CLARIFYING THE NATURE OF PAIN-RELATED ANXIETY: IMPLICATIONS FOR ASSESSMENT AND TREATMENT OF CHRONIC MUSCULOSKELETAL PAIN

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ABSTRACT

Pain-related anxiety and anxiety sensitivity (AS) are important constructs in fear-anxiety-avoidance models of chronic pain (Asmundson, P. J. Norton, & Vlaeyen, 2004). Pain-related anxiety (McCracken & Gross, 1998) includes dimensions of cognitive anxiety (e.g., concentration difficulties as result of pain), behavioural avoidance, fearful thinking about pain, and physiological reactivity to pain (e.g., autonomic arousal, nausea). AS (Reiss, Peterson, Gursky, & McNally, 1986) is the trait tendency to fear the physiological sensations of anxiety due to the belief such sensations signal imminent harm. Evidence suggests an association between AS and pain-related anxiety (e.g., Muris, Schmidt, Merckelbach, & Schouten, 2001; P. J. Norton & Asmundson, 2003); however, the nature of this relationship remains unclear. An overlapping but empirically distinct relationship has been suggested (Carleton, Abrams, Asmundson, Antony, & McCabe, 2009) but there is also evidence pain-related anxiety may be a manifestation of AS (Greenberg & Burns, 2003). The current study sought to assess the posited view that pain-related anxiety may be an expression of AS. An experimental design was used in an attempt to extend the findings of Greenberg and Burns (2003) with a non-clinical analogue sample. Participants were healthy adults (N = 61, 62% women, M age = 31, SD = 11.45) who completed measures of pain-related anxiety, AS, social anxiety, fear of negative evaluation, and general negative affectivity (i.e., depression, trait anxiety). They underwent a pain induction task intended to elicit pain-related anxiety and a mental arithmetic task intended to elicit social-evaluative anxiety. Data gathered at baseline, during, and post-experimental tasks included (a) cardiovascular variables to provide indices of anxious arousal; (b) self-report measures of pain-related anxiety, social-

evaluative anxiety, and general negative affectivity; and (c) behavioural performance measures (i.e., correct answers on the mental arithmetic task, pain tolerance). Two hypotheses were tested: 1. Consistent with the view that pain-related anxiety may be a manifestation of AS, it was hypothesized that a measure of pain-related anxiety (i.e., Pain Anxiety Symptoms Scale-20[PASS-20]; McCracken & Dhingra, 2002) would significantly and substantively predict scores on post-task dependent measures for *both* the pain-related anxiety and social-evaluative anxiety induction tasks in regression models while controlling for effects of general negative affectivity; 2. It was hypothesized that the predictive effects of pain-related anxiety (PASS-20) on dependent measure scores would be accounted for by scores on a measure of AS (Anxiety Sensitivity Index-3 [ASI-3]; Taylor et al., 2007) in regression models. Neither of these hypotheses was supported. For the first hypothesis, results revealed that PASS-20 scores predicted positive variance in only the pain induction post-task measure of current pain-anxiety. Contrary to prediction, the PASS-20 did not account for variance in any of the mental arithmetic task dependent measures. For the second hypothesis, the results similarly failed to reject the null hypothesis. Despite exhibiting a high degree of correlation with the PASS-20, ASI-3 scores failed to account for positive variance in either the pain induction or mental arithmetic post-task dependent measures. Results indicated that AS was not associated with pain-related anxiety in a sample of participants not reporting current pain. These findings may lend support to the view that the apparently robust relationship observed between AS and pain-related anxiety among persons with chronic pain, may, in part, be a consequence of a persistent pain experience.

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1. INTRODUCTION AND LITERATURE REVIEW

1.1 Introduction

In recent decades our primarily biological understanding of pain has broadened to include psychological and social dimensions of the pain experience (e.g., Melzack & Wall, 1965; Melzack & Wall, 1996; Melzack & Casey, 1968; Turk, Meichenbaum, & Genest, 1983). An important result of this wider conceptualization has been the advancement and elaboration of what are known as biopsychosocial models of pain (e.g., Asmundson, P. J. Norton, & Vlaeyen, 2004; P. J. Norton & Asmundson, 2003; Vlaeyen & Linton, 2000; Turk et al., 1983; Turk & Monarch, 2002). As the name implies, these models integrate the interacting influences of biological, psychological, and social perspectives to provide a more comprehensive understanding of the pain experience. Among the contributions of biopsychosocial pain models is the description of several negative affect-related constructs posited as being influential to the development and maintenance of chronic musculoskeletal pain. Important among these constructs are *pain-related anxiety* (McCracken & Dhingra, 2002; McCracken, Zayfert, & Gross, 1992), *fear of pain* (Asmundson, Vlaeyen, & Crombez, 2004), *anxiety sensitivity* (AS; Asmundson & G. R. Norton, 1995; Asmundson & Taylor, 1996), and *pain catastrophizing* (Sullivan, Stanish, Waite, Sullivan, & Tripp, 1998). While these constructs have consistently been associated with chronic musculoskeletal pain, much remains to be learned regarding the interrelationships among these constructs and their relative contributions to the development and maintenance of chronic musculoskeletal pain.

One research area in need of further clarification is the nature of the relationship between pain-related anxiety and AS. Some researchers suggest pain-related anxiety may be a construct specific to the pain experience (e.g., McCracken, Zayfert, & Gross, 1992); however, there is empirical evidence that suggests pain-related anxiety may be better understood as a manifestation of AS (Asmundson & G. R. Norton, 1995; Greenberg & Burns, 2003). What seems apparent is that these constructs overlap considerably and further research aimed at disentangling what is shared and what is distinct is warranted. Clarification of the relationship between these constructs carries important implications for both assessment and treatment of chronic musculoskeletal pain. If pain-related anxiety is better understood as analogous to a pain-focused phobia, then treatment should include exposure to the feared pain-related objects. Feared objects include continued or worsening pain, movement, re-injury, as well as more abstract fears including alterations to identity, failure to fulfill social roles, and being a burden to others (Morely & Eccleston, 2004). Alternatively, if pain-related anxiety is more appropriately viewed as a manifestation of AS, then interventions targeting the general fear of somatic sensations (e.g., interoceptive exposure) should be included in treatment protocols.

This dissertation is structured as follows. First, to provide relevant background, the theoretical and empirical literature concerning pain and its historical conceptualizations will be reviewed. Thereafter, the literature concerning chronic musculoskeletal pain and its relationship to anxiety-related symptomatology will be discussed. Following this review of the relevant background literature, the constructs of pain-related anxiety and AS will be described and discussed in the context of

experimental and clinical pain research. Subsequent to reviewing the relevant literature, the purpose and hypotheses, method, results, and discussion will follow.

1.2. Pain

Pain is a ubiquitous human experience. The International Association for the Study of Pain (IASP) defines pain as: "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage." (IASP Subcommittee on Taxonomy, 1994, p. S212). Notably, this definition stresses the aversive emotional nature of pain rather than referring to a direct relationship between pain and identifiable injury or pathology. Defining pain in this manner acknowledges that reported pain severity (i.e., little or no pain to excruciating pain) does not necessarily exhibit a linear relationship with the degree of actual, potential, or described tissue damage.

In everyday experience, pain is believed to be fundamentally adaptive and protective (Millan, 1999). Indeed, acute pain has been posited as serving three purposes. First, pain experienced prior to injury (e.g., painful encounters with hot objects) has obvious survival value in that it normally results in immediate withdrawal from the painful stimulus, thereby preventing further injury. Second, when pain prevents further injury it facilitates the learning necessary to avoid potentially injurious objects or situations in the future. Finally, pain associated with injury or illness imposes limitations on activity that enable the body's natural healing processes which lead to recovery and survival (Melzack & Wall, 1982). In contrast to experiences of acute pain, pain is termed chronic when it persists beyond the time period typically necessary to facilitate healing.

Commonly used definitions of chronic pain are described and discussed in further detail below.

1.2.1. Chronic pain.

Chronic pain has been defined in several similar although somewhat nuanced ways. The IASP definition of chronic pain takes into account pain duration and appropriateness to associated injury or illness. The organization outlines three categories of pain that include *less than one month*, *between one and six months*, and *more than six months* with chronic pain defined as pain that persists beyond the normal time required for tissue to heal (typically three months). With respect to the appropriateness of pain, the IASP recognizes that acute pain normally functions in an adaptive manner (i.e., protects against re-injury and facilitates healing), whereas chronic pain has no apparent biological value (IASP Subcommittee on Taxonomy, 1994). The American College of Rheumatology (ACR) applies a differing set of criteria to define chronic pain occurring in the context of fibromyalgia. The ACR (Wolfe et al., 1990) defines *chronic widespread pain* when the following are present for at least three months: (a) pain in the left side of the body, (b) pain in the right side of the body, (c) pain above the waist, (d) and pain below the waist. In addition, axial skeletal pain (i.e., cervical spine or anterior chest or thoracic spine or low back) must also be present to meet the definition. The American Society of Anesthesiologists defines chronic pain as "pain of any etiology not directly related to neoplastic involvement, associated with a chronic medical condition or extending in duration beyond the expected temporal boundary of tissue injury and normal healing, and adversely affecting the function or well-being of the individual." (American Society of Anesthesiologists, 2010, p.810).

Government health departments have also provided definitions of chronic pain. Health and Welfare Canada defines chronic pain as pain that persists beyond the normal time of healing, is associated with protracted illness (or is a severe symptom of a recurring condition), and persists for three months or longer (Health Services and Promotion Branch, Health and Welfare Canada, 1990). In the United Kingdom the Clinical Standards Advisory Group of the National Health System has defined chronic pain as pain persisting beyond the expected time frame for healing or that occurs in disease processes in which healing may never occur (Clinical Standards Advisory Group, 2000). While the abovementioned definitions have common elements, there are differences in the criteria that, in turn, contribute to variability in the prevalence rates reported in epidemiological studies of chronic pain. Below, representative literature concerning the prevalence and impact of chronic pain is reviewed.

The reported prevalence of chronic pain varies substantially, with general population prevalence estimates ranging from 8% to more than 60% depending on the chronic pain definition used, the methodology employed, and the nature of the samples evaluated (H. C. Philips, 2006). In a review of the chronic pain epidemiological literature, Opsina and Harstall (2002) grouped published studies by both sample characteristics and chronic pain definitions employed by researchers. Researchers who employed IASP criteria reported general population chronic pain prevalence ranging from 10.5% to 55.2% (weighted mean = 35.5%). In studies that used the ACR criteria, a narrower range of between 10.1% and 13% (weighted mean = 11.8%) was reported. Among studies of chronic pain epidemiology in specific populations, the reported prevalence (using IASP criteria) for children (ages 0-18) was 19.5% for males and 30.4%

for females. For studies examining elderly populations, prevalence rates (using IASP criteria) ranged from 32.9% (23.7% for men and 40.1% for women) to 50.2% (with no sex break-down).

More recently, a large (n = 46,394) computer-assisted telephone study of chronic pain prevalence was conducted in fifteen European countries and Israel (Breivik, Collett, Ventafridda, Cohen, & Gallacher, 2006). Using criteria consisting of a six month pain duration and severity greater than or equal to 5 on a numeric pain rating scale (i.e., NRS; 0 = no pain, 10 = the worst pain imaginable), 19% of adults reported pain in the past month as well as pain multiple times during the past week. Interviews of a subset of respondents (i.e., n = 4839, ~ 600 in each country) showed that 66% had moderate pain (NRS = 5-7), 34% had severe pain (NRS = 8-10), 46% had constant pain, 54% had intermittent pain, and 59% reported pain lasting for between 2 and 15 years. Regarding chronic pain impact, 21% had been diagnosed with depression, 61% were less able or unable to work outside of the home, 19% had lost their job, and 13% had changed their occupation due to their pain.

Chronic pain prevalence and impact has also been examined in Canada in an investigation not included in the Opsina and Harstall (2002) review. In this study, Moulin and colleagues (Moulin, Clark, Speechley, & Morley-Forster, 2002) employed a stratified sample (n = 2012) weighted for sex, age, and region according to 1996 census data to study chronic pain prevalence and impact. Using a six month (continuous or intermittent) pain duration criterion, 29% of respondents reported experiencing chronic non-cancer pain (27% of men and 31% of women) and 80% of those with chronic pain reported experiencing severe pain (i.e., NRS = 8-10). Prevalence was higher (39%) in

persons over age 55 and the average number of years in pain was 10.7. With respect to chronic pain impact, 49% reported significant difficulty attending social and family events, 61% reported being unable to participate in their usual recreational activities, and 58% reported being unable to carry out their usual daily activities at home.

Taken together, these epidemiological findings indicate chronic pain is a major public health problem that negatively impacts the functioning and well being of persons affected (C. J. Phillips et al., 2008; C. J. Phillips, 2006). Moreover, chronic pain imposes staggering costs to the economy with estimates ranging into the hundreds of billions of dollars per year in disability expenditures, heath care costs, and lost productivity. For example, in the United States lost productivity due to pain-related reduced performance and absenteeism is estimated to cost employers US $61 billion annually (W. F. Stewart, Ricci, Chee, Morganstein, & Lipton, 2003). In Canada, the direct healthcare costs of chronic pain are estimated at more than 6 billion dollars per year with lost productivity (i.e., job loss, sick time) costing an additional 37 billion dollars annually (C. J. Phillips & Schopflocher, 2008). To summarize, the above-reviewed literature indicates chronic pain is common in developed countries and imposes significant human and economic costs. Efforts to effectively treat pain have prompted the development of several theoretical models to better understand acute and chronic pain. Below, prominent pain models are discussed.

1.3. Theoretical Models of Pain

Theoretical understandings of pain have been developed and elaborated over several centuries of clinical observation and empirical investigation. Pain models can be organized into three broad categories that include biomedical models, psychodynamic

models, and biopsychosocial models. Below, the central tenets of these various theoretical positions are outlined. It is beyond the scope of this dissertation to comprehensively detail these models so, rather, an overview is provided in order to frame the rationale for and purposes of the current investigation.

1.3.1. Biomedical models of pain.

One of the earliest biomedical models of pain is traced to Descartes who, in the sixteenth century, proposed what is now referred to as *specificity theory*. Specificity theory views pain as a primarily sensory neurological experience in which the level of pain experienced corresponds (or is expected to correspond) to the degree of tissue damage (e.g., the prick of a needle should result in less pain than a deep flesh wound). According to this model, pain is believed to begin with the action of a physical stimulus (e.g., heat, injury) on specialized receptors which then transmit signals to pain centres in the brain (Melzack & Wall, 1982). The core assumption underlying biomedical models is that pain is resultant to external factors that impinge on an otherwise normally functioning system.

Biomedical models have been criticized for a number of reasons. First, there is little evidence to support a systematic relationship between the degree of physical harm (i.e., severity of injury or disease-related tissue damage) and the level of pain and disability (Linton & Buer, 1995; Rose, Klenerman, Atchison, & Slade, 1992). Indeed, the majority of individuals with low back pain exhibit no presently detectable tissue damage. Moreover, many symptom-free individuals evidence considerable structural abnormalities that, according to a strictly biomedical model of pain, would be expected to be associated with significant pain and disability (Jensen, Turner, & Romano, 1994).

Second, biomedical pain models have been found distinctly wanting with respect to conceptualizing chronic pain; specifically, that pain can persist after tissues have healed is in direct contradiction with the core assumption that pain should correspond with the degree of tissue damage. A final criticism of biomedical models is that they fail to consider the importance of social (e.g., illness behaviour) and psychological factors (e.g., anxiety, depression) to the pain experience.

1.3.2. Psychodynamic models of pain.

Early models of pain that focused on psychological factors were based on psychodynamic theory (e.g., Merskey & Spear, 1967). Several such models have been advanced, including Freud's theory centering on emotional pain (Breuer & Freud, 1893-1895 [1974]) and Engel's conceptualization of *psychogenic pain* and the *pain-prone* patient (Engel, 1959). Freud proposed the concept of *hysterical pain*, posited to arise out of the repression of emotional conflict (e.g., inappropriate sexual urges) from which pain (and other physical symptoms) were said to develop via *conversion* – a process by which psychic energies are converted into physical symptoms (reviewed in Hodgkiss, 2000). Engel (1959) argued that pain is a primarily psychological phenomenon and that there are pain-prone individuals for whom pain is an expression of *psychic regulation*. Psychodynamic models of pain have been largely unsupported by empirical research and have consequently been discarded in favour of more comprehensive and empirically-supported models. Nonetheless, psychodynamic models can be credited with directing research attention towards psychological aspects of the pain experience (Asmundson & Wright, 2004). Unfortunately, the psychodynamic perspective has also led to the

persistent pejorative belief that persons complaining of pain in the absence of apparent injury or pathology are not experiencing real pain.

1.3.3. Gate control theory and the body-self neuromatrix.

Developed by Melzack and Wall in 1965, gate control theory was the first widely accepted theory of pain that integrated physiological and psychological mechanisms. Expanding on the basic nociceptive processes of biomedical models, gate control theory posited that mechanisms in the central nervous system (CNS) modulated (i.e., via inhibitory or excitatory processes) nociceptive information reaching the brain (Melzack & Katz, 2004; Melzack & Wall, 1965; Melzack & Wall, 1996). Melzack and Wall (1965) proposed the existence of a *gating mechanism* in the dorsal horn of the spinal cord that could either inhibit or facilitate nociceptive transmission. This gate was posited to either open (i.e., via excitatory processes) or close (i.e., via inhibitory processes) ascending nociceptive pathways in response to descending neuronal communication from the brain. Importantly, the descending neuronal communication that affects the operation of the gate comprises both psychological processes (e.g., attentional processes, cognitions, emotions) and competing small- and large-fibre nervous communication from the peripheral nervous system (PNS; e.g., sensation). Thus, if descending inputs facilitate the opening of the gating mechanism, an ascending nociceptive message is then transmitted to the brain and results in the experience of pain. According to gate control theory, the intensity of a pain experience is related to the magnitude of the ascending nociceptive communication from the gating mechanism – the point in the pain circuitry where modulation by descending neuronal communication is posited to occur.

Gate control theory represented a significant advance over biomedical models of pain. The theory provided an integrated framework with which to understand the complex interactions among physiological processes (e.g., tissue damage, stress hormones) and psychological factors (e.g., attentional processes, cognitions, emotions) in relation to the pain experience (Melzack & Katz, 2004). While the posited mechanism of gate modulation was able to account for the inconsistent relationship between tissue damage and pain intensity, there remained other pain phenomena that the theory was not able to explain. In particular, the mysterious experience of phantom limb pain was problematic for gate control theory. Specifically, the clinical observation that some individuals with spinal cord damage (e.g., paraplegics) reported pain in the absence of nociceptive pathways suggested the existence of other pain generating mechanisms.

In order to account for phantom pain, researchers proposed a new theory termed the *body-self neuromatrix* (Melzack, 1999; Melzack & Katz, 2004). The neuromatrix is posited to comprise a complex network of interconnected brain regions (e.g., thalamus, limbic system, cerebral cortex, somatosensory projections), all of which are known to play a role in the pain experience. The theory proposes that inputs (e.g., ascending PNS neuronal communication, cognitions, emotions, stress hormones) to the neuromatrix enable a continuously updated representation of the body that reflects the current environment and situation (e.g., sense data, proprioceptive information, tissue damage). Concurrently, outputs from the body-self neuromatrix provide a conscious experience of the body-self – including pain – and prompt reactions to inputs and experiences (e.g., approach/avoidance behaviour in response to stimuli). In this manner the body-self neuromatrix generates a representation of the body, including the conscious experience of

sensations, movement, and pain (Melzack & Katz, 2004). Regarding origins, the neuromatrix is theorized to be genetically determined but modifiable by experience. It is posited to generate a representation of the whole body from birth onwards, irrespective of PNS or spinal cord inputs. Accordingly, the body-self neuromatrix provides a framework that can account for how paraplegics, amputees, and even those born without limbs are able to experience sensations, movement, and pain in body regions that do not have (or possibly never had) direct neuronal communication with the CNS.

1.3.4. Biopsychosocial models of pain.

The term *biopsychosocial* aptly describes pain models that explicitly consider the interacting influences of biological, psychological, and social aspects of the pain experience. Biopsychosocial models include the operant model, Glasgow model, biobehavioural models, fear-avoidance models, and diathesis-stress models (for recent reviews see Asmundson & Wright, 2004; Gatchel, Peng, Peters, Fuchs, & Turk, 2007). These models were developed to improve the conceptualization, assessment, and treatment of individuals experiencing chronic musculoskeletal pain. Biopsychosocial models accept the tenets of recent biomedical approaches (e.g., gate control theory) and expand on them through elaboration and empirical investigation of psychosocial constructs posited important to the pain experience (e.g., pain-related anxiety; McCracken, 1997, and AS; Asmundson & G. R. Norton, 1995; Asmundson & Taylor, 1996). A comprehensive review of the biopsychosocial models of pain is beyond the scope of this review; however, the *fear-avoidance* models of chronic pain (Asmundson, P. J. Norton, & G. R. Norton, 1999; Asmundson et al., 2004; Lethem, Slade, Troup, &

Bentley, 1983; P. J. Norton & Asmundson, 2003; H. C. Philips, 1987; Vlaeyen & Linton, 2000) are critical to the current investigation and warrant elaboration.

1.3.5. Fear avoidance models of chronic pain.

In general, fear-avoidance models posit that individuals who experience injury and corresponding pain will interpret their pain as either threatening or non-threatening (P. J. Norton & Asmundson, 2003; Vlaeyen & Linton, 2000). Those who interpret their pain as non-threatening are believed to engage in appropriate activity restriction (e.g., keeping weight off a sprained ankle for a few weeks) necessary to facilitate healing after which they gradually re-engage in their usual activity levels and return to an approximation of pre-injury functioning. In contrast, individuals who interpret pain and injury as threatening are believed more likely to experience catastrophic thoughts concerning the pain or injury itself (e.g., *I'm going to die*) or about the consequences of the pain or injury (e.g., *How will I ever work again?*). These negative and fearful cognitions may lead to increased sensitivity and reactivity to pain which is expressed behaviourally as escape and avoidance behaviours in reaction to or anticipation of pain. In turn, avoidance-based activity restriction results in muscular deconditioning, contributes to depressive symptoms, and (paradoxically) ultimately results in increased pain and risk of further injury. To summarize, interpreting pain and injury as threatening is thought to fuel a maladaptive cycle of pain avoidance, increasing disability, and further pain (Asmundson, P. J. Norton et al., 1999; Asmundson et al., 2004; Sullivan et al., 1998; Vlaeyen & Linton, 2000). A comprehensive review of these models and the empirical evidence supporting them has been recently published by Leeuw et al. (2007).

Fear of pain and pain-related anxiety have been central to fear avoidance models since first formulated by Vlaeyen and Linton (2000). AS was later introduced into the model as a predisposing risk factor for pain catastrophizing (P. J. Norton & Asmundson, 2003), and the most recent reformulation of the model included a distinction between fear of pain and pain-related anxiety (Asmundson et al., 2004; see Figures 1 and 2). Below, the literature concerning associations between anxiety symptomatology and chronic musculoskeletal pain is reviewed.

Figure 1. Amended Vlaeyen-Linton fear-avoidance model of chronic pain

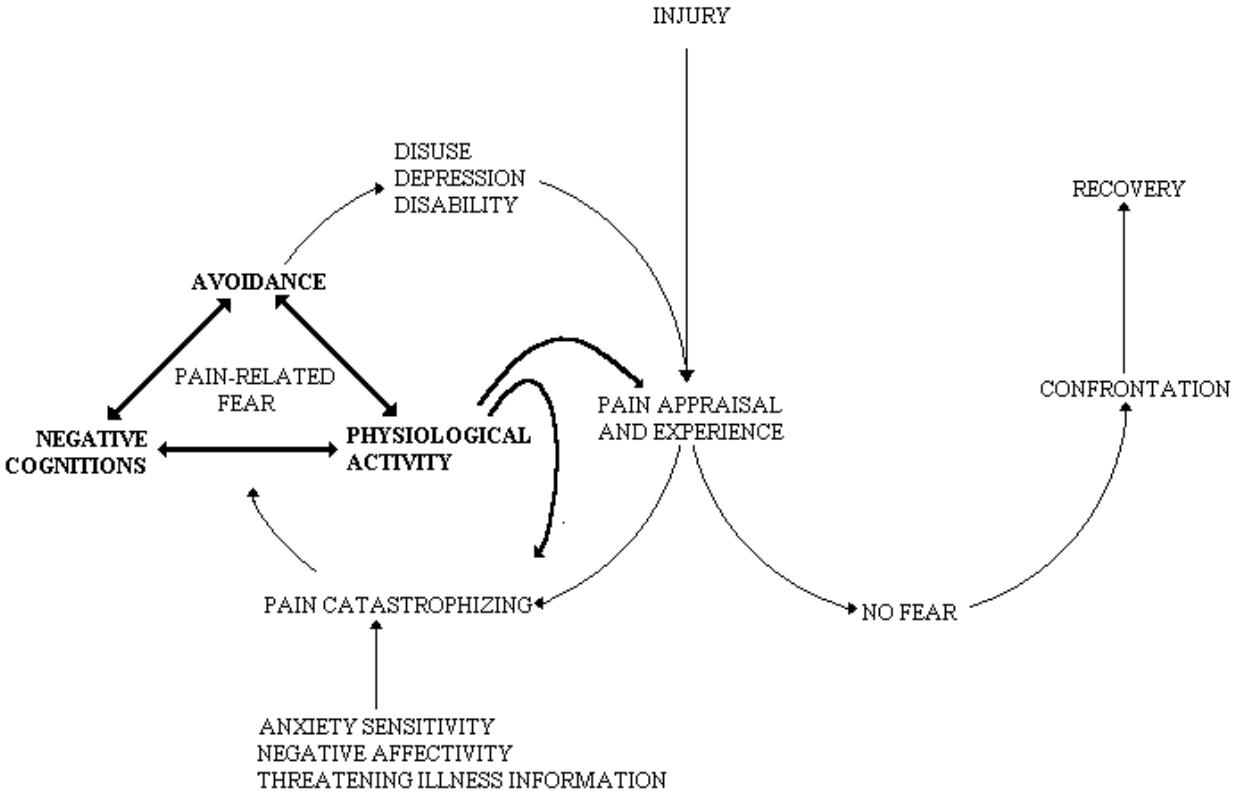

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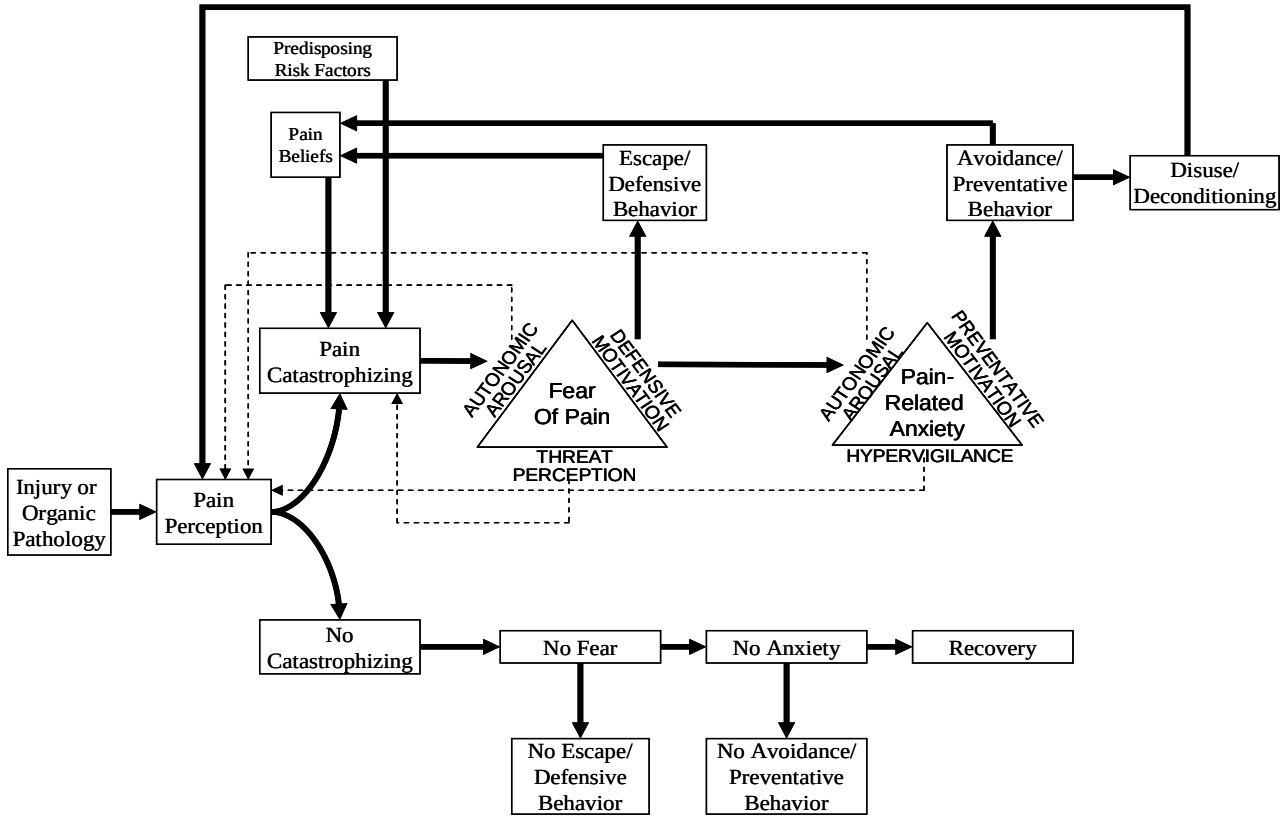
Figure 2. Fear-anxiety-avoidance model of chronic pain



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1.4 Anxiety and chronic musculoskeletal pain

Anxiety has long been observed to be associated with chronic musculoskeletal pain. Indeed, individuals diagnosed with various musculoskeletal medical conditions (e.g., arthritis, low back pain, fibromyalgia) are frequently found to have co-occurring anxiety disorders. For example, in large nationally representative epidemiological studies, the 12 month prevalence of any anxiety disorder in persons with arthritis-related chronic pain has been reported to range between 26.5% and 35.1% (McWilliams, Cox, & Enns, 2003; McWilliams, Goodwin, & Cox, 2004; Von Korff et al., 2005), a prevalence rate that is considerably higher than the 18.1% reported for the general population (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Consistent with these epidemiological findings, an investigation of the pooled results of studies of persons with back or neck pain in 17 countries found that, relative to those not reporting back or neck pain, they were almost twice as likely to have had past year panic disorder with agoraphobia or social anxiety disorder, as well as being nearly three times as likely to have generalized anxiety disorder (GAD) or posttraumatic stress disorder (PTSD) (Demyttenaere et al., 2007).

Collectively, these findings are representative of the documented relationships regarding anxiety symptomatology in persons with chronic musculoskeletal pain. Researchers have proposed a number of plausible explanations for this apparently consistent association. Perhaps the most straightforward interpretation views pain-related anxiety primarily as but one component of the negative affectivity (i.e., anxiety, depression, anger) commonly experienced by persons with chronic pain (e.g., Gatchel et al., 2007). Anxiety and worry are ubiquitous among persons with chronic pain,

particularly when symptoms remain unexplained (e.g., fibromyalgia, idiopathic back pain). In addition to the uncertainty regarding the origin and meaning of symptoms, pain-related anxiety also centres on concerns about the future. Common concerns focus on fears that pain will worsen, that physical capacities will be diminished, that disability is inevitable, and that employability will be imperiled. Individuals with chronic pain may also be anxious about how others perceive them, worrying, for example, that people do not believe they are suffering, or that will be told they will simply have to learn to live with their pain.

Of particular importance to fear-avoidance models, pain-related anxiety also centres on activities (e.g., bending, lifting) believed to increase pain or worsen associated medical conditions. Such anxieties are thought to underlie avoidance behaviours that, in turn, lead to inactivity, disuse and, greater disability (Boersma & Linton, 2006). Persons with pain-related anxiety are also prone to develop attentional biases toward somatic sensations, scanning their bodies for aversive symptoms that may foretell pain or signal a worsening of their condition. Even mildly aversive sensations may come to be appraised as intolerable, thereby contributing to maintained physiological arousal, increased muscle tension, and ultimately, increased or continuing pain (Gatchel, 2005; Robinson & Riley, 1999).

An alternative view of the relationship between anxiety and chronic pain derives from Mowrer's two-factor theory of fear conditioning (Mowrer, 1947) and conceptualizes pain syndromes as resultant to fear and related avoidance. Two-factor theory posits that fears are initially learned through classical conditioning and are thereafter maintained via avoidance of cues associated with the learned fear and anxiety. In the context of chronic

musculoskeletal pain, two-factor theory proposes that persons with injury or pathology who initially experience fear or anxiety during activity that provokes pain learn to respond with anxiety at the prospect of such activities. Anxious appraisal of actions believed likely to result in pain leads to avoidance of such activities. In turn, these avoidance behaviours and catastrophic appraisals are maintained through negative reinforcement (i.e., activity avoidance precludes exposure to painful experiences and confirms the apparent utility of fearful appraisals). These patterns of fearful appraisals coupled with avoidance behaviour result in physical deconditioning, further avoidance, and ultimately increased pain (Asmundson, P. J. Norton et al., 1999; Fordyce, 1976; Fordyce, Shelton, & Dundore, 1982; Lethem et al., 1983; H. C. Philips, 1987; Vlaeyen & Linton, 2000).

Clearly, avoidance of pain and activity is not always maladaptive. In early stages of recovery from painful injury, appropriate activity restriction facilitates healing and eventual return to a pre-injury functioning. For some individuals however, avoidance behaviour is thought to be maintained and generalized not as an attempt to escape pain but, instead, as functioning to reduce anxious arousal in anticipation of pain. Avoidance of this nature may be viewed by the individual as a way to control or reduce pain but may also lead to the over-prediction of pain severity (Rachman, 1994). Importantly, avoidance of activities anticipated to result in pain also limits exposure to experiences that may disconfirm the belief pain should be feared (H. C. Philips, 1987). This perspective views anxiety in persons with chronic pain not as an associated component of general negative affectivity but, rather, as driving a cycle of fear and avoidance that underlies and maintains pain chronicity. Such a view places anxiety associated with pain

syndromes as analogous to a specific pain-focused phobia variously operationalized as pain-related anxiety (McCracken & Dhingra, 2002; McCracken et al., 1992), fear avoidance (Waddell, Newton, Henderson, Somerville, & Main, 1993), and kinesiophobia (i.e., fear of movement; Kori, Miller, & Todd, 1990).

Evidence to support a specific phobia conceptualization is found in empirical research that demonstrates the construct of pain-related anxiety as distinct from trait anxiety and general negative affectivity. Accordingly, measures developed to assess pain-related anxiety (e.g., Pain Anxiety Symptoms Scale [PASS]; McCracken et al., 1992; PASS-20; McCracken & Dhingra, 2002) have demonstrated that pain-related anxiety predicts variance in measures of disability above and beyond the contributions of negative affect and pain severity (e.g., Burns, Mullen, Higdon, Wei, & Lansky, 2000; Crombez, Vlaeyen, Heuts, & Lysens, 1999; H. D. Hadjistavropoulos, Asmundson, & Kowalyk, 2004; McCracken et al., 1992).

Another view of the relationship between pain-related anxiety and chronic musculoskeletal pain suggests pain-related anxiety may be better conceptualized as a manifestation of AS, the trait tendency to fear the somatic sensations associated with anxious arousal (Asmundson & G. R. Norton, 1995; Asmundson & Taylor, 1996). According to this perspective, highly anxiety-sensitive persons may be anxious and fearful of pain because of the autonomic arousal it produces. Like AS, pain-related anxiety centres on somatic sensations; however, as described above, pain-related anxiety extends to concerns beyond nociception. Considerable empirical evidence supports this suggestion with several researchers reporting strong associations between pain-related anxiety and AS (e.g., Asmundson & G. R. Norton, 1995; Asmundson & Taylor, 1996;

Conrod, 2006; Gonzalez, Zvolensky, Hogan, McLeish, & Weibust, 2011; Muris, Vlaeyen et al., 2001, Muris, Schmidt et al., 2001; Tsao, Allen, Evans, Lu, Myers, & Zeltzer, 2009). Moreover, Greenberg and Burns's (2003) experimental investigation of pain-related anxiety and AS in a sample of chronic pain patients found that in regression models AS accounted for the majority of variance in effects of pain-related anxiety on dependent measures.

Recently, De Peuter and colleagues (2011) proposed that *interoceptive fear conditioning* may provide a novel approach to understanding pain-related fear and anxiety among persons with chronic pain (De Peuter, Van Diest, Vansteenwegen, Ven den Berg, & Vlaeyen, 2011) . Interoceptive conditioning occurs when an interoceptive stimulus (e.g., muscle tension) is repeatedly paired with an aversive stimulus or event (e.g., pain). A contingency is believed to develop between the interoceptive stimuli (i.e., conditioned stimulus) and experiences of pain (unconditioned stimulus), which results in the interoceptive stimulus functioning as a cue signalling a probable pain experience. Based on this contingency the interoceptive cue activates a mental representation of a painful experience, which, in turn, provokes a conditioned defensive response (e.g., autonomic arousal, behavioural avoidance, catastrophic thoughts) in reaction to the anticipated experience of pain (De Peuter et al., 2011). To date, there has been no systematic investigation of this interoceptive conditioning account of pain-related fear and anxiety.

Pain-related anxiety has also been suggested as being akin to a fundamental fear, that is, fears of inherently noxious stimuli that are not reducible to other fears. Reiss's expectancy theory (1991) proposed that pathological fear states (e.g., panic, phobias)

arise from the three fundamental fears of AS, fear of negative evaluation, and illness/injury sensitivity. Factor analytic investigation has provided empirical support for the distinct nature of the fundamental fears; moreover, measures of these constructs have been found to predict significant proportions of variance in other fears and trait anxiety (Taylor, 1993). In an investigation of the construct independence of pain-related anxiety and fear of pain, Carleton and Asmundson (2009) found support for overlapping yet distinct conceptualizations of these constructs, proposing that pain-related anxiety may be a fundamental fear related to AS. Below, the empirical literature concerning pain-related anxiety is discussed in further detail.

1.4.1. Pain-related anxiety.

Pain-related anxiety and fear of pain are related although conceptually distinct constructs. To understand these distinctions it is helpful to consider the ways in which anxiety and fear have been conceptualized in general. Fear has historically been viewed as a reaction to a specific identifiable danger that typically elicits the behavioural response of escape to reduce the organism's proximity to some threat. Physiological correlates of fear include rapid sympathetic nervous system activation (e.g., increased heart rate, vasoconstriction, mydriasis) that prepares the organism for behavioural responses such as flight, fight, and freeze behaviours (Bracha, Ralston, Matsukawa, Williams, & Bracha, 2004; Cannon, 1929). Anxiety, in contrast, is seen as a diffuse state of apprehension that does not focus on a distinct object, is anticipatory in nature, and has physiological and behavioural correlates that include chronic arousal, threat vigilance, and avoidance behaviour (Barlow, 2002).

The terms *pain-related anxiety* and *fear of pain* have often been used interchangeably in the literature; indeed, studies of both fear of pain and pain-related anxiety often employ identical measures to assess these constructs. Early fear avoidance models did not include an explicit role for pain-related anxiety until Asmundson and colleagues (Asmundson et al., 2004) described an amended model – the *fear-anxiety-avoidance* model of chronic musculoskeletal pain – that distinguishes the natures and posited contributions of fear of pain and pain-related anxiety. This model proposes that some individuals are predisposed (e.g., by negative affectivity, AS, illness/injury sensitivity, early learning) to catastrophically interpret their pain which, in turn, produces a fear state (i.e., sympathetic nervous system activation) designed to protect the individual from the perceived threat (i.e., pain). These catastrophic interpretations of pain (and associated fear states) are believed to promote the development of pain-related anxiety, a future-oriented apprehension concerning pain that prompts avoidance rather than escape behaviours. Importantly, pain-related anxiety motivates hypervigilance for threat (pain) via increased attention to internal (e.g., bodily sensations) and external (e.g., pain-producing stimuli, threatening situations) threat cues thereby increasing the likelihood such threats will be detected. Resultant to anxiety-related hypervigilance is avoidance of activities expected to produce pain (e.g., bending, lifting), which underlies an array of negative sequelae (as advanced in all fear avoidance models). The distinction drawn between fear of pain and pain-related anxiety has gained empirical support from confirmatory factor analyses of responses to the PASS-20 (McCracken & Dhingra, 2002) and the Fear of Pain Questionnaire (FPQ; McNeil & Rainwater, 1998) with results suggesting they are related but distinct constructs (Carleton & Asmundson, 2009).

Pain-related anxiety has been operationalized as including symptoms of cognitive anxiety (e.g., *I can't think when in pain*), pain-related behavioural avoidance (e.g., *I try to avoid activities that cause pain*), fearful thinking about pain (e.g., *I think that if my pain gets too severe, it will never decrease*), and pain-related physiological symptoms (e.g., *When I sense pain, I feel dizzy or faint*; McCracken & Gross, 1998). Commonly measured using the 40-item PASS (McCracken et al., 1992) and the shorter PASS-20 (McCracken & Dhingra, 2002), factor analytic investigations have generally supported a four-factor structure (i.e., cognitive, physiological, escape/avoidance, and fear) of these measures in both clinical (Coons, Hadjistavropoulos, & Asmundson, 2004) and non-clinical samples (Abrams, Carleton, & Asmundson, 2007).

Pain-related anxiety has been found to be associated with a range of chronic pain-related outcomes including the prediction of behavioural performance in physical capacity evaluations (Burns et al., 2000); physical complaints beyond pain complaints among chronic pain patients (McCracken, Faber, & Janeck, 1998); as well as physical, emotional, and role functioning in persons with rheumatoid arthritis (Strahl, Kleinknecht, & Dinnel, 2000). Moreover, among rehabilitation patients with low back pain, reductions in pain-related anxiety have been found to better predict long term rehabilitation outcomes than end of treatment functional capacity levels (McCracken, Gross, & Eccleston, 2002). The reported findings concerning the relationship between pain-related anxiety and rehabilitation outcomes are not, however, unequivocal. For example, Brede and colleagues (Brede, Mayer, Neblett, Williams, & Gatchel, 2011) investigated the PASS (McCracken et al., 1992) in a sample of persons with chronic disabling occupational musculoskeletal disorders who were admitted to and completed a

multidisciplinary functional restoration program. Broadly, they found that PASS scores tended to be elevated when other measures of psychosocial distress were also elevated and that the highest PASS scores were associated with an increased likelihood of being diagnosed with DSM-IV (American Psychiatric Association [APA], 2000) Axis I (e.g., depressive and anxiety disorders) or Axis II disorders (e.g., Borderline Personality Disorder) and with an increased likelihood of seeking treatment at one year post-discharge. Moreover, their findings indicated that the PASS failed to discriminate other one-year outcomes including return to work, retention of employment, surgery to the site of the original injury, or a new injury claim associated with the site of the original injury. Despite inconsistencies in the literature, the findings generally suggest that pain-related anxiety is a construct important to the development and maintenance of chronic musculoskeletal pain and disability. Central to the current investigation is the question of whether the posited predisposing construct of AS will significantly and substantively account for pain-related anxiety in a non-clinical sample.

1.4.2. Anxiety sensitivity.

AS is the dispositional tendency to fear the somatic sensations of anxiety (e.g., elevated heart rate, dizziness, sweating) due to the belief that such sensations signal harmful physical (e.g., serious illness), social (e.g., embarrassment), or psychological (e.g., mental illness) consequences (Reiss & McNally, 1985; Taylor, 1999). AS functions as an anxiety amplifier via an escalating cycle of fearful responding to the very sensations produced by anxiety. In functional terms, persons with elevated AS tend to be alarmed by the sensations of anxiety-related arousal, which then leads to an intensification of anxiety and corresponding further increased arousal (Reiss, 1991). AS is thought to

underlie individual differences in general fearfulness and is posited to be a vulnerability factor for the development of anxiety disorders (Reiss & McNally, 1985; Taylor, 1999). A recent meta-analytic investigation supports this formulation, with large effect sizes indicating significantly higher AS among persons with anxiety disorders relative to non-clinical control groups (Olatunji & Wolitzky-Taylor, 2009).

AS has been demonstrated to be distinct from trait anxiety (i.e., the tendency to respond fearfully to a wide range of stressors; Spielberger, Gorsuch, Luschene, Vagg, & Jacobs, 1983) and has been shown to account for variance unrelated to trait anxiety (e.g., Zinbarg, Brown, Barlow, & Rapee, 2001). Etiologically, AS is posited to arise from a combination of genetic factors (Stein, Jang, & Livesley, 1999) in conjunction with learning experiences that lead to the formation of beliefs about the potentially harmful effects of physiological arousal (e.g., Watt, Stewart, & Cox, 1998). Consistent with such suppositions, evidence suggests exposure to stressful events in both young adults (Schmidt, Lerew, & Joiner, 2000) and adolescents (McLaughlin & Hatzenbuehler, 2009) are associated with elevated AS.

AS has most commonly been measured using the 16-item Anxiety Sensitivity Index (ASI; Reiss et al., 1986). Items assess fear of the arousal-related sensations of anxiety with reference to cognitive concerns (e.g., *When I cannot keep my mind on a task, I worry that I might be going crazy*), physical concerns (e.g., *It scares me when I feel faint*), and social concerns (e.g., *Other people notice when I feel shaky*). Respondents are instructed to endorse items on a Likert scale with response options ranging from 0 (very little) to 4 (very much).

The factor structure of the ASI has generated considerable debate, with initial support reported for a unitary structure (Peterson & Heilbronner, 1987; Reiss et al., 1986; Taylor, Koch, & Crockett, 1991); however, a consensus later emerged supporting a three-factor hierarchical structure with *fear of socially observable anxiety reactions* (e.g., *It is important to me not to appear nervous*), *fear of somatic sensations* (e.g., *It scares me when my heart beats rapidly*), and *fear of cognitive dyscontrol* (e.g., *It scares we when I am unable to keep my mind on a task*) subsumed under an overarching global AS construct (e.g., Lilienfeld, Turner, & Jacob, 1993; Zinbarg, Barlow, & Brown, 1997).

Despite this consensus, the ASI was found to be unstable across a number of investigations, with researchers variously reporting two- (Zvolensky et al., 2003), four- (Taylor & Cox, 1998b), and six- (Taylor & Cox, 1998a) factor structures. Attempts to address this instability led to the development of the ASI-Revised (ASI-R; Taylor & Cox, 1998b) and the Anxiety Sensitivity Profile (ASP; Taylor & Cox, 1998a), neither of which addressed the factorial instability. Later research led to the development of the ASI-3 (Taylor et al., 2007), an 18-item measure of AS that appears to have resolved the instability of the earlier measures. To date, the ASI-3 has been demonstrated to be a valid, reliable, and stable measure with a replicable factor structure consistent with that of the original ASI.

Controversy remains concerning the question of whether the latent structure of AS is continuous (i.e., dimensional) or categorical in nature. Taxometric methods, a class of statistical procedures developed to assess the latent structure of phenomena (Meehl & Golden, 1982), have been employed to evaluate the latent structure of AS; but, to date, the findings remain equivocal. Some researchers have reported a continuous latent

structure (e.g., Asmundson, Weeks, Carleton, Thibodeau, & Fetzner, 2011; Broman-Fulks et al., 2008), whereas others have found a taxonic structure that includes normative and high (pathological) AS groups (e.g., Bernstein et al., 2006; Bernstein, Zvolensky, Stewart, & Comeau, 2007) as well as distinct taxa for young men and women (Bernstein, Zvolensky, Weems, Stickle, & Leen-Feldner, 2005).

Early investigations of AS focused on its role in the etiology and maintenance of the anxiety disorders; however, recent research has supported its relevance across a broad range of domains, including mood disorders (Cox, Enns, Freeman, & Walker, 2001; Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995), hypochondriasis (Otto, Demopulos, McLean, Pollack, & Fava, 1998; Weems, Hammond-Laurence, Silverman, & Ferguson, 1997), substance abuse (S. H. Stewart, Samoluk, & MacDonald, 1999), and PTSD (Taylor, 2004). Furthermore, AS is posited to play a central role in the development and maintenance of chronic pain (Asmundson, P. J. Norton, & G. R. Norton, 1999; Asmundson & G. R. Norton, 1995; Asmundson, P. J. Norton, & Veloso, 1999; Asmundson & Taylor, 1996; Plehn, Peterson, & Williams, 1998). As described earlier, AS has been highlighted as an important component of the fear-anxiety-avoidance model of chronic pain (Asmundson, Noton, & Veloso, 1999; Asmundson et al., 2004; P. J. Norton & Asmundson, 2003). In addition, AS has been shown to be clinically relevant both as a treatment target and outcome measure in intervention protocols developed for some of the abovementioned conditions including Panic Disorder (e.g., Craske, Maidenberg, & Bystritsky, 1995) and PTSD (Wald & Taylor, 2008). Indeed, recent meta-analytic findings suggest cognitive behavioural therapy is broadly effective in reducing AS, with large effect sizes found across both treatment-seeking and at-risk

samples (Smits, Berry, Tart, & Powers, 2008). Preliminary evidence also suggests interventions aimed at reducing AS may reduce pain-related anxiety (Flink, Nicholas, Boersma, & Linton, 2009; Watt, Stewart, Lefaivre, & Uman, 2006).

1.4.3. Anxiety sensitivity and pain.

In what was likely the first study to explore AS in relation to chronic pain, Asmundson and G. R. Norton (1995) investigated AS profiles in persons with unexplained chronic back pain. Independent of pain severity, persons with high AS reported significantly greater cognitive anxiety, more fear of the negative consequences of pain, and more negative affect than those with low AS. Moreover, a significantly higher proportion of those in the high AS group were using pain medications relative to persons in the low AS group. Asmundson and G. R. Norton (1995) concluded that several aspects of the psychological distress associated with chronic pain are significantly influenced by AS.

Considerable evidence has since accumulated to support the involvement of AS in: (a) patients with chronic pain (i.e., higher pain intensity, emotional distress, depression, pain-related anxiety, disability, and more physician visits; McCracken & Keogh, 2009); (b) pain induced in laboratory settings (e.g., Keogh & Cochrane, 2002; Keogh & Mansoor, 2001); and (c), important components of the fear-avoidance model, such as pain catastrophizing and fear of pain (Asmundson et al., 2004; P. J. Norton & Asmundson, 2003). Indeed, a recent meta-analytic review of studies examining the association between AS and pain (Ocañez, McHugh, & Otto, 2010) included 41 published articles reporting on investigations of both clinical and non-clinical samples. Results of this meta-analysis indicated that in studies of clinical samples (n = 14)

aggregate effect sizes demonstrated AS was strongly related to fear of pain (g = 1.15), moderately to strongly related to negative affect (g = .95), and modestly to moderately related to disability (g = .45). In studies of non-clinical samples (n = 27) aggregate effect sizes indicated AS had a moderate to strong relationship with fear of pain (g = .96), a moderate relationship with negative affectivity (g = .64), a moderate to large relationship with affective appraisal of pain (g = .79), a moderate relationship with sensory appraisal of pain (g = .65), a small to moderate relationship with pain severity (g = .36), and a small negative relationship with pain threshold/tolerance (g = -.27).

The available empirical findings strongly support a systematic relationship between AS and the pain experience; that is, AS has been strongly associated with pain-related anxiety and fear. The nature of these relationships has not been well delineated to date; but, it has been posited that AS is a predisposing factor for pain catastrophizing (e.g., P. J. Norton & Asmundson, 2003) as well as accounting for substantive proportions of variance in measures of pain-related anxiety and fear (e.g., Greenberg & Burns, 2003; Muris, Schmidt et al., 2001).

1.4.4. Anxiety sensitivity and pain-related anxiety.

Available research suggests an overlapping but empirically distinct relationship between AS and pain-related anxiety. For example, Carleton and colleagues (2009) found that pain-related anxiety did not differ across the spectrum of anxiety and depressive disorders but was elevated among individuals diagnosed with these disorders, relative to those without diagnoses. In contrast, AS was found to be differentially elevated across these disorders, with ASI scores being significantly higher among persons

with panic disorder than for those with depressive, social anxiety, or obsessive compulsive disorder (Carleton et al., 2009)

There have also been several investigations that assess the relationship between AS and pain-related anxiety. In laboratory studies, AS has consistently been reported to be a substantive predictor of pain-related anxiety. For example, Muris, Vlaeyen et al. (2001) investigated AS and pain-related anxiety in healthy adolescents and found that AS (measured with the Childhood Anxiety Sensitivity Index [CASI]; Silverman, Fleisig, Rabian, & Peterson, 1991) accounted for substantial variance in scores on a simplified version of the PASS (McCracken et al., 1992). The most pronounced effects were found for PASS total, cognitive, somatic, and fear scores (R^2 values ranging between .34 and .46), whereas results were comparatively attenuated (R^2 = .14) for escape/avoidance scores. Similar findings were reported by Tsao and colleagues (2009) in an investigation of healthy children ages 8-18. Using structural equation modeling, AS (CASI) was found to account for 29% of the variance in anticipatory pain-related anxiety, which, in turn, was found to predict a majority of the variance in pain intensity scores (Tsao et al., 2009). Conrod (2006) found that AS significantly predicted anticipatory anxiety across neutral, social, and physical stress experimental conditions, suggesting the nature of the stressor may be less relevant to anxious responding than the presence of elevated AS. In an investigation using CO^2 challenge – an experimental protocol in which participants breathe carbon dioxide-enriched air to induce a biological challenge – Gonzalez and colleagues (2011) examined AS and pain-related anxiety as predictors of fearful responding to bodily sensations. Both AS and pain-related anxiety were found to be significant and unique predictors of post-challenge panic attacks, post-challenge panic

attack symptoms, and intensity of cognitive panic attack symptoms. In contrast, AS alone predicted post-challenge physical panic symptoms. Results were interpreted as suggesting AS and pain-related anxiety, while related, may be independently relevant constructs underlying reactivity to bodily sensations (Gonzalez et al., 2011).

In the first clinical study exploring the role of AS in persons with chronic low back pain, Asmundson and G. R. Norton (1995) reported a strong association between AS and cognitive anxiety as well as moderate associations for physiological and escape/avoidance anxiety dimensions of the PASS (McCracken et al., 1992). A later study using structural equation modeling found that even when controlling for pain severity, AS promoted pain related escape/avoidance behaviours via its influence on pain-related fear and anxiety as measured by the PASS (Asmundson & Taylor, 1996). Consistent with these results, an investigation of the role of AS with respect to pain and pain-related anxiety among persons with panic disorder and age-matched controls found that AS predicted both pain and pain-related anxiety during a cold pressor task, with mediation analyses suggesting the effect of AS on pain reports was via pain-related anxiety (Schmidt & Cook, 1999). Similar data have been reported in a study of heterogeneous chronic pain patients in which AS was found to predict substantial proportions of PASS total and subscale scores (Zvolensky, Goodie, McNeil, Sperry, & Sorrell, 2001).

Investigating a sample of chronic pain patients, Greenberg and Burns (2003) used pain-related anxiety induction (cold pressor) and social-evaluative anxiety (mental arithmetic) tasks to determine whether pain-related anxiety is better conceptualized as a specific (i.e., pain-focused) phobia or as a manifestation of AS. Participants were 70

patients (57.1% men) recruited at a healthcare facility specializing in treatment of chronic musculoskeletal pain. Subsequent to completing measures of pain-related anxiety, social evaluative anxiety, AS, and negative affect (i.e., depressive symptoms, trait anxiety), participants underwent both cold-pressor and mental arithmetic tasks. Cardiovascular data (i.e., heart rate, systolic/diastolic blood pressure) were collected during experimental tasks, and a brief checklist comprised of items assessing pain-related anxiety, social evaluative anxiety, and negative affect was completed immediately upon task completion.

These data were analyzed to determine whether pain-related anxiety predicted variance in post-task measures over and above that accounted for by AS. Results indicated pain-related anxiety was associated with pain-relevant responses during the pain induction task but also to evaluation-relevant responses during the social-evaluative anxiety task. Hierarchical regression analyses indicated AS accounted for almost all effects of pain-related anxiety on post-task responses, whereas a measure of fear of negative evaluation was associated only with evaluation-relevant responses (primarily during the mental arithmetic task). The authors interpreted the results as supporting a conceptualization of pain-related anxiety as a manifestation of AS (Greenberg & Burns, 2003). Collectively, the available empirical literature indicates an overlapping, distinct, and, as yet, insufficiently defined relationship between AS and pain-related anxiety. The importance of both pain-related anxiety and AS to the development and maintenance of chronic pain and disability suggests the relationship between these constructs warrants further investigation.

1.5. Literature review summary

The preceding review has covered representative literature concerning the nature, prevalence, and impact of chronic pain as well as historical and current conceptualizations of pain and chronic musculoskeletal pain. The evidence indicates that chronic musculoskeletal pain is a major public health issue that negatively impacts countless individuals and their families as well as imposing a significant economic burden on society. The advent of biopsychosocial models of chronic pain has led to the identification and elaboration of several negative affect-related constructs posited as important to the development and maintenance of chronic pain.

Pain-related anxiety is central to fear-anxiety-avoidance models of chronic pain and several differing conceptualizations of this construct have been advanced. Below, these perspectives are briefly reiterated. First, given that anxiety and worry are prominent among persons with chronic pain, pain-related anxiety can be viewed as simply one aspect of the negative affectivity commonly associated with chronic pain (e.g., Gatchel et al., 2007). Second, pain-related anxiety has been conceptualized as akin to a specific phobia reinforced and maintained by fear and avoidance of stimuli believed to carry threat of pain. This theoretical position underlies early fear-avoidance models of chronic musculoskeletal pain (e.g., Vlaeyen & Linton, 2000). Later refinements of these models included AS as a predisposing vulnerability factor (P. J. Norton & Asmundson, 2003) and, with the *fear-anxiety-avoidance* model, distinguished the roles of fear of pain and pain-related anxiety (Asmundson et al., 2004). A third perspective suggests pain-related anxiety may function in a manner similar to that of the fundamental fears (Reiss, 1991; Taylor, 1993), possibly via a relationship with the fundamental fear of AS

(Carleton & Asmundson, 2009). A fourth view proposes that interoceptive fear conditioning may provide a novel understanding of pain-related fear and anxiety (De Peuter et al., 2011). These authors suggest that learned contingencies develop between relatively benign interoceptive sensations (e.g., muscle twinges) and pain experiences. Based on these contingencies, interoceptive sensations come to act as cues that activate mental representations of pain experiences, thereby provoking defensive responses that include pain-related anxiety (e.g., biased attention to pain cues, behavioural avoidance, autonomic arousal). A fifth conceptualization posits that pain-related anxiety may be a manifestation of AS, the dispositional tendency to fear the somatic sensations of anxiety. Considerable evidence supports the existence of a strong relationship between AS and pain-related anxiety (e.g., Asmundson & G. R. Norton, 1995; Asmundson & Taylor, 1996). Indeed, the experimental findings reported by Greenberg and Burns (2003) indicated that the effects of pain-related anxiety were explained almost entirely by underlying AS.

To summarize, the available literature suggests that the relationship between pain-related anxiety and AS is robust but not clearly delineated. With reference to pain-related anxiety, AS has been variously conceptualized as a predisposing vulnerability factor in fear avoidance and fear-anxiety-avoidance models of chronic pain (e.g., P. J. Norton & Asmundson, 2003), as a fundamental fear associated with pain-related anxiety (Carleton & Asmundson, 2009), and as a construct that subsumes pain-related anxiety (Greenberg & Burns, 2003). Given these contradictions in the literature, further investigation concerning the relationship between pain-related anxiety and AS is warranted.

2. CURRENT INVESTIGATION

2.1. Purpose and hypotheses

The purpose of this investigation was to extend the findings of Greenberg and Burns (2003) by evaluating the relationship between pain-related anxiety and AS using a non-clinical sample and state-of-the-art pain induction and physiological monitoring equipment. The results of the Greenberg and Burns (2003) investigation supported an AS conceptualization of pain-related anxiety in a sample of persons with low-back pain. It remains unclear, however, whether a similar relationship exists between AS and pain-related anxiety in persons not experiencing current or chronic pain. Examining this question with a non-clinical analogue sample may further our understanding of basic processes that may underlie the mechanisms by which some individuals who sustain injury will go on to develop chronic pain. The rationale for studying the relationship between pain-related anxiety and AS with a non-clinical analogue sample stems from the fact that individuals who develop pain chronicity were not always that way. The use of non-clinical analogue samples enables the investigation of posited vulnerability factors (e.g., AS, pain-related anxiety) in individuals who are comparatively unaffected by a persistent pain experience (for a more complete discussion of analogue research please see Tull, Bornovalova, Patterson, Hopko, & Lejuez, 2008). Accordingly, the current investigation employed state-of-the-art physiological monitoring and pain induction equipment in an attempt to extend the results of Greenberg and Burns (2003) with a sample of healthy individuals not reporting current pain.

Although the cold pressor task employed by Greenberg and Burns (2003) has been widely used in experimental studies of pain (e.g., Keogh & Mansoor, 2001; Schmidt

& Cook, 1999; Van Damme, Crombez, Van Nieuwenborg-DeWever, & Goubert), the Medoc PATHWAY Pain and Sensory Evaluation System – ATS model (Medoc Advanced Medical Systems Ltd., Ramat Yishay, Israel) provides several advantages over this methodology. Foremost among these refinements is the computer-programmable nature of the system that enables precise control (e.g., presentation intensity, duration) of thermal stimuli. A further refinement offered by the MEDOC equipment is the capacity for precise computer-based data collection of various physiological parameters during experimental tasks.

Two experimental tasks were administered to induce both pain-related anxiety and social-evaluative anxiety. Dependent measures included physiological, behavioural, and self-report data gathered during and immediately after experimental tasks. If pain-related anxiety is better conceptualized as a pain-focused specific phobia, then PASS-20 scores were expected to significantly predict physiological, behavioural, and self-report responses signifying pain-related anxiety only during the pain-anxiety induction task. In addition, these effects were expected to remain statistically significant when controlling for AS and negative affect (i.e., depression, trait anxiety). Alternatively, if pain-related anxiety is better viewed as a manifestation of AS, then PASS-20 scores should significantly predict physiological, behavioural, and self-report responses signifying general fearfulness during both the pain-anxiety and social-evaluative anxiety induction tasks. Furthermore, these effects should be held largely in common with scores on the ASI-3. Accordingly, this investigation had two hypotheses:

1. Consistent with the view that pain-related anxiety may be a manifestation of AS, it was hypothesized that scores on a measure of pain-related anxiety (i.e., PASS-

20; McCracken & Dhingra, 2002) would significantly and substantively predict positive variance in scores on post-task dependent measures (i.e., physiological, behavioural, and self-report indices) for *both* the pain-related anxiety and social-evaluative anxiety induction tasks. In addition, it was hypothesized that these effects would remain statistically significant when controlling for effects of general negative affectivity (i.e., depression, trait anxiety).

2. It was further hypothesized that in hierarchical regression models the predictive effects of pain-related anxiety (PASS-20) on variance in dependent measures will be accounted for by scores on a measure of AS (ASI-3; Taylor et al., 2007).

2.2. Method and materials

2.2.1. Participants.

Study participants were recruited from the local community and university via posters and social media advertising (e.g., Facebook), as well as word of mouth (i.e., several participants referred friends and family members). Potential participants contacted the Anxiety and Illness Behaviours Lab by telephone or email to arrange a telephone screening appointment. Upon eligibility determination, participants were provided a link to the pre-experiment questionnaire battery and an appointment was arranged for them to attend the lab to complete the experimental tasks. Eligibility exclusion criteria assessed during telephone screening included the following: (a) a history of bipolar or psychotic disorders, (b) regular use of benzodiazepine or antipsychotic medications, (c) current alcohol or substance abuse problems, (d) current acute or chronic pain conditions, and (e) an inability to read English well enough to complete the questionnaires. In addition to the noted exclusion criteria, a modified

version of the Mini-International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) was administered during screening to assess for the possible presence of clinically significant current symptoms of DSM-IV (APA, 2000) Axis I psychological disorders. Similarly, the general self-reported health of potential participants was assessed during screening by administering the Physical Activity Readiness Questionnaire (PAR-Q).

2.2.2. Measures.

Self-report trait measures.

Anxiety Sensitivity Index-3 (ASI-3; Taylor et al., 2007; see Appendix I). The ASI-3 is an 18-item self-report measure that assesses the dispositional tendency to fear anxiety-related arousal sensations due to the belief such sensations signal imminent harmful consequences (e.g., *It scares me when my heart beats rapidly*; Reiss et al., 1986; Taylor, 1999). Items are rated on a 5-point Likert scale ranging from 0 (very little) to 4 (very much). The ASI-3 has three subscales that measure: (a) fear of cognitive dyscontrol (e.g., *It scares me when I am unable to keep my mind on a task*), (b) fear of somatic sensations (e.g., *When my stomach is upset, I worry that I might be seriously ill*), and (c), fear of socially observable anxiety reactions (e.g., *When I begin to sweat in a social situation, I fear people will think negatively of me*). The development of the ASI-3 was prompted, in part, by the psychometric instability of the original ASI (Taylor et al., 2007). Studies of factorial validity support a robust 3-factor structure for the ASI-3 consistent with the three originally theorized dimensions of AS (i.e., cognitive, physical, social concerns; e.g., Zinbarg et al., 1997). Relative to the original ASI, the ASI-3 has demonstrated improved internal consistency and factorial validity as well as good convergent, discriminant, and criterion-related validity (Taylor et al., 2007). In the first

independent study to examine the properties of the ASI-3, Osman et al. (2010) reported the measure to have excellent properties consistent with the findings of the development studies conducted by Taylor et al. (2007). The bi-factor model (i.e., a global AS factor subsuming dimensions of cognitive, physical, and social concerns) was found to be the best fit to the data. Also consistent with the Taylor (2007) studies, no systematic sex-differences were found on ASI-3 total and subscale scores indicating no need for sex-specific norms.

Brief Fear of Negative Evaluation-Straightforward Items (BFNE-S; Carleton, Collimore, McCabe, & Antony, 2011; see Appendix II). The BFNE-S is an 8-item version of the original BFNE (Leary, 1983) that assesses fears of negative evaluation (e.g., *I am afraid that people will find fault with me*). The measure consists of the eight straightforwardly worded items (i.e., items 1, 3, 5, 6, 8, 9, 11, 12) from the original BFNE (Leary, 1983). Items are responded to on a 5-point Likert scale, ranging from 0 (not at all characteristic of me) to 4 (extremely characteristic of me). The BFNE-S was developed to address suggestions (Rodebaugh et al., 2004; Weeks et al., 2005) that the straightforwardly-worded items were more reliable and valid indicators of the fear of negative evaluation than the reverse-scored items. The BFNE-S has demonstrated acceptable internal consistency (i.e., scale alphas > .92), factorial validity, and construct validity in undergraduate (Carleton, Collimore, & Asmundson, 2007; Rodebaugh et al., 2004) and clinical (Weeks et al., 2005) samples. A suggested cut-off score of 25 has been proposed as being indicative of clinically significant social anxiety (Carleton et al., 2011). The BFNE-S was included in this investigation to provide a manipulation check for the planned social-evaluative anxiety task (described below) as well as to provide data

for use in regression analyses to evaluate variance accounted for in post-task measures relative to AS and pain-related anxiety.

Center for Epidemiologic Studies – Depression scale (CES-D; Radloff, 1977; see Appendix III). The CES-D is a widely-used 20-item measure designed to assess symptoms of depression in the general population. Items are phrased as self-statements (e.g., *I did not feel like eating; My appetite was poor; I felt hopeful about the future*) and respondents are instructed to rate how frequently each item applied to them during the past week using a 4-point Likert scale ranging from 0 (Rarely or none of the time [less than 1 day]) to 3 (Most or all of the time [5-7 days]). Higher scores indicate more depressive symptoms.

Pain Anxiety Symptoms Scale-20 (PASS-20; McCracken & Dhingra, 2002; see Appendix V). The PASS-20 is a 20-item measure developed from the original 40-item PASS (McCracken, Gross, Sorg, & Edmands, 1993). Items on the PASS-20 are rated on a 6-point Likert scale ranging from 0 (never) to 5 (always). The scale assesses four distinct components of pain-related anxiety that include: (a) cognitive anxiety (e.g., *I cannot think straight when in pain*); (b) pain-related fear, (e.g., *Pain sensations are terrifying*); (c) escape and avoidance (e.g., *I try to avoid activities that cause pain*); and (d), physiological anxiety (e.g., *Pain makes me nauseous*). The PASS-20 has good internal consistency and correlates highly with the earlier PASS (McCracken & Dhingra, 2002). Factorial validity for both PASS-20 total and subscale scores has been demonstrated in both clinical (e.g., Coons et al., 2004) and non-clinical (Abrams et al., 2007) samples. Neither the instructions for completing the PASS-20, nor the item content, preclude its use in persons not reporting current pain (Abrams et al., 2007).

State-Trait Anxiety Inventory (STAI; Spielberger et al., 1983; not appended for copyright reasons). The *State-Trait Anxiety Inventory* is a 40-item self-report measure designed to assess both state (e.g., *I feel nervous*) and trait anxiety (e.g., *I feel like a failure*). Items are endorsed on a 4-point Likert scale ranging from *not at all* to *very much so* for the state scale and *almost never* to *almost always* for the trait scale. The STAI has been shown to have good internal consistency, good stability for trait anxiety and low stability for state anxiety (as expected), as well as adequate validity (Spielberger et al., 1970).

Self-report dependent measures.

Pain-affectivity checklists (after Greenberg & Burns, 2003; see Appendix IV) were designed for the purposes of this investigation. A separate checklist of relevant items was employed for each of the experimental tasks. Item content followed the approach taken by Greenberg and Burns (2003) to parsimoniously assess variables of interest subsequent to the two task conditions (i.e., pain induction, mental arithmetic). Participants endorsed checklist items on a scale ranging from 0 (not at all) to 10 (extremely). Five of the items were common to both checklists and included the following: one item assessed current pain (i.e., *Please rate the degree of pain you are currently experiencing*); and four items assessed general negative affectivity (i.e., *Please rate the degree you currently feel… anxious, irritated, tense, nervous*). For the mental arithmetic task (intended to induce social-evaluative anxiety) the checklist included the following four items: *Please rate the degree you were… concerned about making a good impression, bothered about being judged on your performance, worried you would do poorly on this task, afraid you would embarrass yourself*. Scores on these four items

were summed to create the composite dependent variable, *mental arithmetic social-evaluative anxiety* (i.e., MA-SA). For the pain induction task (intended to induce pain-related anxiety) the checklist included the following four items: *Please rate the degree to which you were… distressed by the pain*, *afraid of being hurt by doing this task, scared your pain will increase,* and *preoccupied with the pain*. Scores on these four items were similarly summed to comprise the composite dependent variable, *pain induction pain-anxiety* (PI-PA).

Biophysiological measures.

During pain induction and mental arithmetic tasks, participants had aspects of their autonomic nervous system (ANS) functioning monitored and recorded using the BIOPAC MP 150 Data Acquisition System (MP 150 Data Acquisition System, Ethernet for Macintosh, BIOPAC Systems Inc., Goleta, CA). Heart rate data were collected using a 'C' series electrocardiogram amplifier with shielded leads from BIOPAC. Respiration rate data were collected using a chest respiratory belt (RSP100C amplifiers, TSD201 transducers from BIOPAC). Systolic and diastolic blood pressure data were collected via a pressure sensor attached to the wrist over the radial artery (NIBP100B-R from BIOPAC).

For heart and respiration rate, a five-minute resting baseline period was recorded using BioPac with the final two minute period comprising retained baseline data. Task data were collected for the time period during which the task took place. For all dependent measures the pain tolerance task was used as it was considered the most demanding of the three pain tasks (i.e., warmth detection, pain threshold, pain tolerance). Baseline blood pressure data were recorded at the end of the five-minute baseline period,

whereas task blood pressure data were collected immediately after task completion. Again, the pain tolerance task was used as it was viewed the most demanding of the three pain induction tasks. Systolic and diastolic blood pressure was measured five times with retained baseline values being the mean of these values. Task blood pressure measures were taken immediately after completion of each of the experimental tasks. Only the first reading was retained as it was observed that blood pressure tended toward baseline values as subsequent readings were taken. To calculate a mean of all collected post-task values would have obscured task effects on this parameter.

Behavioural indices.

Two behavioural indices were assessed, including pain tolerance (i.e., the mean of the three pain tolerance values in degrees Celsius) and the number of correct subtractions on the mental arithmetic task. For the mental arithmetic task, an incorrect answer was not viewed as rendering subsequent responses as incorrect. So long as a correct subtraction was reported, it was scored as correct, irrespective of whether an incorrect answer preceded it.

2.2.3. Equipment.

Thermal stimulation pain (i.e., heat pain) was delivered using the Medoc Pathway Pain and Sensory Evaluation System – ATS model (Ramat Yishay, Israel). The Pathway system enables precise programmable control of thermal heat stimuli using the Advanced Thermal Stimulator (ATS) thermode. The thermode consists of a 30 mm diameter round contact area that delivers stimuli temperatures ranging between 0°C and 55°C at a rate of change of up to 8°C per second. Included in the PATHWAY system are several hardware and software mechanisms engineered to ensure participant safety. When the

system is activated, hardware test procedures are performed automatically to ensure system sensors are functioning and will prevent the system from being used if any malfunction is detected. System software also continuously monitors thermode functioning and is designed to automatically disable the system in the event of a malfunction. Specifically, the system monitors the temperature of the thermode and prevents it from heating higher than 55°C. If the temperature should somehow reach 57°C, an emergency hardware failsafe will engage to automatically disconnect power to the thermode. A final safety feature of the PATHWAY system comprises manually operated mechanisms – available to both participant and system operator – that are designed to stop the trial at any time. An emergency stop button was accessible to the system operator that, when activated, would immediately end the trial. The participant could also terminate the trial at any time by activating a manual electrical trigger (held in the participant's hand) attached to the machine.

2.2.4. Procedure.

Upon determination of eligibility, participants were provided with an internet link to access the online pre-experiment questionnaire battery (i.e., ASI-3, Taylor et al., 2007; BFNE-S, Carleton et al., 2011; CES-D, Radloff, 1977; PASS-20, McCracken & Dhingra, 2002; STAI, Spielberger et al., 1983). They were also provided a unique participant number that was used across types of data collection to ensure that all data gathered were linked to the same individual. When participants had completed the pre-experiment questionnaire battery, an appointment was made for a convenient date and time to attend the lab where they completed the experimental tasks.

Participants were tested individually. When a participant arrived at the lab he or she was greeted by the experimenter and brought to the experimental room and was comfortably seated. The experimental procedure was explained and questions were answered. Prior to completing the pre-experiment online questionnaire battery, the participant was provided with information describing the experiment as well as a response field in which to indicate his or her consent to take part. All participants were asked if they had read the information explaining the experiment. Many participants reported that they had not reviewed this information so, to ensure informed consent, a brief explanation was provided before the experiment began.

Two experimental tasks were completed by each participant. One task, intended to induce social-evaluative anxiety, comprised a mental arithmetic manipulation; the other task, intended to induce pain-related anxiety, consisted of a pain induction task. The two tasks are described in detail below. Task presentation was randomly counterbalanced to address the possibility of order effects

For both tasks, the participant was comfortably seated facing a computer monitor and was attached to the biophysiological monitoring equipment. This procedure was carried out by a male researcher for male participants and a female researcher for female participants. After the biophysiological monitoring equipment was attached, an adaptation period of five minutes ensued during which acquisition of baseline heart and respiration rate was obtained.

Experimental tasks.

The experimental tasks proceeded subsequent to the collection of baseline data. Depending on task order assignment, participants began with either the mental arithmetic

task or the pain induction task. Task order was randomized in blocks of two by using a coin flip. Participants assigned to the mental arithmetic/pain induction task order received standardized task instructions and began when ready. Immediately upon task completion participants had their blood pressure measured while they completed the mental arithmetic post-task checklist (Appendix V). Subsequent to completion of the checklist, participants underwent a five-minute recovery period after which five minutes of baseline data were again gathered prior to administration of the pain induction task. As with the first task, the final two minutes of this five-minute period comprised retained baseline data. The pain-induction task was then performed. As with the mental arithmetic, immediately upon completion of the task the participant's blood pressure was measured while he or she completed the post-task checklist.

The mental arithmetic task consisted of a timed backward subtraction task. Participants were instructed to mentally subtract 7 from 8259 and provide their answers verbally to the researcher. They were instructed to perform the task as quickly and accurately as possible and continue until told to stop after two minutes had elapsed. While the participant performed this task the experimenter recorded the participant's answers on a document which was later used to score the number of correct subtractions. This variable provided an index of performance behaviour. To facilitate the induction of social-evaluative anxiety during the task, the experimenter provided the participant with two standardized comments at approximately 20 second intervals (i.e., "You need to go faster"; "You're making too many mistakes"). Immediately after completion of the mental arithmetic task, participants were administered the mental arithmetic post-task

checklist (i.e., pain-affectivity checklist; described in the Measures section) to assess current pain, general negative affectivity, and anxiety specific to social evaluation.

Pain-related anxiety was induced via administration of quantitative sensory testing procedures (QST; Rolke et al., 2006) using Medoc Pathway equipment. Commonly employed for the investigation of pain perception, QST investigates pain perception via several modalities (e.g., thermal, mechanical) by administering controlled external stimuli to consenting research participants. Because QST is a psychophysical test, the procedures require a participant who is able and willing to report their subjective experience of the stimuli. As is true of other research paradigms that gather subjective responses, QST has been found to be sensitive to testing conditions (e.g., Chong & Cros, 2004; Shy et al., 2003). Variables such as stimulus modality, equipment properties, ramping rate (i.e., rate of increase/decrease of stimulus intensity), trial duration, as well as participant and experimenter variables have all been observed to affect QST results. In order to maintain reliability, participant instructions were standardized and experimenters were trained in the use of the equipment and procedures.

Pain-related anxiety induction was performed via administration of pain threshold and tolerance testing. The procedure involved the following steps: (a) standardized instructions regarding the nature of the task were provided to the participant; (b) physiological monitoring equipment was attached; (c) the stimulator thermode was affixed to the upper inner non-dominant forearm; (d) baseline data collection was performed; (e) when ready, the participant underwent pain threshold and tolerance testing; and (e) immediately after testing, the participant was asked to complete the pain-induction post-task checklist (Appendix VI).

Threshold testing included warmth detection threshold (WDT) and heat pain threshold (HPT). Tolerance testing was conducted to determine heat pain tolerance (HT). Each threshold and tolerance test was estimated by averaging the participant's responses over three trials, with an inter-trial interval of 30 seconds. Each trial began at a baseline temperature of 32°C and increased in temperature at a rate of 0.5°C per second. Trials ended when the participant depressed a manual trigger (i.e., computer mouse), establishing the trial result at the current temperature and signaling that he or she can: (a) just perceive the sensation of warmth, (b) just perceive the sensation of heat pain, and (c) feel that heat pain has become intolerable.

At the conclusion of the two experimental tasks, the participant was provided with a five-minute resting period during which physiological monitoring equipment was removed. He or she was then provided with a brief explanation and printed information regarding the nature and purposes of the study. An offer was made to answer questions and discuss any concerns the participant may have had. No participant expressed any notable concerns. Before leaving the lab each participant was provided with a $20.00 Tim Hortons gift card as compensation for time and effort.

3. RESULTS

3.1. Sample characteristics

Participants who completed the study included 23 men and 38 women (N = 61; M age = 31, SD = 11.45, age range: 18-61). Participants reported their marital status as single (37.7%), married/common-law (27.9%), in a relationship but not cohabitating (21.3%), or separated/divorced (1.6%). The highest education levels obtained by participants were reported to include high school (6.6%), some college/university

(16.4%), college diploma/certificate (9.8%), undergraduate degree (24.6%), some graduate school (23%), and graduate degree (19.7%). Regarding employment status, 45.9% of participants indicated that they were students, 42.6% reported being employed full-time, 27.9% reported working part-time, 3.3% reported being underemployed (i.e., working less than desired), 3.3.% reported being self-employed on a part-time basis, 1.6% reported being unemployed, 1.6% reported being retired, and 1.6% reported awaiting pending employment. Reported ethnic backgrounds of participants included Caucasian (78.7%), South Asian (9.8%), East Asian (6.6%), First Nations/Métis (3.3%), and Other (1.6%).

Nine participants were excluded from the study during the screening phase. Specifically, three were excluded because they reported antipsychotic or anxiolytic medication use, one was excluded due to limited English language proficiency, three were excluded due to reported current pain conditions, and two were excluded due to health issues identified on the Physical Activity Readiness Questionnaire (PAR-Q). A further two participants were excluded during the experimental phase of the study, one due to elevated blood pressure and the other due to a fracture injury sustained between the screening and experimental phases of the study. No participants were excluded on the basis of their MINI screen results assessing DSM-IV Axis one symptoms. Three participants who reported current distress were provided resource information and encouraged to consider attending an appropriate health provider (e.g., University of Regina's Counselling Services); all three of these participants completed the study.

3.2. Preliminary analyses

3.2.1. Descriptive statistics.

The means, standard deviations, skew, kurtosis, scale alphas and mean inter-item correlations (where applicable) for study trait measures (i.e., PASS-20, BFNE-S, ASI-3, STAI-T, CES-D) and associated subscales are presented in Table 1 below. All data were assessed for normality (i.e., scatterplot inspection, review of indicators of skew and kurtosis). The distributions of trait measure scores (i.e., PASS-20, BFNE-S, ASI-3, STAI-T, CES-D) tended to be positively skewed (i.e., toward lower scores) and exhibited high levels of variance, as indicated by inspection of histograms, and generally low kurtosis values (i.e., < 1.0).

Independent sample t-tests were conducted to assess possible sex differences on trait (i.e., PASS-20, BFNE-S, ASI-3, STAI-T, CES-D) and dependent (i.e., post-task subjective pain report, number of correct subtractions, pain tolerance, pain induction negative affectivity, mental arithmetic negative affectivity, pain anxiety during pain induction, social anxiety during mental arithmetic) measures. For trait measures, statistically significant sex differences were found only for the BFNE-S, with women reporting higher scores than men, $t(59) = 2.25$, $p = .028$, M difference = 4.89, $r^2 = .08$, a finding consistent with previous research (e.g., Carleton et al., 2007). On dependent measures, statistically significant sex differences were found only for pain tolerance. Men reported tolerating higher temperatures than women $t(35) = 3.13$, $p = .004$, M difference = 1.90, $r^2 = .22$. Levene's test indicated unequal variances ($F = 14.37$, $p < .001$), so the degrees of freedom were adjusted from 59 to 35. This finding was

consistent with previous empirical research indicating that women are more sensitive to pain (for a recent review see Wiesenfeld-Hallin, 2005).

Two-tailed Pearson bivariate correlational analyses were performed to assess relationships among trait and dependent measures. Statistically significant positive correlations (all *p*s < .001) were found among all pre-experiment trait measures (i.e., PASS-20, BFNE-S, ASI-3, STAI-T, CES-D; see Table 2.). Following the recommendations of Cohen (1988), all correlation coefficients were interpreted as being in the moderate to high range. Important to the purposes of this investigation, correlations between measures of AS (ASI-3) and both pain-related anxiety (PASS-20) and fear of negative evaluation (BFNE-S) were in the high range (r^2 = .546 and .557, respectively), whereas a moderate correlation was found between measures of pain-related anxiety (PASS-20) and fear of negative evaluation (BFNE-S; r^2 = .261).

Relationships between trait and dependent measures were also evaluated with Pearson correlational analyses. Due to the large number of relationships assessed these results are presented in two separate tables below (Tables 4a, 4b). If pain-related anxiety and fear of negative evaluation characterize concerns associated with specific stimuli, then a pattern of correlations was expected wherein PASS-20 and the BFNE-S scores would be positively associated predominantly with dependent measures (i.e., cardiovascular change scores, negative affect, pain, behavioural indices) specific to the pain induction and mental arithmetic tasks, respectively. Such a pattern of associations was not found. Only two statistically significant correlations, both negative, were found between trait measures and cardiovascular change scores (i.e., the ASI-3 and BFNE-S were both negatively correlated with mental arithmetic systolic blood pressure change

scores [MA-SBP]). These generally null findings were consistent with those reported by Greenburg and Burns (2003) for relationships between trait and cardiovascular indices. A positive statistically significant correlation was found between the PASS-20 and the pain induction pain-anxiety checklist (PI-PA), while the BFNE-S was positively correlated with both PI-PA and the mental arithmetic social evaluative anxiety checklist (MA-SA).

Regarding associations between trait measures and post-task checklists and behavioural indices, the current data exhibited markedly fewer statistically significant correlations than reported in the previous study. For their clinical sample, Greenburg and Burns (2003) reported a pattern of statistically significant generally positive overlapping correlations between the PASS and ASI and post-task checklists and behavioural indices. Their results also showed that FNE was distinctly and positively associated with variables describing social-evaluative fears. A similar pattern of results was conspicuously absent from the current data (see Table 4b). For the current sample, the BFNE-S and PASS-20 exhibited coinciding positive correlations only for the pain induction post-task pain anxiety checklist (PI-PA). The BFNE-S and CES-D similarly exhibited an overlapping positive correlation for the mental-arithmetic post-task social anxiety checklist. The ASI-3 was not statistically significantly correlated with any of the non-cardiovascular dependent measures.

Table 1. Descriptive statistics for trait measures

	M	*SD*	Skew	Kurtosis	Scale α	*M* inter-item *r*
PASS-20	19.246	16.856	1.132	0.228	.952	.515
PASS-20 cog	6.984	5.402	1.072	0.374	.934	.745
PASS-20 fear	3.115	4.443	1.84	2.754	.915	.688
PASS-20 esc/av	5.967	5.263	0.868	-0.089	.839	.527
PASS phys	3.180	3.952	1.197	0.326	.826	.493
BFNE-S	19.787	8.505	0.435	-0.934	.957	.739
ASI-3	11.426	9.161	1.108	0.365	.878	.304
ASI-3 cog	2.148	3.224	1.856	3.104	.847	.510
ASI-3 soc	6.885	4.807	0.899	0.155	.795	.397
ASI-3 phys	2.393	2.906	2.224	7.153	.709	.284
STAI-T	37.525	11.153	1.182	1.615	.941	.448
CES-D	9.738	10.622	2.578	8.494	.937	.442

Note. $N = 61$; PASS-20 = Pain Anxiety Symptoms Scale-20; PASS-20 cog = cognitive subscale; PASS-20 esc/av = escape/avoidance subscale; PASS-20 phys = physiological subscale; BFNE-S = Brief Fear of Negative Evaluation-Straightforward Items; ASI-3 = Anxiety Sensitivity Index-3; ASI-3 cog = cognitive concerns subscale; ASI-3 soc = social concerns subscale; ASI-3 phys = physiological concerns subscale; STAI-T = State-Trait Anxiety Inventory-Trait scale; CES-D = Center for Epidemiologic Studies-Depression Scale

Table 2. Zero-order correlations among trait measures

	PASS-20	BFNE-S	ASI-3	STAI-T	CES-D
PASS-20	-	0.511	0.739	0.569	0.566
BFNE-S		-	0.746	0.652	0.488
ASI-3			-	0.672	0.587
STAIT-T				-	0.813
CES-D					-

Note: N = 61; All correlations significant at the .001 level; PASS-20 = Pain Anxiety Symptoms Scale-20; BFNE-S = Brief Fear of Negative Evaluation-Straightforward Items; ASI-3 = Anxiety Sensitivity Index-3; STAI-T = State-Trait Anxiety Inventory-Trait Scale; CES-D = Center for Epidemiologic Studies-Depression Scale

Table 3. Descriptive statistics for dependent measures

	M	*SD*	Skew	Kurtosis	Scale α	*M* inter-item r
PI-NA	8.787	5.811	2.124	5.148	.908	.719
PI-PA	14.377	7.847	0.965	0.607	.846	.583
MA-NA	15.148	8.469	0.725	-0.378	.900	.697
MA-SA	22.541	11.372	0.193	-1.076	.941	.801
TOL	48.726	2.277	-0.051	1.025	-	-
MA √	17.852	11.101	1.054	1.025	-	-
MA pain	1.344	0.728	2.044	3.192	-	-
PI pain	2.639	1.924	1.421	1.269	-	-

Note. PI-NA = pain induction negative affect; PI-PA = pain induction post-task pain anxiety; MA-NA = mental arithmetic negative affect; MA-SA = mental arithmetic post-task social evaluative anxiety; TOL = pain tolerance (degrees Celsius); MA √ = number of correct subtractions; MA pain = subjective pain rating post mental arithmetic task; PI pain = subjective pain rating post pain induction task;

Table 4a. Zero-order correlations among trait scales and cardiovascular measures

		PASS-20	BFNE-S	ASI-3	STAI-T	CES-D
MA-SBP	*r*	-.145	**-.356****	**-.296***	-.155	-.057
n = 61	*p*	.266	**.005**	**.020**	.234	.662
MA-DBP	*r*	.125	-.213	-.028	-.002	.060
n = 61	*p*	.336	.100	.829	.987	.646
MA-HR	*r*	-.063	-.177	-.070	-.123	-.001
n = 58	*p*	.638	.183	.600	.358	.993
MA-RESP	*r*	.035	.081	.086	.057	-.103
n = 58	*p*	.794	.546	.520	.670	.441
PI-SBP	*r*	-.132	-.025	.036	-.182	-.149
n = 61	*p*	.312	.848	.784	.162	.251
PI-DBP	*r*	.041	.071	.165	-.083	-.141
n = 61	*p*	.755	.589	.205	.526	.277
PI-HR	*r*	-.107	-.127	-.232	-.179	-.236
n = 61	*p*	.411	.328	.072	.167	.067
PI-RESP	*r*	.219	.145	.126	.089	.124
n = 61	*p*	.090	.263	.334	.494	.342

Note. MA-SBP = systolic blood pressure residualized change for mental arithmetic task; MA-DBP = diastolic blood pressure residualized change for mental arithmetic task; MA-HR = heart rate residualized change for mental arithmetic task; MA-RESP = respiration rate residualized for during mental arithmetic task; PI-SBP = systolic blood pressure residualized change score for pain induction task; PI-DBP = diastolic blood pressure residualized change for pain induction task; PI-HR = heart rate residualized change for pain induction task; PI-RESP = respiration rate residualized change for pain induction task

Significant correlations in bold; ** = significant at $p < .01$ level; * = significant at $p < .05$ level

Table 4b. Zero-order correlations between trait scales and post-task checklists / behavioural indices

		PASS-20	BFNE-S	ASI-3	STAI-T	CES-D
PI-NA	*r*	.196	.249	.093	.209	.109
(*n* = 61)	*p*	.130	.053	.474	.107	.403
MA-NA	*r*	.063	.117	-.001	.162	.127
(*n* = 61)	*p*	.631	.369	.992	.212	.328
PI-PA	*r*	**.372****	**.260***	.218	.071	.151
(*n* = 61)	*p*	**.003**	**.043**	.091	.588	.245
MA-SA	*r*	.152	**.327***	.152	.216	**.282***
(*n* = 61)	*p*	.241	**.010**	.241	.094	**.028**
MA √	*r*	-.059	-.027	-.003	-.102	-.081
(*n* = 61)	*p*	.650	.836	.980	.433	.535
TOL	*r*	-.193	-.155	-.091	-.192	-.069
n = 61	*p*	.135	.231	.486	.138	.595
PI pain	*r*	.231	.166	.207	.088	.148
(*n* = 61)	*p*	.073	.200	.110	.499	.256
MA pain	*r*	.068	.044	.118	.084	.180
(*n* = 61)	*p*	.604	.734	.367	.519	.165

Note. PI-NA = pain induction negative affect; MA-NA = mental arithmetic negative affect; MA √ = number of correct subtractions; TOL = pain tolerance temperature; PI-PA = pain induction post-task pain anxiety; MA-SA = mental arithmetic post-task social evaluative anxiety; PI pain = subjective pain rating post pain induction task; MA pain = subjective pain rating post mental arithmetic task

Significant correlations in bold; ** = significant at $p < .01$ level; * = significant at $p < .05$ level

3.2.2. Baseline-task cardiovascular changes.

Cardiovascular changes (i.e., heart rate, systolic/diastolic blood pressure, respiration rate) between baseline and task periods were assessed with paired sample *t*-tests. Task values were statistically significantly higher than baseline measurements for both mental arithmetic systolic, $t(60) = 4.87$, $p < .001$, $r^2 = .28$, and diastolic, $t(60) = 4.05$, $p < .001$, $r^2 = .21$, blood pressure. All other baseline-task comparisons were not found to significantly differ (i.e., all *p*s > .10; see Table 5 below). Current reported pain was assessed with one item for both experimental conditions post-task. A paired-sample *t*-test was performed to assess the expectation that significantly higher levels of current pain would be reported post-pain induction than post-mental arithmetic. Consistent with expectation, pain levels were reported to be higher post-pain induction than post-mental arithmetic, $t(60) = 6.67$, $p < .001$, $r^2 = .43$.

Table 5. Baseline and task means for dependent measures / paired samples *t*-tests (baseline/task mean differences)

Variable	**Period**			**Period**		
	MA baseline	**MA task**	***M* difference**	**PI baseline**	**PI task**	**M difference**
HR (bpm)	66.65 (10.23)	68.07 (9.49)	1.42, $p = .287$	67.71 (9.44)	65.33 (8.18)	2.39, $p = .287$
SBP	**120.07 (10.90)**	**126.85 (13.29)**	**4.87, $p < .001$**	122.79 (11.00)	124.52 (19.88)	1.74, $p = .469$
DBP	**72.74 (8.68)**	**77.15 (10.41)**	**4.41, $p < .001$**	74.90 (7.60)	76.67 (10.55)	1.77, $p = .105$
RESP (bpm)	11.13 (3.02)	10.46 (2.93)	0.68, $p = .182$	11.04 (3.40)	10.58 (3.16)	0.46, $p = .314$
NA		15.15 (8.47)			8.79 (5.81)	
SA		22.54 (11.37)				
PA					14.37 (7.85)	
MA √		17.85 (11.10); range = 1 – 51				
TOL (deg. C)					48.73 (2.28); range = 43.33 – 52.14	

Note. MA = mental arithmetic; PI = pain induction; HR (bpm) = heart rate (beats per minute); SBP = systolic blood pressure; DBP = diastolic blood pressure; RESP (bpm) = respiration (breaths per minute); NA = negative affectivity; SA = social anxiety; PA = pain anxiety; MA √ = number of correct subtractions; TOL (deg. C) = pain tolerance (degrees Celsius)

Statistically significant comparisons in bold

3.2.3. Task order effects.

Task order effects were evaluated to assess the possibility that participants who completed the pain induction task first may have exhibited a pain-related anxiety carry-over effect that inflated results on the mental arithmetic task. If such an effect had occurred, then PASS-20 scores should have been significantly correlated with fearful responses only among participants who performed the pain induction task first (i.e., pain induction/mental arithmetic task order). To assess for the potential presence of this effect, for each task presentation order (i.e., pain induction/mental arithmetic; mental arithmetic/pain induction) Fisher *r* to *z* transformations were performed on significant PASS-20 correlations with post-task measures (i.e., cardiovascular, negative affectivity, social-evaluative anxiety, pain-related anxiety variables). Post-task pain anxiety (PI-PA) – the one variable positively correlated with the PASS-20 – was assessed using a freely available web-based calculator (i.e., http://vassarstats.net/rdiff.html) to determine whether the correlation coefficients significantly differed across the two presentation orders. Results indicated that there were no statistically significant differences between the correlations for each task order ($p = .459$). Thus, it was concluded that no task order effects were evident.

3.3. Main analyses

3.3.1. Hypothesis 1.

Hierarchical regression analyses were performed to assess the primary hypothesis that PASS-20 scores would significantly and substantively predict scores on post-task measures (i.e., physiological, behavioural, and self-report indices) for both the pain-related anxiety and social-evaluative anxiety induction tasks while controlling for effects

of general negative affectivity (i.e., depressive symptoms, trait anxiety). In the first of these analyses, measures of negative affect (i.e., CES-D, and STAI-T scores) were entered on the first step with PASS-20 scores following on the second step. Following the approach of Greenberg and Burns (2003), dependent measures for these analyses were the post-task variables (i.e., physiological, behavioural, and self-report indices) found to correlate significantly with the PASS-20. The pain induction post-task pain anxiety (PI-PA) measure was the only dependent variable found to be significantly correlated with the PASS-20 and, accordingly, was the dependent variable in these analyses. The first model entering the CES-D and STAI-T did not significantly predict variance in PI-PA scores, $F(2, 58) = .92$, $p = .403$, adjusted $R^2 = .00$. Adding the PASS-20 on the second step resulted in a statistically significant model, $F(3, 57) = 3.98$, $p = .012$, that substantially increased the variance accounted for, $\Delta R^2 = .142$. Thus, 14% of the variance in PI-PA scores was uniquely accounted for by the PASS-20.

A similar second set of analyses was performed to evaluate the extent to which BFNE-S scores accounted for variance in post-task variables while controlling for the effects of negative affectivity. As in the previous analyses, measures of negative affectivity (i.e., CES-D, STAI-T) were entered on the first step and then followed with the BFNE-S on the second step. Dependent measures were the post-task variables found to correlate significantly with the BFNE-S. Only the pain-induction post task measure of pain anxiety (PI-PA) and the mental arithmetic post-task measure of social evaluative anxiety (MA-SA) were significantly positively correlated with the BFNE-S and, accordingly, comprised the dependent measures for two sets of analyses. The first model (identical to the first step in the previous analyses) entering the CES-D and STAI-T failed

to significantly predict variance in PI-PA scores, $F(2, 58) = .92$, $p = .403$, adjusted $R^2 = .00$. Adding the BFNE-S on the second step also failed to result in a statistically significant model, $F(3, 57) = 2.58$, $p = .06$. For this set of analyses the BFNE-S was not found to statistically significantly predict variance in PI-PA scores. Next evaluated was the extent to which the BFNE-S would predict variance in MA-SA scores above and beyond that accounted for by measures of negative affectivity. The first model entering the CES-D and STAI-T failed to significantly predict variance in MA-SA scores, $F(2, 58) = 2.52$, $p = .089$, adjusted $R^2 = .05$. Adding the BFNE-S on the second step resulted in a significant model, $F(3, 57) = 3.35$, $p = .025$, that substantially increased the variance accounted for, $\Delta R^2 = .07$. Thus, the BFNE-S was found to uniquely account for 7% of the variance in MA-SA scores.

To summarize, for the current sample, the PASS-20 and BFNE-S were predictive only of variance in task-relevant variables. Neither the STAI-T nor CES-D was found to be significant predictors of variance in PI-PA or MA-SA scores. Thus, the results did not support the primary hypothesis that PASS-20 scores would significantly and substantively predict scores on post-task dependent measures (i.e., physiological, behavioural, and self-report indices) for both the pain-related anxiety and social-evaluative anxiety induction tasks while controlling for effects of general negative affectivity (i.e., depressive symptoms, trait anxiety).

3.3.2. Hypothesis 2.

A further set of hierarchical regression analyses was performed to assess the second hypothesis that variance accounted for in dependent measures by pain-related anxiety (PASS-20) would be held largely in common with AS (ASI-3). The first of these

analyses used the same dependent measure (PI-PA) as in the initial analyses of PASS-20 scores as a predictor of post-task variable scores. ASI-3 scores were entered on the first step followed by the PASS-20 scores on the second step. The first step entering the ASI-3 failed to result in a significant model, $F(1, 59) = 2.95$, $p = .091$, adjusted $R^2 = .03$. Adding the PASS-20 on the second step resulted in a significant model, $F(2, 58) = 4.93$, $p = .011$, substantially increasing the variance accounted for, $\Delta R^2 = .10$. The PASS-20 was thus found to account for 10% of the variance in PI-PA scores whereas the ASI-3 was not found to be a significant predictor. Contrary to hypothesis 2, these results indicate that, for the current data, PASS-20 scores do not share significant variance with the ASI-3. These findings are consistent with the results of the correlational analyses, wherein no significant relationships were observed between the ASI-3 and dependent measures.

Similar analyses were performed to assess the unique and common variance accounted for by the ASI-3 and the BFNE-S in dependent measures significantly correlated with the BFNE (i.e., PI-PA, MA-SA). The first model (identical to the analyses above with the PASS-20) entering only the ASI-3 did not significantly predict variance in PI-PA scores, $F(1, 59) = 2.95$, $p = .091$, adjusted $R^2 = .03$. The addition of the BFNE-S on the second step also did not result in a statistically significant model, $F(2, 58) = 2.14$, $p = .127$. As with the analyses for the PASS-20, these results indicated that the BFNE-S was not a statistically significantly predictor of variance in PI-PA scores.

The final set of analyses evaluated the shared and common variance accounted for by the ASI-3 and BFNE-S in MA-SA scores. The first model entering only the ASI-3 did not result in a significant model, $F(1, 59) = 1.40$, $p = .241$, adjusted $R^2 = .00$. Adding the BFNE-S in the second step resulted in a significant model, $F(2, 58) = 4.18$, $p = .020$, and

substantially increased the variance accounted for, $\Delta R^2 = .10$. Thus, the BFNE-S accounted for 10% of the variance in SA-MA scores. Consistent with findings described above, the ASI-3 was not a significant predictor of dependent measures.

Plausible reasons for these generally null findings will be considered in more depth in the discussion to follow. In an attempt to conduct a finer grained analysis, correlations between PASS-20 and ASI-3 subscale scores and all dependent measures were examined. Of interest was the possibility that factorially distinct aspects of these constructs (represented by the subscales) may have been positively correlated with the dependent measures but overlooked due to aggregation of total scale scores. Several statistically significant correlations between PASS-20 subscale scores (i.e., cognitive, escape/avoidance, fear, physiological subscales), ASI-3 subscale scores, and dependent measures were found (Table 6). Due to the numerous relationships examined only those found to be statistically significant are reported and discussed.

Several small to medium sized correlations (Cohen, 1988) were found between PASS-20 subscale scores and dependent measures for the pain induction condition; however, this is an unremarkable finding as PASS-20 total scores had already been found to be significantly positively correlated with the PI-PA checklist. Somewhat intriguing was the small association found between the PASS-20 physiological subscale and the pain induction task respiration change-score. This result was consistent with the observation that many participants slowed or held their breath during the pain tolerance task such that respiration rates were lower for the tolerance task, albeit not statistically significantly (see Table 5 for pre- and post-task values). Regarding the ASI-3 subscales, two small correlations were identified between the social concerns and physiological

concerns subscales and the pain induction task dependent variables of current pain and post-task pain anxiety (PA-PI), respectively. Although the reasons for these observed relationships are unclear, there are possible explanations. Regarding the association between ASI-3 social concerns and PI pain scores, it may be that elevated concerns about the social consequences of observable anxiety symptoms motivated participants to report higher levels of pain on the pain induction task, perhaps as a way to attribute their observable anxiety to the experimentally induced pain. While speculative, this suggestion may be an avenue for empirical investigation. Concerning the association between ASI-3 physiological concerns and post-task pain anxiety scores, both constructs reflect concerns about physical sensations and it is, thus, unsurprising that a significant correlation was observed.

Table 6. PASS-20/ASI-3 subscale correlations with dependent measures

Relationship examined	*r*	*p*	r^2
PASS-20 cog / PI pain	.331	.009	.110
PASS-20 cog / PI-PA	.391	.002	.153
PASS-20 cog / PI-NA	.347	.006	.120
PASS-20 esc-av / PI-PA	.326	.010	.106
PASS-20 fear / PI-PA	.264	.040	.070
PASS-20 phys / PI-PA	.321	.012	.103
PASS-20 phys/ PI resp chg	.273	.033	.075
ASI-3 soc / PI pain	.264	.040	.070
ASI-3 phys / PI-PA	.277	.031	.078

Note. Only statistically significant correlations reported. PASS-20 cog = cognitive subscale; PASS-20 esc-av = escape/avoidance subscale; PASS-20 phys = physiological subscale; ASI-3 soc = social concerns subscale; ASI-3 phys = physiological concerns subscale; PI pain = subjective pain rating post pain induction task; PI-PA = pain induction post-task pain anxiety; PI RESP = respiration rate residualized change for pain induction task

4. DISCUSSION

The current investigation sought to extend the findings of Greenberg and Burns (2003) using state-of-the-art pain-induction methods and biophysiological data acquisition with a non-clinical analogue sample. The objective of this study was to assess whether pain-related anxiety may, for a non-clinical sample, be better understood as a distinct pain-related phobia or, rather, as a manifestation of AS. These theoretical perspectives hold differing implications for the conceptualization, assessment, and treatment of chronic musculoskeletal pain. If pain-related anxiety is better understood as a distinct pain-related phobia then, analogous to evidence-based treatment for specific phobias (e.g., Grös & Antony, 2006), intervention should include *in vivo* exposure to the feared object (i.e., pain). Assessment procedures would identify the cognitive (e.g., pain-related catastrophic thoughts, attentional biases), behavioural (e.g., specific avoided activities), and physiological (e.g., anxious arousal) dimensions of the pain phobia such that these can be addressed in exposure-based cognitive-behavioural treatment. Alternatively, if pain-related anxiety is better viewed as a manifestation of AS it will then be important to routinely evaluate AS as part of assessment procedures. Treatment protocols for highly pain-anxious/anxiety sensitive patients would then appropriately include interventions such as interoceptive exposure that specifically target AS (e.g., Watt et al., 2006). It was with these theoretical perspectives in mind that the current investigation was undertaken.

Two hypotheses were tested in this investigation. First, it was predicted that a measure of pain-related anxiety would, in regression models, significantly and substantively account for variance in dependent measures representing generally fearful

responses during *both* pain-anxiety and social-evaluative anxiety experimental induction tasks. This hypothesis was consistent with the view that pain-related anxiety may be a manifestation of AS, a construct predictive of fearful responding to the physical sensations of anxiety. Second, to assess whether pain-related anxiety may arise from AS, it was further hypothesized that variance in dependent measures accounted for by pain-related anxiety scores (PASS-20) would, in regression models, be explained by scores on a measure of AS (ASI-3).

For the first hypothesis, the results of correlation and hierarchical regression analyses indicated that pain-related anxiety was predictive of positive variance only for the pain-induction post-task measure of pain anxiety (PI-PA). Contrary to prediction, the PASS-20 did not significantly account for variance in any of the mental arithmetic task dependent measures. For the second hypothesis, despite exhibiting a high degree of correlation with the PASS-20, the ASI-3 did not account for significant variance in either the pain induction or mental arithmetic post-task dependent measures. These results failed to reject the null hypothesis for either of the two main hypotheses.

Before discussing the current results some consideration of the importance of replication and null findings to the broader scientific enterprise is warranted. Replication stands as a foundational principle of science and it is crucial that reported findings be tested via independent replication. Similarly, null or so-called negative findings are also important in that these results serve to moderate conclusions and refine research directions. The discipline of Psychology has been criticized for widespread under-reporting of both replication studies and null findings (Laws, 2013). Indeed, a pervasive bias against the publication of so called negative findings has been well documented in

Psychology and the other social sciences (e.g., Ferguson & Heene, 2012). A further bias exists against publication of replications, with some journals reportedly refusing to consider reports of such investigations, favouring instead novel findings (Nueliep, & Crandall, 1993). These biases do a disservice to scientific inquiry.

The current investigation might be viewed as a partial replication in that the approach taken was generally similar to that of Greenberg and Burns (2003), with the differences lying mainly in methodological refinements and the nature of the sample. The current results, although unsupportive of the stated hypotheses, nonetheless provide potentially important information. Although the interpretation of null findings is challenging, the results do suggest future research avenues which will be considered below. We now turn to the discussion of the findings.

Although the results did not support the hypotheses, there were significant findings that bear consideration. First, pain-related anxiety as measured by the PASS-20 was found to predict positive variance in the pain induction post-task measure of pain anxiety. On first examination this finding may seem unsurprising in that a trait measure of pain-anxiety was essentially predicting a state measure of pain-anxiety but this result can also be interpreted as providing support for the predictive validity of the PASS-20. Similarly, the BFNE-S, which assesses the *fear of negative evaluation*, was found to predict positive variance in mental arithmetic post-task social evaluative anxiety scores. Again, this is a finding that might be viewed unsurprising, but as with the PASS-20 the results support the predictive validity of the BFNE-S. These results should be tempered by consideration that both the trait measures (PASS-20, BFNE-S) and the post-task dependent measures were comprised of items with Likert scale response options and the

results may have been influenced to some degree by common method effects (e.g., Podsakoff, MacKenzie, Lee, & Podsakoff, 2003).

Considering the overall objectives of the investigation, the results did not support an AS conceptualization of pain-related anxiety such as was found by Greenberg and Burns (2003). Although the reasons for the mainly null findings are unclear, several possibilities will be examined. The first centres on the question of whether the study design and sample provided adequate statistical power. A number of observations suggest that a lack of statistical power does not fully explain the results. First, statistically significant positive correlations among trait measures (i.e., PASS-20, ASI-3, BFNE-S, STAI-T, CES-D) were observed in the current data. Moreover, the magnitude of these correlations was in a range consistent with those reported in other studies using non-clinical samples (e.g., Carleton et al., 2009; Muris, Vlaeyen et al., 2001). Similar studies employing clinical samples have generally, but not uniformly, reported lower correlations, as might be expected with restricted range samples (Urbina, 2004). A further indication that the null findings may not be attributable to a lack of power derives from examination of the correlations between trait variables of interest and dependent measures for each of the experimental tasks. Few of these correlations were found to be trending towards statistical significance. To illustrate, PASS-20 correlations with dependent measures were statistically significant only for the pain-induction post-task pain anxiety variable. Two other PASS-20 total score correlations may have approached statistical significance with a larger sample; however, these associations were, again, confined to pain induction task dependent variables (i.e., post-task reported pain (p = .073) and respiration rate standardized change score (p = .090). None of the correlations

between the PASS-20 total scores and mental arithmetic task dependent variables were observed to be trending toward statistical significance (all *p*s > .26). Considering that it was expected that all correlations examined would be positive, the correlational analyses were re-computed as one-tailed tests. The results of these procedures remained consistent with those found for the two-tailed tests; that is, the PASS-20 total scores remained significantly correlated with only the pain induction post-task pain anxiety measure. Finally, the observed power of the hierarchical multiple regression analyses was computed using a freely available web-based post-hoc statistical power calculator (i.e., http://www.danielsoper.com/statcalc3/calc.aspx?id=17). Using an estimated medium effect size (i.e., $f^2 = .15$) adequate observed power of greater than .80 was found for all hierarchical regression analyses. Taken together, these considerations suggest that insufficient statistical power does not fully explain the null findings.

A second factor that may have affected the current results relates to the role that selection biases may have played in significantly influencing the composition of the sample. Specifically, consent procedures required that potential participants be informed that they would be undergoing experimental pain induction, the knowledge of which plausibly affected their decision regarding whether to take part. It is reasonable to consider that those who may have been averse to undergoing pain induction procedures would simply have chosen to not participate, thereby limiting access to a fuller range of participants. A further selection bias may be one associated with convenience. Almost half of participants (45.9%) who completed the study were students at the University where the research was conducted, a factor that likely facilitated their participation. In addition to the convenience associated with proximity, students also comprise a group

who may arguably have been interested in research (the sample was highly educated), and may have been motivated to receive the compensation of a $20.00 Tim Hortons gift card. Selection bias may additionally have occurred consequent to providing participants with information explaining that they would be asked to perform a mental arithmetic task. Similar to considering the prospect of undergoing pain induction, it may be that some individuals viewed the mental arithmetic task as unpleasant and thus elected to not participate. In considering the preceding discussion of the characteristics of the current sample it becomes apparent that, in our attempt to recruit a non-restricted range sample, we likely obtained a different kind of restricted range sample – one that plausibly limited the participation of a fuller range of participants.

Methodological considerations represent a third potential explanation for the null results. One possibility is that the experimental tasks employed in the current investigation were not sufficiently anxiety provoking. The mainly null results from analyses comparing pre- and post-task dependent measure mean scores support this suggestion. Post-task pain anxiety measures were positively skewed, indicating that scores tended to cluster at the lower end of the possible range. These results suggest that the pain induction task may have been only partially successful in inducing significant pain-related anxiety. No similar effect was observed for the mental arithmetic task, for which post-task scores on measures of social-evaluative anxiety and negative affectivity reflected a fuller reported range of task-relevant anxiety. Although speculative, it may be that the inclusion of warmth detection and pain threshold testing in the pain induction protocol had the effect of acclimating the participant to the task (i.e., to the thermal stimulation) and thereby reduced their anxiety as the task proceeded. Conversation with

several of the study participants supports this notion. During debriefing procedures several participants reported that the mental arithmetic task was significantly more anxiety provoking than the pain-induction task. A better approach may have been to forego the data provided by the warmth detection and pain threshold testing and, analogous to the cold pressor task used by Greenberg and Burns (2003), present only the more demanding task of pain tolerance testing.

Finally, in considering the current findings, the possibility that the hypothesized effects were simply not present also warrants examination. It may be that the pattern of results reported by Greenberg and Burns (2003) does not similarly manifest in high functioning individuals not experiencing significant current pain. So how do clinical pain samples differ from non-clinical samples? Relative to normative samples, samples of persons with chronic pain evidence significantly elevated scores on measures of AS (e.g., Asmundson & G. R. Norton, 1995; Greenberg & Burns, 2003), pain-related anxiety (e.g., Abrams et al., 2007; McCracken & Dhingra, 2002; McCracken et al., 1992), and pain catastrophizing (e.g., Sullivan et al., 1998). Moreover, persons with chronic pain also frequently present with clinically significant psychopathology, particularly depressive (e.g., Breivik et al., 2006; Currie & Wang, 2004; Holmes, Christelis & Arnold, 2012), anxiety (e.g., McWilliams et al., 2003; McWilliams et al., 2004; Von Korff et al., 2005), and trauma-related disorders (e.g., Demyttenaere et al., 2007). Meta-analytic research has also demonstrated that persons with chronic pain exhibit significantly greater attentional biases toward pain-related information than healthy control groups (Schoth, Nunes, & Liossi, 2012). Collectively, these findings indicate that persons with chronic pain differ substantially from those without.

The differing theoretical perspectives of pain-related anxiety as a specific phobia versus pain-related anxiety as a manifestation of AS invite further interpretation of the current results. In a specific phobia understanding of pain-related anxiety persons are believed to fear pain-related objects including continued or worsening pain, movement, and re-injury. Exposure to these pain-related objects should provoke fear responses such as ANS arousal and escape/avoidance behaviours. Given that the present sample, by design, did not report significant current or chronic pain it was, perhaps, unsurprising that AS was not found to be positively associated with any of the dependent measures. Rather, the only positive associations found were for task-relevant measures of pain-anxiety and social-evaluative anxiety, results that suggested the effects were confined to specific task contexts instead of attributable to the global construct of AS.

Alternatively, an AS conceptualization posits that pain-related anxiety arises out of the dispositional tendency to fear the physical sensations of anxious arousal due to the belief that such sensations signal imminent catastrophic consequences. That AS was not found to be positively associated with any of the dependent measures in the current study may suggest that for persons not experiencing current or chronic pain AS exerts no influence on pain-related anxiety. It may instead be the case that pain-related anxiety manifests from AS resultant to a current, or perhaps historical, persistent pain experience. Given that there is evidence to suggest that elevated levels of AS may arise from learning to catastrophically interpret bodily sensations in general rather than anxiety symptoms in particular (Watt et al., 1998), it may be that a persistent pain experience contributes to the development of the relationship between AS and pain-related anxiety that has so often been documented in samples of chronic pain patients. Indeed, other researchers have

highlighted the need for longitudinal studies to examine whether AS precedes the development of chronic musculoskeletal pain or becomes elevated as a result of it (Asmundson & Katz, 2009).

Individuals with chronic pain have been well-characterized in the research to date; however, our understanding of the pathways leading from acute to chronic pain remains incomplete. There are currently several lines of research related to this important direction. One intriguing avenue is the suggestion by Kleiman and colleagues (Kleiman, Clarke, & Katz, 2011) that pain-related anxiety constructs, including pain-related anxiety and AS, may derive from an underlying, higher order, fundamental fear. Investigating a sample of patients scheduled for major surgery, the researchers employed factor analytic methods to assess the latent structure of pooled items from three commonly used measures of pain-anxiety related constructs, the PASS-20, the ASI, and the Pain Catastrophizing Scale (PCS; Sullivan, Bishop, & Pivik, 1995). They found that twenty items loaded exclusively on one higher order factor they termed *sensitivity to pain traumatization* (SPT). The authors characterized SPT as the propensity to develop anxiety-related somatic, cognitive, emotional, and behavioural responses to pain that bore resemblance to features of a traumatic stress reaction. Notably, the researchers gathered pain histories from participants and conducted follow-up reviews at one year post surgery. They found that SPT scores were significantly higher for participants who reported a history of pain than those who did not, both before surgery and one year post surgery. Although these results have, to our knowledge, not been replicated, the notion of a construct that may subsume the various pain-anxiety related constructs into a coherent fundamental fear is an intriguing development. The authors suggested that SPT

is likely a dimensional construct but this has yet to be empirically tested. In considering the current results in light of these findings, it is plausible that current sample participants, who were reporting neither significant pain nor facing the prospect of major surgery, would likely have reported low SPT scores. We did not gather pain histories from participants, a refinement that may have strengthened the methodology.

This study had several limitations that suggest future research directions. The current results did not support a conceptualization of pain-related anxiety as a manifestation of AS in a sample of persons not reporting current pain. Although the reasons for the current findings are unclear, the results may inform the continuing study of non-clinical samples. First, a future approach may be to conduct similar investigations with samples of healthy individuals not reporting significant pain but who have elevated AS and/or pain-related anxiety. Narrowing the focus to persons with elevated AS and pain-related anxiety may facilitate better understanding of relationships among the constructs of interest. A second approach may be to employ recently developed bootstrapping mediation analyses (e.g., Preacher & Hayes, 2008; Zhao, Lynch, & Chen, 2010) to assess the specific influences of constructs of interest as they relate to chronic pain outcomes. Third, it may be advantageous to conduct focused single case studies of injured persons, following them from the acute phase of injury through to the completion of healing and resumption of normal activities. Such an investigation might proceed by meeting individually with injured participants soon after they are medically stabilized to gather a variety of data including: (a) clinical histories (including pain histories); (b) current psychological status; and (c) measurement of pain-related anxiety, AS, and related constructs. Periodic review would then follow at intervals to assess participant

recovery as they progress through the rehabilitation process. There may exist opportunities to recruit the assistance of third-party-payer (e.g., insurance company) case managers in a so-designed investigation as these organizations have a financial interest in good outcomes for insured clients. Fourth, there is a compelling need for longitudinal research designed to more clearly delineate the pathways from an acute injury to pain chronicity. A naturalistic opportunity to examine these pathways is afforded by organizations that routinely perform medical and psychological assessment of individuals as part of intake procedures. Some candidate groups for such an approach include the military, police agencies, and Health Maintenance Organizations. These agencies commonly undertake the comprehensive evaluation of persons joining them, a process that could include administration of measures assessing constructs posited important to the development and maintenance of chronic musculoskeletal pain. Participants would then be followed over time and when some inevitably sustain injury they could be closely monitored to characterize the relationships among relevant constructs and rehabilitation outcomes. Finally, surgical patients provide yet another naturalistic group to evaluate and follow as they progress from the pre-operative period through surgery and recovery periods. This is a research area that has garnered considerable attention to date (for a review see Katz & Seltzer, 2009). Psychological and social-environmental variables have consistently been associated with the development of chronic post-surgical pain however the nature of these relationships remains unclear (Katz & Seltzer, 2009) and requires further investigation.

To conclude, despite the challenges in interpreting the current mainly null findings, it seems plausible that our attempt to recruit a non-clinical sample reporting no

significant pain resulted in a restricted range sample that may have represented the polar opposite to the chronic low-back pain sample of the Greenberg and Burns (2003) study; that is, the current sample may have been insufficiently pain anxious or anxiety sensitive to exhibit a pattern of results similar to that reported by Greenberg and Burns (2003). The current findings suggest that high-functioning persons not experiencing significant pain simply do not evidence the interrelationships among AS and pain-related anxiety observed in persons with chronic pain. It may be that the robust relationship observed between AS and pain-related anxiety is, at least in part, a consequence of a persistent pain experience; however, this relationship awaits empirical examination.

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6. APPENDICES

Appendix I

Anxiety Sensitivity Index-3

Anxiety Sensitivity Index-3 (ASI-3)

Please circle the number that best corresponds to how much you agree with each item. If any items concern something that you have never experienced (e.g., fainting in public), then answer on the basis of how you think you might feel *if you had* such an experience. Otherwise, answer all items on the basis of your own experience. Be careful to circle only one number for each item and please answer all items.

	Very little	**A little**	**Some**	**Much**	**Very much**
1. It is important for me not to appear nervous.	0	1	2	3	4
2. When I cannot keep my mind on a task, I worry that I might be going crazy.	0	1	2	3	4
3. It scares me when my heart beats rapidly.	0	1	2	3	4
4. When my stomach is upset, I worry that I might be seriously ill.	0	1	2	3	4
5. It scares me when I am unable to keep my mind on a task.	0	1	2	3	4
6. When I tremble in the presence of others, I fear what people might think of me.	0	1	2	3	4
7. When my chest feels tight, I get scared that I won't be able to breathe properly.	0	1	2	3	4
8. When I feel pain in my chest, I worry that I'm going to have a heart attack.	0	1	2	3	4
9. I worry that other people will notice my anxiety.	0	1	2	3	4
10. When I feel "spacey" or spaced out I worry that I may be mentally ill.	0	1	2	3	4
11. It scares me when I blush in front of people.	0	1	2	3	4
12. When I notice my heart skipping a beat, I worry that there is something seriously wrong with me.	0	1	2	3	4
13. When I begin to sweat in a social situation, I fear people will think negatively of me.	0	1	2	3	4
14. When my thoughts seem to speed up, I worry that I might be going crazy.	0	1	2	3	4
15. When my throat feels tight, I worry that I could choke to death.	0	1	2	3	4
16. When I have trouble thinking clearly, I worry that there is something wrong with me.	0	1	2	3	4
17. I think it would be horrible for me to faint in public.	0	1	2	3	4
18. When my mind goes blank, I worry there is something terribly wrong with me.	0	1	2	3	4

Scoring: Physical concerns = sum of items 3, 4, 7, 8, 12, 15; Cognitive concerns = sum of items 2, 5, 10, 14, 16, 18; Social concerns = sum of items 1, 6, 9, 11, 13, 17

Appendix II

Brief Fear of Negative Evaluation-Straightforward Items

Brief Fear of Negative Evaluation-Straightforward Items (BFNE-S)

(Carleton, Collimore, McCabe, & Antony, 2011)

Please circle the number that best corresponds to how much you agree with each item

	Not at all characteristic of me	A little characteristic of me	Somewhat characteristic of me	Very characteristic of me	Entirely characteristic of me
1. I worry about what other people will think of me even when I know it doesn't make any difference.	1	2	3	4	5
2. I am frequently afraid of other people noticing my shortcomings.	1	2	3	4	5
3. I am afraid that others will not approve of me.	1	2	3	4	5
4. I am afraid that other people will find fault with me.	1	2	3	4	5
5. When I am talking to someone, I worry about what they may be thinking about me.	1	2	3	4	5
6. I am usually worried about what kind of impression I make.	1	2	3	4	5
7. Sometimes I think I am too concerned with what other people think of me.	1	2	3	4	5
8. I often worry that I will say or do wrong things.	1	2	3	4	5

Appendix III

Center for Epidemiological Studies-Depression Scale

Center for Epidemiological Studies-Depression Scale (CES-D)

For each statement, please circle the number in the column that best describes how you have been feeling ***in the past week***.

	Rarely or none of the time (less than 1 day)	Some or a little of the time (1-2 days)	Occasionally or a moderate amount of the time (3-4 days)	Most or all of the time (5-7 days)
1. I was bothered by things that usually don't bother me.	0	1	2	3
2. I did not feel like eating; my appetite was poor.	0	1	2	3
3. I felt that I could not shake off the blues, even with the help from family or friends.	0	1	2	3
4. I felt that I was just as good as other people.	0	1	2	3
5. I had trouble keeping my mind on what I was doing.	0	1	2	3
6. I felt depressed.	0	1	2	3
7. I felt that everything I did was an effort.	0	1	2	3
8. I felt hopeful about the future.	0	1	2	3
9. I thought my life had been a failure.	0	1	2	3
10. I felt fearful.	0	1	2	3
11. My sleep was restless.	0	1	2	3
12. I was happy.	0	1	2	3
13. I talked less than usual.	0	1	2	3
14. I felt lonely.	0	1	2	3
15. People were unfriendly.	0	1	2	3
16. I enjoyed life.	0	1	2	3
17. I had crying spells.	0	1	2	3
18. I felt sad.	0	1	2	3
19. I felt that people dislike me.	0	1	2	3
20. I could not get "going".	0	1	2	3

Appendix IV

Pain Anxiety Symptoms Scale-20

Pain Anxiety Symptoms Scale-20 (PASS-20)

(McCracken & Dhingra, 2002)

Please use the following scale to rate how often you engage in each of the following thoughts or activities. Circle the number beside the statement to indicate your rating

	Never					Always
1. I can't think straight when in pain	0	1	2	3	4	5
2. During painful episodes it is difficult for me to think of anything besides the pain	0	1	2	3	4	5
3. When I hurt I think about pain constantly	0	1	2	3	4	5
4. I find it hard to concentrate when I hurt	0	1	2	3	4	5
5. I worry when I am in pain	0	1	2	3	4	5
6. I go immediately to bed when I feel severe pain	0	1	2	3	4	5
7. I will stop any activity as soon as I sense pain coming on	0	1	2	3	4	5
8. As soon as pain comes on I take medication to reduce it	0	1	2	3	4	5
9. I avoid important activities when I hurt	0	1	2	3	4	5
10. I try to avoid activities that cause pain	0	1	2	3	4	5
11. I think that if my pain gets too severe it will never decrease	0	1	2	3	4	5
12. When I feel pain I am afraid that something terrible will happen	0	1	2	3	4	5
13. When I feel pain I think I might be seriously ill	0	1	2	3	4	5
14. Pain sensations are terrifying	0	1	2	3	4	5
15. When pain comes on strong I think that I might become paralysed or more disabled	0	1	2	3	4	5
16. I begin trembling when engaged in an activity that causes pain	0	1	2	3	4	5
17. Pain seems to cause my heart to pound or race	0	1	2	3	4	5
18. When I sense pain I feel dizzy or faint	0	1	2	3	4	5
19. Pain makes me nauseous	0	1	2	3	4	5
20. I find it difficult to calm my body down after periods of pain	0	1	2	3	4	5

Appendix V

Pain-Affectivity Checklist (Mental Arithmetic task)

Pain-affectivity checklist (Mental Arithmetic task)

1. On the scale below please circle the number that reflects how much pain you have right now.

1	2	3	4	5	6	7	8	9	10

No pain at all — The worst imaginable pain

2. On the scale below please circle the number that reflects how anxious you feel right now.

1	2	3	4	5	6	7	8	9	10

Not anxious at all — Extremely anxious

3. On the scale below please circle the number that reflects how irritated you feel right now.

1	2	3	4	5	6	7	8	9	10

Not irritated at all — Extremely irritated

4. On the scale below please circle the number that reflects how tense you feel right now.

1	2	3	4	5	6	7	8	9	10

Not tense at all — Extremely tense

5. On the scale below please circle the number that reflects how nervous you feel right now.

1	2	3	4	5	6	7	8	9	10

Not nervous at all — Extremely nervous

6. On the scale below please circle the number that reflects how concerned you were about making a good impression.

1	2	3	4	5	6	7	8	9	10

Not concerned at all — Extremely concerned

7. On the scale below please circle the number that reflects how bothered you were about being judged on your performance.

1	2	3	4	5	6	7	8	9	10

Not bothered at all — Extremely bothered

8. On the scale below please circle the number that reflects how worried you were that you would do poorly on this task.

1	2	3	4	5	6	7	8	9	10

Not worried at all — Extremely worried

9. On the scale below please circle the number that reflects how afraid you were that you would embarrass yourself.

1	2	3	4	5	6	7	8	9	10

Not at all afraid — Extremely afraid

Appendix VI

Pain-Affectivity Checklist (Pain Induction)

Pain-affectivity checklist (Pain Induction)

1. On the scale below please circle the number that reflects how much pain you have right now.

1	2	3	4	5	6	7	8	9	10

No pain at all — The worst imaginable pain

2. On the scale below please circle the number that reflects how anxious you feel right now.

1	2	3	4	5	6	7	8	9	10

Not anxious at all — Extremely anxious

3. On the scale below please circle the number that reflects how irritated you feel right now.

1	2	3	4	5	6	7	8	9	10

Not irritated at all — Extremely irritated

4. On the scale below please circle the number that reflects how tense you feel right now.

1	2	3	4	5	6	7	8	9	10

Not tense at all — Extremely tense

5. On the scale below please circle the number that reflects how nervous you feel right now.

1	2	3	4	5	6	7	8	9	10

Not nervous at all — Extremely nervous

6. On the scale below please circle the number that reflects the degree to which you were distressed by the pain.

1	2	3	4	5	6	7	8	9	10

Not distressed at all — Extremely distressed

7. On the scale below please circle the number that reflects the degree to which you were afraid of being hurt by doing this task.

1	2	3	4	5	6	7	8	9	10

Not afraid at all — Extremely afraid

8. On the scale below please circle the number that reflects the degree to which you were scared your pain would increase.

1	2	3	4	5	6	7	8	9	10

Not scared at all — Extremely scared

9. On the scale below please circle the number that reflects the degree to which you were preoccupied with the pain.

1	2	3	4	5	6	7	8	9	10

Not at all preoccupied — Extremely preoccupied

Appendix VII

Research Ethics Approval

University of Regina

OFFICE OF RESEARCH SERVICES

Regina, Saskatchewan, Canada S4S 0A2
Phone: 306.585.4775 Fax: 306.585.4893
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DATE: December 9, 2011

TO: Murray Abrams
3859 Montague Street
Regina, SK S4S 3J6

FROM: Dr. Bruce Plouffe
Chair, Research Ethics Board

Re: **Clarifying the Nature of Pain-Related Anxiety: Implications for Assessment and Treatment of Chronic Musculoskeletal Pain (File # 32S1112)**

Please be advised that the University of Regina Research Ethics Board has reviewed your proposal and found it to be:

☑ 1. APPROVED AS SUBMITTED. Only applicants with this designation have ethical approval to proceed with their research as described in their applications. For research lasting more than one year (Section 1F). **ETHICAL APPROVAL MUST BE RENEWED BY SUBMITTING A BRIEF STATUS REPORT EVERY TWELVE MONTHS.** Approval will be revoked unless a satisfactory status report is received. Any substantive changes in methodology or instrumentation must also be approved prior to their implementation.

☐ 2. ACCEPTABLE SUBJECT TO MINOR CHANGES AND PRECAUTIONS (SEE ATTACHED). Changes must be submitted to the REB and approved prior to beginning research. Please submit a supplementary memo addressing the concerns to the Chair of the REB.** Do not submit a new application. Once changes are deemed acceptable, ethical approval will be granted.

☐ 3. ACCEPTABLE SUBJECT TO CHANGES AND PRECAUTIONS (SEE ATTACHED). Changes must be submitted to the REB and approved prior to beginning research. Please submit a supplementary memo addressing the concerns to the Chair of the REB.** Do not submit a new application. Once changes are deemed acceptable, ethical approval will be granted.

☐ 4. UNACCEPTABLE AS SUBMITTED. The proposal requires substantial additions or redesign. Please contact the Chair of the REB for advice on how the project proposal might be revised.

D. Bruce Plouffe
Dr. Bruce Plouffe

cc: Dr. Gordon J. G. Asmundson - Psychology

** supplementary memo should be forwarded to the Chair of the Research Ethics Board at the Office of Research Services (Research and Innovation Centre, Room 109) or by e-mail to research.ethics@uregina.ca

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