach relies on the rejection of three null hypotheses regarding other effects. Consequently, it is the least powerful test of mediation. Hayes (2013) also argues that the Baron and Kenny (1986) approach encourages conclusions of full, partial, or no mediation which makes it difficult to discuss multiple mediation models or moderated mediation models, which is the focus of the present study. Additionally, Sobel’s test (1982), a statistical test of significance for an indirect (mediating) effect, has been criticized due its assumption of a normal sampling distribution of the indirect effect. The distribution is often positively skewed which can result in an underpowered test (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002).
Due to these criticisms, a bootstrapping approach, as used by the PROCESS macro, is preferred because this approach does not make distribution assumptions of the indirect effect (Hayes, 2013). Instead, the bootstrapping approach calculates the statistic of interest (i.e., the indirect or mediating effect) thousands of times over and uses the study data to create an empirically derived representation of the indirect effect’s sampling distribution. From this distribution, a confidence interval (CI) for the indirect effect is constructed. An indirect effect is considered to be significant and a factor is considered to be a significant mediator if its CI does not straddle zero. The PROCESS macro will be used to determine whether the indirect (mediating) effects of the two types of behavioural coping strategies on the catastrophizing-pain relationship are significant (Step 2). For each type of behavioural coping strategy, a bootstrap CI of its indirect effect on the relationship between catastrophizing and pain will be constructed using 10,000 bootstrap samples. If the behavioural coping strategy is determined to be a significant mediator (i.e., the CI does not straddle zero), its indirect effect will be compared between depressed and non-depressed participants (Step 3). If the indirect effect of the behavioural coping strategy is significantly different between depressed and non-depressed participants, the indirect effect is determined to be moderated, or contingent upon, depressive symptomology.
Chapter 4

Results

Data Cleaning

Data were examined with particular attention to missing data and outliers. As there are no current firm guidelines on how to manage missing data (Tabachnick & Fidell, 2007), it was decided that missing data would be imputed for all cases \( (n=36) \) missing less than 20% of items on any particular measure. All other cases \( (n=20) \) were considered missing and removed from further analysis. No outliers were identified using boxplots. Univariate normality was assessed through boxplots and histograms. Examination of boxplots and histograms revealed positive skew for the following measures and subscales: SF-MPQ, SF-MPQ Sensory Subscale, SF-MPQ Affective Subscale, CES-D, PCS, and CPCI Illness-Focused Coping. However, as univariate normality was not required to conduct any of the statistical analyses necessary to meet the study’s aim, the data was not transformed. As a check, post-hoc analyses using transformed data with a square root transformation for all positively skewed measures revealed no major differences in the pattern of findings. As such, all results were interpreted using untransformed data.

Comparison of North American and International Participants

Table 1 includes sample demographics of the participants in the study as a total sample and by country (Canada, USA, Taiwan, India, Denmark). A series of independent \( t \)-tests \( (n=11) \) were conducted to ensure that North American participants did not differ from international participants in terms of age, IC/BPS length, IC/BPS symptoms, IC/BPS problems, pain, catastrophizing, depressive symptomology, illness-focused coping, and wellness-focused coping.
(see Table 2). Following a Bonferroni correction that had a threshold of \( p<.004 \) to indicate significance, comparisons revealed no significant differences between North American and international participants on nine of the 10 measures, except for sensory pain. North American participants (\( M=14.53, SD=8.50 \)) endorsed greater levels of sensory pain than international participants (\( M=9.62, SD=8.65 \)). This mean difference represents an approximately 14\% difference in terms of the range of possible Sensory Pain subscale scores (0-36).

Table 2.

*Differences in Quantitative Characteristics Among North American Participants and International Participants*

<table>
<thead>
<tr>
<th></th>
<th>( t )</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.65</td>
<td>.52</td>
</tr>
<tr>
<td>IC/BPS Length</td>
<td>1.69</td>
<td>.09</td>
</tr>
<tr>
<td>IC/BPS Symptoms</td>
<td>0.64</td>
<td>.52</td>
</tr>
<tr>
<td>IC/BPS Problems</td>
<td>0.69</td>
<td>.49</td>
</tr>
<tr>
<td>Pain (Total)</td>
<td>2.62</td>
<td>.009</td>
</tr>
<tr>
<td>Sensory Pain</td>
<td>3.70</td>
<td>(&lt;.001 )**</td>
</tr>
<tr>
<td>Affective Pain</td>
<td>1.45</td>
<td>.14</td>
</tr>
<tr>
<td>Catastrophizing</td>
<td>0.36</td>
<td>.72</td>
</tr>
<tr>
<td>Depressive Symptomology</td>
<td>1.67</td>
<td>.10</td>
</tr>
<tr>
<td>Illness-Focused Coping</td>
<td>0.63</td>
<td>.53</td>
</tr>
<tr>
<td>Wellness-Focused Coping</td>
<td>1.02</td>
<td>.31</td>
</tr>
</tbody>
</table>

*Note.* **significant following Bonferroni correction** (significance threshold: \( p<.004 \)). Interstitial Cystitis/Bladder Pain Syndrome (ICBPS).

A series of chi-square tests of association (\( n=3 \)) were conducted to ensure that North American participants did not differ from international participants in terms of marital status.
Comparisons revealed no significant differences between North American and international participants in terms of marital status and employment status. However, there was a smaller proportion of North American participants (proportion = .46) than international participants (proportion = .54) who had a lower than high school level of education than would be expected by chance. Additionally, there was a greater proportion of North American participants (proportion = .91) than international participants (proportion = .09) who had some university education or higher than would be expected by chance.

Table 3.

*Differences in Qualitative Characteristics Among North American Participants and International Participants*

<table>
<thead>
<tr>
<th></th>
<th>Pearson $\chi^2$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marital Status</td>
<td>0.65</td>
<td>.28</td>
</tr>
<tr>
<td>Employment Status</td>
<td>1.82</td>
<td>.18</td>
</tr>
<tr>
<td>Level of Education</td>
<td>40.62</td>
<td>&lt;.001 **</td>
</tr>
</tbody>
</table>

*Note.** **significant ($p < .0016$) following Bonferroni correction.

Given that North American and international participants did not differ significantly with respect to the variables of interest, excepting sensory pain, it was decided to treat North American and international participants as one group. Please see Table 4 for the mean scores and standard deviations for each questionnaire. Of interest, the study’s sample had a mean SF-MPQ (total pain) score of 16.50 ($SD = 10.55$) which, in comparison with recently reported SF-MPQ scores in other chronic pain populations, was lower than that of treatment-seeking individuals.
with chronic musculoskeletal pain \((M=24.60; \text{Brown} \& \text{Jones}, 2013)\) and fibromyalgia (approximate \(M=25.00; \text{Liu et al.}, 2012\)), but greater than those of individuals with spinal injury \((M=12.70; \text{Craig et al.}, 2013)\). With regards to IC/BPS symptoms, the study sample’s mean IC/BPS symptom severity score \((M=12.25, SD=4.65)\) and problem score \((M=10.72, SD=4.02)\) were comparable to that of a recent study sampling treatment-seeking women previously diagnosed with IC/BPS \((\text{ICSI} M=11.30 \text{ and ICPI } M=9.90; \text{Konkle et al.}, 2012)\).

Table 4.

### Mean, Standard Deviation, and Questionnaire Score Range of Questionnaires

<table>
<thead>
<tr>
<th>Questionnaire</th>
<th>Mean (Standard Deviation)</th>
<th>Questionnaire Score Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICSI</td>
<td>12.25 (4.65)</td>
<td>0-20</td>
</tr>
<tr>
<td>ICPI</td>
<td>10.72 (4.02)</td>
<td>0-16</td>
</tr>
<tr>
<td>SF-MPQ</td>
<td>16.50 (10.55)</td>
<td>0-45</td>
</tr>
<tr>
<td>PCS</td>
<td>21.59 (12.58)</td>
<td>0-52</td>
</tr>
<tr>
<td>B-CPCI</td>
<td>47.47 (22.03)</td>
<td>0-112</td>
</tr>
<tr>
<td>CES-D</td>
<td>19.19 (13.11)</td>
<td>0-60</td>
</tr>
</tbody>
</table>

*Note.* Interstitial Cystitis Symptom Index (ICSI), Interstitial Cystitis Problem Index (ICPI), Short Form – McGill Pain Questionnaire (SF-MPQ), Pain Catastrophizing Scale (PCS), Brief Chronic Pain Coping Inventory (B-CPCI), Centre for Epidemiologic Studies Depression Scale (CES-D).

**Step 1: Exploratory Factor Analysis on Components of Model**

In order to determine the factor structures of the SF-MPQ, PCS, and CPCI, a principal factor analysis was conducted on each measure, using scree plots (Cattell, 1966) and parallel analysis (Horn, 1965) as factor extraction methods. As the subjectivity of both methods have been noted (see Tabachnick & Fidell, 2007), final model solutions were selected based on results from both methods, in addition to interpretability of the extracted factors. For any solution with two or more factors, an oblique rotation (direct oblimin with Kaiser Normalization) was used.
**SF-MPQ factor analysis.**

According to the scree plot (see Figure 5), two models (1- and 2-factor solutions) warranted further examination. However, examination of the parallel analysis results specified a 3-factor model (see Table 5). Thus, 1-, 2-, and 3-factor solutions were all examined. For the 2- and 3-factor solutions, the interfactor correlations (rs) ranged from |.44| to |.67|; these substantial correlations indicate that an orthogonal rotation would not be justified. For all three solutions, all items had substantial loadings (> |.31|) on at least one factor. However, the 2-factor solution was the most interpretable (see Table 6 for the factor loadings). The first factor, which accounted for 40.97% of the variance, consisted of 12 items and was labeled as *Sensory Pain*. The second factor accounted for 9.44% of the variance, consisted of three items, and was labeled as *Affective Pain*.

![SF-MPQ Scree Plot](image)

*Figure 5.* Scree plot depicting eigenvalues across number of potential factors of the Short Form – McGill Pain Questionnaire (SF-MPQ).
Table 5.

*Comparison of Observed Eigenvalues and Randomly Generated Eigenvalue for the SF-MPQ*

<table>
<thead>
<tr>
<th>Factor</th>
<th>Observed Eigenvalue</th>
<th>Random Eigenvalue</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5.59</td>
<td>0.51</td>
</tr>
<tr>
<td>2</td>
<td>0.91</td>
<td>0.40</td>
</tr>
<tr>
<td>3</td>
<td>0.45</td>
<td>0.32</td>
</tr>
<tr>
<td>4</td>
<td>0.25</td>
<td>0.26</td>
</tr>
<tr>
<td>5</td>
<td>0.13</td>
<td>0.20</td>
</tr>
<tr>
<td>6</td>
<td>0.07</td>
<td>0.15</td>
</tr>
<tr>
<td>7</td>
<td>0.03</td>
<td>0.10</td>
</tr>
<tr>
<td>8</td>
<td>-0.01</td>
<td>0.06</td>
</tr>
<tr>
<td>9</td>
<td>-0.05</td>
<td>0.02</td>
</tr>
<tr>
<td>10</td>
<td>-0.10</td>
<td>-0.03</td>
</tr>
<tr>
<td>11</td>
<td>-0.10</td>
<td>-0.07</td>
</tr>
<tr>
<td>12</td>
<td>-0.13</td>
<td>-0.11</td>
</tr>
<tr>
<td>13</td>
<td>-0.17</td>
<td>-0.15</td>
</tr>
<tr>
<td>14</td>
<td>-0.21</td>
<td>-0.19</td>
</tr>
<tr>
<td>15</td>
<td>-0.22</td>
<td>-0.23</td>
</tr>
</tbody>
</table>

*Note.* Factors with observed eigenvalue greater than randomly generated eigenvalue are in boldface. Short Form – McGill Pain Questionnaire (SF-MPQ).
Table 6.

Factor Loadings for the SF-MPQ

<table>
<thead>
<tr>
<th>Item</th>
<th>Factor 1</th>
<th>Factor 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Throbbing (1)</td>
<td>.58</td>
<td>.12</td>
</tr>
<tr>
<td>Shooting (2)</td>
<td>.78</td>
<td>-.16</td>
</tr>
<tr>
<td>Stabbing (3)</td>
<td>.85</td>
<td>-.13</td>
</tr>
<tr>
<td>Sharp (4)</td>
<td>.84</td>
<td>-.12</td>
</tr>
<tr>
<td>Cramping (5)</td>
<td>.54</td>
<td>.011</td>
</tr>
<tr>
<td>Gnawing (6)</td>
<td>.45</td>
<td>.18</td>
</tr>
<tr>
<td>Hot-Burning (7)</td>
<td>.34</td>
<td>.23</td>
</tr>
<tr>
<td>Aching (8)</td>
<td>.52</td>
<td>.15</td>
</tr>
<tr>
<td>Heavy (9)</td>
<td>.34</td>
<td>.33</td>
</tr>
<tr>
<td>Tender (10)</td>
<td>.36</td>
<td>.24</td>
</tr>
<tr>
<td>Splitting (11)</td>
<td>.48</td>
<td>.22</td>
</tr>
<tr>
<td>Tiring-Exhausting (12)</td>
<td>.43</td>
<td>.34</td>
</tr>
<tr>
<td>Sickening (13)</td>
<td>.16</td>
<td>.63</td>
</tr>
<tr>
<td>Fearful (14)</td>
<td>-.079</td>
<td>.76</td>
</tr>
<tr>
<td>Punishing-Cruel (15)</td>
<td>.048</td>
<td>.72</td>
</tr>
</tbody>
</table>

Note. Factor loadings greater than .30 are in boldface. Short Form – McGill Pain Questionnaire (SF-MPQ).

PCS factor analysis.

Based on the scree plot, only one model (1-factor solution) warranted further examination (see Figure 6). However, examination of the parallel analysis results specified a 2-factor model (see Table 7). Thus, both the 1-factor solution and the 2-factor solution were examined. For the 2-factor solution, the interfactor correlation ($r$) was $.18$. All items had substantial loadings ($> |.54|$) on the first factor for both the 1- and 2-factor solutions. However, in the 2-factor solution, no item loaded higher onto the second factor relative to the first factor. Furthermore, the 1-factor solution was more interpretable (see Table 8 for the factor loadings). This single factor accounted for 58.38% of the variance. It consisted of all 13 items and was labeled Pain Catastrophizing.
Figure 6. Scree plot depicting eigenvalues across number of potential factors of the Pain Catastrophizing Scale (PCS).
Table 7.

Comparison of Observed Eigenvalues and Randomly Generated Eigenvalue for the PCS

<table>
<thead>
<tr>
<th>Factor</th>
<th>Observed Eigenvalue</th>
<th>Random Eigenvalue</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7.61</td>
<td>0.47</td>
</tr>
<tr>
<td>2</td>
<td>0.41</td>
<td>0.35</td>
</tr>
<tr>
<td>3</td>
<td>0.20</td>
<td>0.28</td>
</tr>
<tr>
<td>4</td>
<td>0.17</td>
<td>0.21</td>
</tr>
<tr>
<td>5</td>
<td>0.03</td>
<td>0.16</td>
</tr>
<tr>
<td>6</td>
<td>0.01</td>
<td>0.11</td>
</tr>
<tr>
<td>7</td>
<td>-0.01</td>
<td>0.06</td>
</tr>
<tr>
<td>8</td>
<td>-0.05</td>
<td>0.01</td>
</tr>
<tr>
<td>9</td>
<td>-0.07</td>
<td>-0.03</td>
</tr>
<tr>
<td>10</td>
<td>-0.07</td>
<td>-0.08</td>
</tr>
<tr>
<td>11</td>
<td>-0.09</td>
<td>-0.12</td>
</tr>
<tr>
<td>12</td>
<td>-0.11</td>
<td>-0.16</td>
</tr>
<tr>
<td>13</td>
<td>-0.15</td>
<td>-0.21</td>
</tr>
</tbody>
</table>

*Note.* Factors with observed eigenvalue greater than randomly generated eigenvalue are in boldface. Pain Catastrophizing Scale (PCS).
Table 8.

*Factor Loadings for the PCS*

<table>
<thead>
<tr>
<th>Item</th>
<th>Factor 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>I worry all the time about whether the pain will end (1)</td>
<td>.78</td>
</tr>
<tr>
<td>I feel I can't go on (2)</td>
<td>.74</td>
</tr>
<tr>
<td>It's terrible and I think it's never going to get any better (3)</td>
<td>.81</td>
</tr>
<tr>
<td>It's awful and I feel that it overwhelms me (4)</td>
<td>.86</td>
</tr>
<tr>
<td>I feel I can't stand it anymore (5)</td>
<td>.88</td>
</tr>
<tr>
<td>I become afraid the pain will get worse (6)</td>
<td>.78</td>
</tr>
<tr>
<td>I keep thinking of other painful events (7)</td>
<td>.54</td>
</tr>
<tr>
<td>I anxiously want the pain to go away (8)</td>
<td>.73</td>
</tr>
<tr>
<td>I can't seem to keep it out of my mind (9)</td>
<td>.82</td>
</tr>
<tr>
<td>I keep thinking about how much it hurts (10)</td>
<td>.84</td>
</tr>
<tr>
<td>I keep thinking about how badly I want the pain to stop (11)</td>
<td>.83</td>
</tr>
<tr>
<td>There's nothing I can do to reduce the intensity of the pain (12)</td>
<td>.64</td>
</tr>
<tr>
<td>I wonder whether something serious may happen (13)</td>
<td>.60</td>
</tr>
</tbody>
</table>

*Note.* Factor loadings greater than .30 are in boldface. Pain Catastrophizing Scale (PCS).

**B-CPCI factor analysis.**

According to the scree plot, three models (1-, 2-, and 4-factor solutions) warranted further examination (see Figure 7). Examination of the parallel analysis results specified a 5-factor solution (see Table 9). However, more than 25 iterations were required to extract five factors from the data and the extraction process was terminated for the 5-factor model. Thus, 1-, 2-, and 4-factor solutions were all examined. For the 2- and 4-factor solutions, the interfactor correlations ($r$) ranged from $|0.07|$ to $|0.42|$. Both the 1-factor and 4-factor solutions had a number
of items that did not load highly onto any of the factors (factor loadings < |.27|). In contrast, in the 2-factor solution all items had substantial loadings (> |.37|) on at least one of the two factors. Additionally, the 2-factor solution was the most interpretable (see Table 10 for the factor loadings). The first factor, which accounted for 30.21% of the variance, consisted of 8 items and was labeled as *Illness-Focused Behavioural Coping*. Factor 2 accounted for 13.92% of the variance, also consisted of 8 items, and was labeled as *Wellness-Focused Behavioural Coping*.

*Figure 7. Scree plot depicting eigenvalues across number of potential factors of the Brief Chronic Pain Coping Inventory (B-CPCI).*
Table 9.

*Comparison of Observed Eigenvalues and Randomly Generated Eigenvalue for the B-CPCI*

<table>
<thead>
<tr>
<th>Factor</th>
<th>Observed Eigenvalue</th>
<th>Random Eigenvalue</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4.27</td>
<td>0.54</td>
</tr>
<tr>
<td>2</td>
<td>1.62</td>
<td>0.43</td>
</tr>
<tr>
<td>3</td>
<td>0.77</td>
<td>0.34</td>
</tr>
<tr>
<td>4</td>
<td>0.60</td>
<td>0.28</td>
</tr>
<tr>
<td>5</td>
<td>0.30</td>
<td>0.23</td>
</tr>
<tr>
<td>6</td>
<td>0.15</td>
<td>0.18</td>
</tr>
<tr>
<td>7</td>
<td>0.05</td>
<td>0.13</td>
</tr>
<tr>
<td>8</td>
<td>-0.01</td>
<td>0.08</td>
</tr>
<tr>
<td>9</td>
<td>-0.03</td>
<td>0.04</td>
</tr>
<tr>
<td>10</td>
<td>-0.08</td>
<td>0.00</td>
</tr>
<tr>
<td>11</td>
<td>-0.11</td>
<td>-0.04</td>
</tr>
<tr>
<td>12</td>
<td>-0.12</td>
<td>-0.08</td>
</tr>
<tr>
<td>13</td>
<td>-0.19</td>
<td>-0.12</td>
</tr>
<tr>
<td>14</td>
<td>-0.21</td>
<td>-0.16</td>
</tr>
<tr>
<td>15</td>
<td>-0.24</td>
<td>-0.20</td>
</tr>
<tr>
<td>16</td>
<td>-0.26</td>
<td>-0.24</td>
</tr>
</tbody>
</table>

*Note.* Factors with observed eigenvalue greater than randomly generated eigenvalue are in boldface. Brief Chronic Pain Coping Inventory (B-CPCI).
Table 10.

**Factor Loadings for the B-CPCI**

<table>
<thead>
<tr>
<th>Item</th>
<th>Factor 1</th>
<th>Factor 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Limited my standing time (1)</td>
<td>.66</td>
<td>.024</td>
</tr>
<tr>
<td>Lay down on a bed (2)</td>
<td>.69</td>
<td>-.071</td>
</tr>
<tr>
<td>Asked for help with a chore or task (3)</td>
<td>.76</td>
<td>-.035</td>
</tr>
<tr>
<td>I got support from a friend (7)</td>
<td>.37</td>
<td>.32</td>
</tr>
<tr>
<td>Avoided activity (9)</td>
<td>.67</td>
<td>-.23</td>
</tr>
<tr>
<td>Rested in a chair or recliner (10)</td>
<td>.57</td>
<td>.045</td>
</tr>
<tr>
<td>Asked for help in carrying, lifting, or pushing something (11)</td>
<td>.74</td>
<td>.028</td>
</tr>
<tr>
<td>Talked to a friend or family member for support (15)</td>
<td>.41</td>
<td>.21</td>
</tr>
<tr>
<td>Focused on relaxing my muscles (4)</td>
<td>.42</td>
<td>.43</td>
</tr>
<tr>
<td>I didn't let the pain interfere with my activities (5)</td>
<td>-.27</td>
<td>.58</td>
</tr>
<tr>
<td>Exercised to improve my overall physical condition for at least 5 minutes (6)</td>
<td>.029</td>
<td>.48</td>
</tr>
<tr>
<td>Told myself my pain will get better (8)</td>
<td>.19</td>
<td>.49</td>
</tr>
<tr>
<td>Used deep, slow breathing to relax (12)</td>
<td>.32</td>
<td>.42</td>
</tr>
<tr>
<td>Did not let the pain affect what I was doing (13)</td>
<td>-.23</td>
<td>.49</td>
</tr>
<tr>
<td>Stretched the muscles where I hurt and held that stretch for at least 10 seconds (14)</td>
<td>.23</td>
<td>.43</td>
</tr>
<tr>
<td>Reminded myself that there are people who are worse off than I am (16)</td>
<td>.26</td>
<td>.39</td>
</tr>
</tbody>
</table>

*Note.* Factor loadings greater than .30 are in boldface. Brief Chronic Pain Coping Inventory (B-CPCI).

**Summary.**

The SF-MPQ was composed of two factors (*Sensory Pain* and *Affective Pain*), the PCS had a one-factor structure (*Pain Catastrophizing*), and the B-CPCI was composed of two factors (*Illness-Focused Coping* and *Wellness-Focused Coping*). These factors were subsequently used in the study’s hypothesized models in Steps 2-4.
Steps 2-4: Independent Examination of Sensory Pain and Affective Pain

Based on the derived factors from the EFA, particularly the finding that the SF-MPQ had a two-factor structure, it was decided that the construct of pain would be examined as two factors: Sensory Pain and Affective Pain. Consequently, two separate models were conceptualized: one in which Sensory Pain was the predicted outcome and one in which Affective Pain was the predicted outcome. Results from Steps 2-4 for the Sensory Pain model will be presented, followed by results from Steps 2-4 for the Affective Pain model.

Sensory pain.

Step 2: Mediation analysis (process analysis).

Model 4 of the PROCESS macro was used to test if illness-focused and wellness-focused coping strategies individually mediated the relationship between catastrophizing and sensory pain (see Figure 8). There was a significant direct effect of catastrophizing on sensory pain, $b=0.20$, $SE=0.033$, $p<.001$. Illness-focused coping was a significant mediator. A bias-corrected bootstrap CI for the indirect effect of illness-focused coping ($b=0.049$), based on 10,000 bootstrap samples, was entirely above zero (95% CI: 0.025 to 0.082). However, the indirect effect of wellness-focused coping ($b=-0.006$) was not significant. The CI for this indirect effect, based on 10,000 bootstrap samples, straddled zero (95% CI: -0.024 to 0.0046). Based on these results, a single mediation model was chosen over a multiple mediation model as the best foundation for the moderated mediation model in Step 3. Specifically, wellness-focused coping was removed as a mediator.
Figure 8. The path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between catastrophizing and sensory pain.

**Step 3: Moderated mediation analysis (CPA).**

The moderator variable of depressive symptomology was treated as a dichotomous variable using the CES-D cut-off score of 27 suggested by Geisser and colleagues for individuals suffering from chronic pain (Geisser et al., 1997). Cases with total CES-D scores of 27 or higher were coded as depressed and cases with scores lower than 27 were coded as not depressed. It was predicted that depressive symptomology would moderate the mediating effect of illness-focused coping on the relationship between catastrophizing and sensory pain. Specifically, depressive symptomology was predicted to moderate the relationship between illness-focused coping and sensory pain (see Figure 9).

Model 14 of Hayes’s (2013) PROCESS macro was used to investigate this proposed moderated mediation model. There was a significant direct effect of catastrophizing on sensory pain, $b=0.15$, $SE=0.039$, $p<.001$. A bias-corrected bootstrap CI testing the equality of the indirect effect of illness-focused coping between depressed and non-depressed participants, based on
10,000 bootstrap samples, straddled zero (95% CI: -0.059 to 0.016; index of moderated mediation= -0.015). This result indicates that the previously established indirect effect of illness-focused coping did not differ significantly between depressed and non-depressed individuals.

Figure 9. The path model depicts illness-focused coping as a mediator (Me) of the relationship between catastrophizing and sensory pain. Depressive symptomology is predicted to moderate (Mo) the relationship between illness-focused coping and sensory pain.

**Step 4a: Mediation analysis (process analysis) of alternate model.**

Step 4 was broken down into Steps 4a and 4b in order to mirror the procedures of Step 2 (Mediation Analysis) and Step 3 (Moderated Mediation Analysis). Model 4 of the PROCESS macro was used to test if illness-focused and wellness-focused coping strategies individually mediated the relationship between sensory pain and catastrophizing (see Figure 10). There was a significant direct effect of sensory pain on catastrophizing, $b=0.49$, $SE=0.081$, $p<.001$. Both illness-focused coping and wellness-focused coping were significant mediators of this relationship. A bias-corrected bootstrap CI for the indirect effect of illness-focused coping ($b=0.10$), based on 10,000 bootstrap samples, was entirely above zero (95% CI: 0.023 to 0.18). In contrast, the bias-corrected bootstrap CI for the indirect effect of wellness-focused coping ($b=-0.075$), based on 10,000 bootstrap samples, was entirely below zero (95% CI: -0.14 to -0.031).
Based on these results, a multiple mediation model was chosen as the best foundation for the moderated mediation model in Step 4b.

![Diagram](image)

**Figure 10.** The alternate path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between sensory pain and catastrophizing.

**Step 4b: Moderated mediation analysis (CPA) of alternate model.**

As with Step 3, the moderator variable of depressive symptomology was treated as a dichotomous variable using the CES-D cut-off score of 27 suggested by Geisser and colleagues for individuals suffering from chronic pain (Geisser et al., 1997). Cases with total CES-D scores of 27 or higher were coded as depressed and cases with scores lower than 27 were coded as not depressed. It was predicted that depressive symptomology would moderate the mediating effects of illness-focused coping and wellness-focused coping on the relationship between sensory pain and catastrophizing (see Figure 11).
Figure 11. The alternate path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between sensory pain and catastrophizing. Depressive symptomology is predicted to moderate (Mo) the relationship between illness-focused coping and catastrophizing, and the relationship between wellness-focused coping and catastrophizing.

Model 14 of Hayes’s (2013) PROCESS macro was used to investigate this proposed moderated mediation model. There was a significant direct effect of sensory pain on catastrophizing, $b=0.30, SE=0.073, p<.001$. A bias-corrected bootstrap CI testing the equality of the indirect effect of illness-focused coping between depressed and non-depressed participants, based on 10,000 bootstrap samples, straddled zero (95% CI: -0.21 to 0.054; index of moderated mediation=-0.077). Similarly, the bias-corrected bootstrap CI testing the equality of the indirect effect of wellness-focused coping between depressed and non-depressed participants, based on 10,000 bootstrap samples, also straddled zero (95% CI: -0.10 to 0.085; index of moderated mediation=-0.077). This result indicates that the previously established indirect effects of illness-focused coping and wellness-focused coping did not differ significantly between depressed and non-depressed individuals.
**Affective pain.**

**Step 2: Mediation analysis (process analysis).**

Model 4 of the PROCESS macro was used to test if illness-focused and wellness-focused coping strategies individually mediated the relationship between catastrophizing and affective pain (see Figure 12). There was a significant direct effect of catastrophizing on affective pain, \( b=0.10, SE=0.010, p<.001 \). As with the catastrophizing-sensory pain model, illness-focused coping was a significant mediator. A bias-corrected bootstrap CI for the indirect effect of illness-focused coping (\( b=0.013 \)), based on 10,000 bootstrap samples, was entirely above zero (95% CI: 0.0069 to 0.024). However, the indirect effect of wellness-focused coping (\( b=0.003 \)) was not significant; the CI for this indirect effect, based on 10,000 bootstrap samples, straddled zero (95% CI: -0.006 to 0.0035). Based on these results, a single mediation model was chosen over a multiple mediation model as the best foundation for the moderated mediation model in Step 3. Specifically, wellness-focused coping was removed as a mediator.

![Figure 12](image.png)

*Figure 12.* The path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between catastrophizing and affective pain.
**Step 3: Moderated mediation analysis (process analysis).**

As with Step 3 for the Sensory Pain model, depressive symptomology as the moderating variable was treated as a dichotomous variable. Cases with total CES-D scores of 27 or higher were coded as depressed and cases with scores lower than 27 were coded as not depressed. It was predicted that depressive symptomology would moderate the mediating effect of illness-focused coping on the relationship between catastrophizing and affective pain. Specifically, depressive symptomology was predicted to moderate the relationship between illness-focused coping and affective pain (see Figure 13).

![Path Model Diagram](image1)

*Figure 13.* The path model depicts illness-focused coping as a mediator (Me) of the relationship between catastrophizing and affective pain. Depressive symptomology is predicted to moderate (Mo) the relationship between illness-focused coping and affective pain.

Model 14 of Hayes’s (2013) PROCESS macro was used to investigate this proposed moderated mediation model. There was a significant direct effect of catastrophizing on affective pain, $b=0.09$, $SE=0.012$, $p<.001$. A bias-corrected bootstrap CI testing the equality of the indirect effect of illness-focused coping between depressed and non-depressed participants, based on 10,000 bootstrap samples, did not straddle zero (95% CI: 0.0001 to 0.026; index of moderated mediation=0.011). This result indicates that the established indirect effect of illness-focused
coping was significantly stronger for depressed participants ($b=0.021, SE=0.007$) in comparison
to non-depressed participants ($b=0.010, SE=0.0036$).

While the CI level testing this difference between depressed and non-depressed
participants was entirely above zero, the lower limit of the CI (LLCI) was very close to zero
(LLCI: 0.0001). This may be the result of the dichotomization of depressive symptomology
using the conservative CES-D cut-off score of 27 as recommended by Geisser and colleagues
(1997). Thus, analyses were re-run to investigate if the index of moderated mediation would be
stronger and the CI further away from zero if depression was dichotomized using the less
conservative CES-D cut-off score of 19 suggested by Turk and Okifuji (1994) for chronic pain
patients. The value of index of moderated mediation was identical (index of moderated
mediation=0.011) but the bias-corrected bootstrap CI, based on 10,000 bootstrap samples, was
further from zero (95% CI: 0.002 to 0.023). As with the model using the more conservative CES-
D cut-off score of 27, the indirect effect of illness-focused coping was significantly stronger for
depressed participants ($b=0.019, SE=0.006$) in comparison to non-depressed participants
($b=0.008, SE=0.0034$).

**Step 4a: Mediation analysis (process analysis) of alternate model.**

Model 4 of the PROCESS macro was used to test if illness-focused and wellness-focused
coping strategies individually mediated the relationship between affective pain and
catastrophizing (see Figure 14). There was a significant direct effect of affective pain on
catastrophizing, $b=2.23, SE=0.22, p<.001$. However, neither illness-focused coping nor wellness-
focused coping were significant mediators of the relationship between affective pain and
catastrophizing. A bias-corrected bootstrap CI for the indirect effect of illness-focused coping
(b=0.14), based on 10,000 bootstrap samples, straddled zero (95% CI: -0.030 to 0.33).

Additionally, the bias-corrected bootstrap CI for the indirect effect of wellness-focused coping (b=-0.028), based on 10,000 bootstrap samples, also straddled zero (95% CI: -0.12 to 0.015).

Given that neither behavioural coping strategy were significant mediators of the affective pain-catastrophizing relationship, a CPA was not conducted (Step 4b for this model was skipped).

---

**Figure 14.** The alternate path model depicts illness-focused coping and wellness-focused coping as mediators (Me) of the relationship between affective pain and catastrophizing.
Chapter 5

Discussion

The aim of the present study was to examine the relationships between catastrophizing, behavioural coping strategies, depressive symptomology, and pain in an IC/BPS patient population. Two key findings emerged from the study’s mediation and moderated mediation analyses. First, illness-focused behavioural coping strategies were found to drive the clinically observed relationship between catastrophizing and pain (both sensory and affective) in women suffering from IC/BPS. Second, this mediating effect of illness-focused behavioural coping strategies was stronger among individuals with higher levels of depressive symptomology. Overall, these findings support a model in which psychosocial factors play important roles in the experience of pain in IC/BPS.

Mediating Role of Illness-Focused Behavioural Coping Strategies

Individuals who had a greater tendency to catastrophize about their pain were more likely to have engaged in illness-focused behavioural coping strategies, such as resting and asking for assistance. In turn, these illness-focused behavioural coping strategies predicted higher levels of pain. These results can be interpreted using Lazarus and Folkman’s (1984) transactional model of stress and the fear of re-injury model (Lethem et al., 1983). The transactional model of stress predicts that individuals who view a stressful event as a threat and who believe that they will be unable to cope effectively are more likely to feel helpless and less likely to employ active coping strategies to deal with the stressful event (Lazarus & Folkman, 1984). With respect to chronic pain, individuals who catastrophize about their pain tend to magnify the negative implications of pain and feel helpless in the face of painful experiences.
Thus, women with IC/BPS who had a greater tendency to catastrophize may have felt more helpless and were less likely to have used active, wellness-focused behavioural strategies to cope with their pain. Instead, these women were more likely to have employed more passive, illness-focused behavioural coping strategies. According to the fear of re-injury model, continued use of passive, illness-focused behavioural coping strategies, such as resting and guarding one’s body from injury, leads to greater levels of pain through avoidance, increased pain perception, and deconditioning (Lethem et al., 1983).

These findings are consistent with past studies that found significant positive associations between catastrophizing and pain (Benyon et al., 2013; Nickel et al., 2010; Sullivan & Neish, 1999; Thompson et al., 2010) and positive associations between illness-focused behavioural coping strategies and greater pain interference (Chan et al., 2012; Finan et al., 2012; Tan et al., 2011), among a variety of pain conditions, including IC/BPS. Furthermore, the study’s finding that wellness-focused behavioural coping strategies did not significantly mediate the relationship between catastrophizing and pain is consistent with past research that did not find wellness-focused behavioural coping strategies to be associated with improved treatment or to predict changes in pain intensity (Jensen et al., 1994; Weijenborg et al., 2009).

The present study’s findings are not only consistent with the existing chronic pain literature, but they extend our understanding of the interactions between several key psychosocial variables and pain. This study has demonstrated that the long-observed relationship between catastrophizing and pain is mediated, at least partly, by use of illness-focused coping behaviours. It is partly through the use of these illness-focused coping behaviours, such as guarding painful areas of the body from contact and asking for assistance with tasks, that negative, exaggerated appraisals about pain (i.e., catastrophizing thoughts) come to impact an
individual’s subjective pain experience. In other words, the present study demonstrates a link between thoughts (i.e., catastrophizing), behaviours (i.e., behavioural coping strategies), and pain.

**Moderating Role of Depression in Affective Pain**

Results from the present study also highlight the importance of examining the psychological context in which catastrophizing and illness-focused behavioural coping strategies interact. Depressive symptomology was found to moderate the mediating effect of illness-focused behavioural coping strategies on the relationship between catastrophizing and affective pain. This is not to say that use of illness-focused behavioural coping strategies does not impact individuals with lower levels of depressive symptomology. Rather, the mechanistic effect of these illness-focused coping strategies in driving the relationship between catastrophizing and affective pain is stronger among individuals who have endorsed greater levels of depressive symptomology. Thus, for individuals with IC/BPS and clinically meaningful levels of depressive symptomology, the use of illness-focused behavioural coping strategies, such as pain-contingent rest and asking for assistance, has a stronger effect in driving the relationship between catastrophizing and affective pain.

The long-recognized comorbidity between depression and chronic pain has been extensively reviewed (for review see Campbell, Clauw, & Keefe, 2003). In particular, the reciprocal nature of the relationship between depression and pain has been the focus of several studies. Depressive symptoms have been found to predict future pain in a variety of pain conditions, including low back pain (Leino & Magni, 1993) and chronic widespread pain (Mundal, Grawe, Bjorngaard, Linaker, & Fors, 2014). Animal models have found induced pain
to lead to depression-like behaviour and that depression-like behaviours can exacerbate pain (for a recent review see Li, 2015). While these animal studies have focused on the possible neurobiological mechanisms that belie the reciprocal relationship between depression and chronic pain, the present study’s findings indicate that depressive symptomology may indirectly impact pain by exacerbating the indirect effect of illness-focused behavioural coping strategies on affective pain.

It is important to note that depressive symptomology moderated the indirect effect of illness-focused behavioural coping for affective pain only and not for sensory pain. Affective pain is conceptually distinct from depression (Lerman, Rudich, & Shahar, 2010). The affective component of pain pertains to the feelings of fear, tension, and unpleasantness related to a pain experience (Turk & Okifuji, 2010). Sensory pain and affective pain can vary independently. Experimental studies have demonstrated that emotional factors influence the unpleasantness of a pain experience (an aspect of affective pain), without influencing sensory components of pain, in experimentally-induced pain. Villemure and colleagues (2003) found that ratings of pain unpleasantness, but not pain intensity (an aspect of sensory pain), in response to painful heat stimuli were influenced by odour-induced mood. Thus, it is possible that among individuals experiencing both chronic pain and higher levels of depressive symptomology, the affective component of their pain is more easily modulated; that is, affective pain is more vulnerable to the effects of catastrophizing and behavioural coping strategies. However, further research investigating the differential relationships between depression and the two components of pain among chronic pain populations is warranted.
Alternate Models

Examination of alternate models revealed that both sensory and affective pain had a direct effect on catastrophizing. Thus, it is not clear from this present study whether catastrophizing predicts pain or pain predicts catastrophizing. It is also possible that the relationship between catastrophizing and pain is recursive: greater levels of catastrophizing increase levels of pain and, in turn, higher levels of pain increase catastrophizing thoughts. However, past research investigating the causal relationship between catastrophizing have found that changes in catastrophizing predicts changes in levels of pain, and not vice versa (Campbell, Quartana, Buenaver, Haythornthwaite, & Edwards, 2010). Results from Campbell and colleagues’ (2010) study would suggest that changes in catastrophizing precede and contribute to changes in pain at a later time point. However, this experimental study sampled from a healthy, pain-free population and, as such, these results may not be entirely generalizable to a chronic pain population, such as IC/BPS. Study designs that investigate this issue of causality within a chronic pain population are discussed in greater detail in the Future Research section.

Both illness-focused and wellness-focused behavioural coping strategies mediated the relationship between sensory pain and catastrophizing. However, depressive symptomology did not moderate these mediating effects. Neither illness-focused behavioural coping nor wellness-focused behavioural coping were significant mediators of the relationship between affective pain and catastrophizing. Thus, the original model in which catastrophizing predicts pain (both sensory and affective) and this predictive relationship is mediated by behavioural coping strategies appears to fit the data better than the alternative models and, equally importantly, is better justified theoretically.
Clinical Implications

As present biomedical treatment outcomes for IC/BPS remain poor or inconclusive (Peters, 2012), findings from this study have the potential to greatly improve programs for the management of pain related to IC/BPS. The study’s results highlight the indirect role of illness-focused behavioural coping strategies which drive the relationship between catastrophizing and pain. Given these findings, pain management programs would be advised to incorporate cognitive-behavioural therapy (CBT) techniques into their treatment modules in order to target catastrophizing and reduce use of illness-focused behavioural coping strategies. Psychological interventions for chronic pain, including CBT, has been found to be effective in reducing pain among individuals suffering from a variety of chronic pain conditions (for recent reviews see Jensen & Turk, 2014; Kerns, Sellinger, & Goodin, 2011). With regards to chronic visceral pain, an eight week CBT-based symptom management program for CP/CPPS found significant decreases in catastrophizing thoughts and pain (Tripp, Nickel, & Katz, 2011). To date, there have been no published studies regarding the effectiveness of CBT programs for IC/BPS pain and symptom management.

CBT techniques for managing chronic pain can be categorized as cognitive, behavioural, or supportive educational (Skinner, Wilson, & Turk, 2012). Cognitive techniques include cognitive restructuring to identify and challenge maladaptive pain-related cognitions (including catastrophizing thoughts), reduce fear of activity, and replace maladaptive pain-related cognitions with more adaptive, coping thoughts. Past research investigating the processes of change among chronic pain patients undergoing CBT have found that changes in catastrophizing mediate changes in treatment outcome (Turner, Holtzman, & Mancl, 2007). Behavioural techniques to decrease muscle tension and reduce emotional distress from pain include relaxation
training, pacing (i.e., changing activity structure), and behavioural activation. Importantly, behavioural activation includes increasing physical activity and reducing activity avoidance, and would discourage use of illness-focused behavioural coping strategies. Indeed, an active coping style was found to predict more positive treatment outcomes among patients with chronic musculoskeletal pain who underwent a multidisciplinary treatment program (Boonstra, Reneman, Waaksma, Schiphorst Preuper, & Stewart, 2014). Finally, educational supportive techniques include psychoeducation. Psychoeducation about pain through a biopsychosocial perspective would allow individuals with IC/BPS to understand the impact of psychosocial factors, such as catastrophizing and illness-focused behavioural coping strategies, on their pain experience (Jensen & Turk, 2014; Kerns et al., 2011).

Finally, routine screening measures of catastrophizing, behavioural coping strategies, and depressive symptomology would be helpful in identifying subsets of the IC/BPS patient population for whom CBT interventions will be most effective. In particular, a preliminary screening of depressive symptomology would be highly recommended given the finding that the effects of illness-focused behavioural coping strategies on pain are stronger among individuals with higher levels of depressive symptoms. Chronic pain management would be improved through early identification of individuals who would benefit most from CBT interventions that target depressive symptoms, catastrophizing, and illness-focused behavioural coping strategies. Indeed, given the prevalence rates of chronic pain and its enormous economic burden, the need to “match” individuals with the most appropriate treatment programs has been long-recognized (Heapy, Stroud, Higgins, & Sellinger, 2006).
Limitations

The present study had a cross-sectional design which excludes the possibility of concluding any causal relationships between catastrophizing, illness-focused behavioural coping strategies, and pain (Tabachnick & Fidell, 2007). Therefore, interpretation of the moderated mediation model is strictly limited to predictive relationships: higher levels of catastrophizing predict more use of illness-focused behavioural coping strategies which in turn predict higher levels of pain. It cannot be concluded that higher levels of catastrophizing cause increased use of illness-focused behavioural coping strategies and that these coping strategies result in more pain, as would be hypothesized based on Lazarus and Folkman’s (1984) transactional model of stress and the fear of re-injury model (Lethem et al., 1983). However, while the present study design is imperfect, cross-sectional study designs allow researchers to investigate relationships between factors in a cost-effective manner. Future research designs that circumvent this limitation will be discussed.

Additionally, it is possible that there was self-selection bias among the study’s participants. The study’s participants (i.e., individuals who sought treatment, agreed to participate in this study, and completed the questionnaire package) may be different from the general IC/BPS population. For example, these treatment-seeking women may have had more severe IC/BPS symptoms than the general IC/BPS population. As other similarly designed studies (i.e., patients recruited from urology clinics) may face this same limitation, it would be more useful to compare results with a population-based study sampling women with IC/BPS symptoms who were not seeking treatment. One such study by Berry and colleagues (2011) reported a mean severity of IC/BPS symptoms \( (M=11.50) \) comparable to that of this sample \( (M=12.25, SD=4.65) \). Interestingly, the mean rating of IC/BPS related problems among the
present study’s sample ($M=10.72$, $SD=4.02$) was lower than that reported by Berry and colleagues (2011). Konkle and colleagues (2012) noted a similar pattern when comparing symptom severity and functional impact between treatment-seeking individuals with IC/BPS and population based (i.e., non-treatment seeking) individuals who meet criteria for IC/BPS. Thus, it appears that participants in this study experience similar levels of IC/BPS symptom severity as individuals with IC/BPS symptoms who may or may not be actively seeking treatment for their symptoms. Therefore, while self-selection bias remains a possible limitation, it does not appear that participants in the sample were more motivated to participate due to greater levels of IC/BPS symptomology or problems related to IC/BPS symptoms.

Finally, despite recruiting international participants, the overwhelming majority of participants were educated Caucasian women with access to tertiary care services and who had been diagnosed by an urologist. It is important to note that the study’s North American participants reported higher levels of education and higher levels of sensory pain than international participants. However, no other significant differences were found between these two demographic groups with respect to age, IC/BPS length, IC/BPS symptoms, IC/BPS problems, pain, catastrophizing, depressive symptomology, illness-focused coping, wellness-focused coping, marital status, and employment status. Furthermore, post-hoc analyses found no differences in the pattern of results when international participants were removed from the study sample. While caution is warranted in making statements regarding generalizability due to the small sample size of international participants and the nature of the urology clinics involved, the present study has demonstrated initial support for international application of the aforementioned clinical implications.
Future Research

Future research investigating the process through which catastrophizing comes to predict pain among individuals with IC/BPS should be designed such that causality can be discussed. However, given that individuals with IC/BPS suffer from chronic pain and report lower indices of well-being, it would be unethical to experimentally induce catastrophizing in order to assess if increased catastrophizing causes increased chronic pain. Furthermore, results from experimental studies designed to only increase catastrophizing over short periods of time will not truly capture the chronic pain experience of individuals with IC/BPS.

Given the need to ethically consider causation, a longitudinal study design is recommended in order to establish temporal precedence (Warner, 2013). With a longitudinal study, predictor, process, and outcome variables can be assessed at several time points. This would allow researchers to test a mediation model predicting that catastrophizing measured at Time 1 predict levels of pain obtained at Time 3 and that this relationship is mediated by levels of behavioural coping strategies used at Time 2. While causality cannot be established, inferences about causality are more theoretically justified based on this longitudinal design in comparison to the present study’s cross-sectional design (Warner, 2013).

Similarly, structural equation modeling (SEM) can be used instead of a moderated mediation model to investigate the relationship between catastrophizing, behavioural coping strategies, and pain. SEM is a statistical analysis that allows researchers to model both non-directional (i.e., correlational) and directional (i.e., causal) relationships between factors (Schumacker & Lomax, 2010). While results from multiple moderated mediation models can be compared against each other informally, there is currently no formal testing available to determine which model best fits the data. In contrast, researchers can employ SEM to identify
several specific models to analyze and compare which model fits the data best. While SEM cannot infer causality, combining a longitudinal study design with SEM analysis will bolster a study’s conclusions about the probability of a causal relationship between catastrophizing and pain.

Finally, the present study highlighted the need to consider contextual factors when analyzing factors that contribute to pain. The mediating effect of illness-focused behavioural coping strategies was stronger among individuals with higher levels of depressive symptomology. However, no other contextual variables were considered in this study, including personality. Neuroticism, in particular, has been linked with catastrophizing and pain-related variables (Asghari & Nicholas, 2006). Additionally recent meta-analysis by Dixon-Gordon and colleagues (2015) found particularly high prevalence rate of personality disorders among individuals suffering from a variety of pain conditions in comparison to the general population. Thus, future research investigating the role of other contextual factors, such as neuroticism and other maladaptive personality traits, in the pain experience of individuals with IC/BPS is warranted.

**Final Conclusions**

The present study contributes to the growing body of research on the impact of psychosocial factors and IC/BPS by expanding the focus from associations between psychosocial factors and pain-related outcomes to modeling the relationship between catastrophizing, behavioural coping strategies, depressive symptomology, and pain. Illness-focused behavioural coping strategies were found to mediate the relationship between catastrophizing and pain. Higher levels of catastrophizing predicted higher levels of illness-focused coping strategies.
which, in turn, predicted higher levels of pain. Additionally, depressive symptomology was found to moderate this indirect effect of illness-focused behavioural coping strategies, specifically for the relationship between catastrophizing and affective pain. Clinical implications of this study were discussed, particularly advocating for the use of CBT techniques in pain management programs for IC/BPS in order to target catastrophizing and illness-focused behavioural coping. Future research with a longitudinal design and use of SEM to model relationships between psychosocial factors were recommended based on the present study’s limitations.
References


16340588


Health Problems. *Psychosomatic Medicine*, 68(1), 121-128. doi: 10.1097/01.psy.0000197673.29650.8e 16449422


