SMOKING TO COPE WITH PAIN: THE MOTIVATING EFFECTS OF PAIN

INDUCTION ON SMOKING URGE AND BEHAVIOUR

A Thesis

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Hollyanne Ellen Ruth Parkerson, candidate for the degree of Master of Arts in Psychology, has presented a thesis titled, *Smoking to Cope with Pain: The Motivating Effects of Pain Induction on Smoking Urge and Behaviour*, in an oral examination held on July 31, 2013. The following committee members have found the thesis acceptable in form and content, and that the candidate demonstrated satisfactory knowledge of the subject material.

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Committee Member: Dr. Heather Hadjistavropoulos, Department of Psychology

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*via teleconference
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ABSTRACT

Millions of Canadians live with chronic pain. Substantial evidence has linked tobacco smoking with both acute and chronic pain conditions. Contemporary models of pain and smoking posit a cyclical relationship wherein individuals smoke to reduce pain in the short term; however, smoking to cope with pain indirectly exacerbates pain in the long term. Recent findings suggest situational pain is sufficient to increase smoking urge and immediate smoking behaviour (Ditre & Brandon, 2008). The purpose of the current investigation is to replicate and extend the findings of Ditre and Brandon (2008) by investigating the effects of (a) situational pain on smoking urge and behaviour using a reliable pain induction technique (electrical stimulation) and an alternate thermal mode (heat), and (b) anxiety vulnerabilities and smoking expectancies that may be contributing to increased tobacco dependence in a university and community sample (n=34; 55.9% male). Participants were randomly assigned to a heat pain or no pain control condition. Pre-manipulation group differences occurred in the Anxiety Sensitivity Index-3 (ASI-3) social subscale. A series of 2 x 2 ANCOVAs were conducted using pain (heat pain, no pain) and sex (female, male) as the two independent variables, self-reported urge to smoke and immediate smoking behaviour as the dependent variables, and the ASI-3 social subscale as the covariate. Participants in the heat pain group reported significantly greater smoking urge and more immediate smoking behaviour than those in the no pain group. Individuals with higher levels of ASI-3 social reported greater smoking urge and more immediate smoking behaviour. Further, results of a chi-square difference test indicated significantly more individuals in the heat pain group attempted to smoke (100%) after the pain manipulation than those in the control group (62.5%). Structural equation modeling was conducted to assess direct and indirect relationships of the
aforementioned variables (i.e., smoking urge, immediate smoking behaviour, ASI-3 social) along with smoking expectancies related to craving/addiction. The model demonstrated excellent fit to the data. Results of the current investigation provide support for the findings of Ditre and Brandon (2008), indicating that situational pain is sufficient to increase both smoking urge and immediate smoking behaviour. Current results also indicate a role of the ASI-3 social subscale as well as smoking expectancies related to craving/addiction in the reciprocal relationship between pain and smoking. Findings suggest the necessity for comprehensive assessment of pain-related vulnerabilities and smoking expectancies that may lead to increased nicotine dependence in individuals seeking treatment for injury as well as chronic pain.
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DEDICATION

I dedicate this project to my husband, Micah Parkerson, and to my parents, James and Teresa Hominuke.
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1.0 Introduction

1.1 Overview

Tobacco smoking has long been associated with numerous health conditions including, but not limited to, cancer and cardiopulmonary disease (U.S. Department of Health and Human Services, 1999). More recently, smoking has been implicated in the development and aggravation of both acute and chronic pain (John, Hanke, Meyer, Völzke, Baumeister, & Alte, 2006) and the literature has made a convincing case implicating tobacco smoking in the development and maintenance of specific pain conditions such as chronic back pain. Almost 3 million Canadians live with chronic pain (Statistics Canada, 2009); yet, a recent survey of the Canadian population indicated that the prevalence of back pain is significantly higher for smokers (23%) than for non-smokers (16%), with former smokers reporting higher prevalence of chronic pain compared to individuals who have never smoked (Alkhelayf, Wai, Tsai, & Agbi, 2010). The youth population is at greatest risk; smokers between ages 20 and 29 are 80% more likely to have chronic low back pain than non-smokers in the same age range (Alkhelayf et al., 2010). Although the health risks associated with smoking are now commonly recognized, millions of Canadians continue to smoke. The current investigation sought to further delineate the relationship between pain and smoking.

This thesis is structured as follows. First, theoretical and empirical literature about nicotine dependence will be reviewed. Thereafter, the theoretical and empirical literature about pain as well as predispositional and pain-related anxiety constructs will be described and discussed in terms of their effect on pain, smoking urge, and smoking behaviours. Finally, the purposes and hypotheses, methodology, and results of the current investigation will be outlined, as well as the relevance of the findings.
1.2 Nicotine Dependence

1.2.1 How nicotine acts. Among the thousands of chemicals found in tobacco (e.g., ammonia, acetone, methanol, phelol, hydrogen cyanide), nicotine is the most active in the central nervous system (Stolerman & Jarvis 1995; Balfour 2004). As nicotine enters the body, it quickly passes to the blood stream through the lungs, the mucosal membrane of the mouth and nose, or directly through the skin. Nicotine reaches the central nervous system within 10 to 19 seconds (Benowitz, 1990), stimulating the nicotinic acetylcholine receptors (nAChRs) located throughout the brain (midbrain, tegmentum, striatum, and nucleus accumbens), muscles, adrenal glands, heart, and other organs (Benwell, Balfrm, & Khadra, 1994). Nicotine also stimulates the release of dopamine indirectly via stimulation of the nAChRs (Balfour, 2004). Stimulation of the nAChRs and dopamine receptors creates the subjective experience of relaxation and pleasure, respectively (Nestler, 1999). The sudden burst of nicotine also stimulates the adrenal system, releasing epinephrine and glucose, which results in a temporary increase in blood pressure, respiration, and heart rate. This process has been posited to enhance attention and alertness (Sarter, Bruno, & Givens, 2003; Sarter & Parikh, 2005). Nicotine levels quickly peak and decline as the substance becomes absorbed by peripheral tissues and subsequently eliminated by the body (Benowitz, Porchet, Sheiner, & Jacob, 1988).

1.2.2 Models of nicotine dependence. Several models have been proposed that aid in our understanding of nicotine dependence. Broadly speaking, these models fall within the biological, behavioural, and cognitive-social-learning domains (see Shadel, Shiffman, Niaura, Nichter, & Abrams, 2000). Each model addresses different features of dependence phenomena and together they provide a comprehensive understanding of the
complex interactions involved in nicotine dependence. A brief description of each model has been provided below to frame the rationale and purposes of the proposed investigation.

Biological models of nicotine dependence highlight the direct effects of nicotine on the nervous system (e.g., Clarke, 1990; Pomerleau, 1995, Frawley, 1998). For example, neuroadaptation models suggest that dependence occurs primarily at a neurocellular level, such that repeat presence of nicotine results in increased nicotine tolerance (e.g., desensitivity of nAChRs in the central nervous system and an increase in receptor sites for the substance to bind to; Jain & Mukherjee, 2003). As increased tolerance occurs, higher doses of nicotine are needed to achieve the desired subjective experience. Cessation of nicotine usage causes a withdrawal experience wherein the central nervous system overcompensates for a lack of nicotine at the neuronal level, the subjective experience of which is typically opposite of the effect provided by nicotine usage (e.g., irritability, anxiety, depression; Centers for Disease Control, 2010; Pecknold, 1993). As increased tolerance occurs, higher doses of nicotine are needed to avoid withdrawal symptoms. Biological models posit that tolerance and withdrawal symptoms reinforce smoking behaviour and play a large role in the development and maintenance of nicotine dependence – notions that have been empirically well-supported (see Watkins, Koob, & Markou, 2002).

Behavioural models have been applied to nicotine dependence by building on the foundation provided by biological models (see Shadel et al., 2000). These models acknowledge the pharmacological effects of nicotine but focus on the learning processes that contribute to the development and maintenance of nicotine dependence. For
example, classical conditioning models conceptualize smoking as the unconditioned stimulus, and the effects of nicotine (subjective and physiological) as the unconditioned response (Bevins & Palmatier, 2004). Any stimuli (e.g., situational cues, sensory aspects of smoking) repeatedly paired with nicotine usage become the conditioned stimuli. The conditioned stimuli, in turn, elicit a conditioned response such as increased urge to smoke or withdrawal symptoms (Childress, Ehrman, Rohsenow, Robbins, & O’Brien, 1992; Niaura, 2000; Berridge & Robinson, 2003). Therefore, any stimuli associated with smoking can serve as priming stimuli resulting in a strong appetitive drive to increase nicotine usage.

Positive and negative reinforcement models of nicotine dependence address nicotine dependence from an operant conditioning perspective. Such models posit that nicotine usage is positively reinforced by its involvement with the dopamine system, which is associated with pleasure and reward processes (e.g., the mesolimbic dopaminergic system; Balfour, 2004). Negative reinforcement in this context generally encompasses the processes by which smoking relieves or reduces withdrawal states, as well as negative affective states associated with withdrawal (Eissenberg, 2004). Due to the co-occurrence of withdrawal and negative affective states, a person may learn to associate any negative affective state with withdrawal regardless of whether it is related to withdrawal. As such, the use of smoking to relieve aversive withdrawal states may generalize to smoking to relieve aversive affect states in general (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004). The association between smoking and escape of both withdrawal and negative affective states has been posited as a key factor in maintaining nicotine dependence. Empirical evidence is generally supportive of tenets of
behaviour models in the development and maintenance of nicotine dependence (e.g., classical conditioning, see Field & Cox, 2008; positive and negative reinforcement, see Watkins et al., 2002).

Cognitive social learning models, as applied to nicotine dependence, focus on the role of the environment and an individual’s cognitive processes in the development of nicotine dependence (Abrams & Niaura, 1987; Marlatt & Gordon, 1985; Shadel et al., 2000; Tiffany, 1990). Such models highlight the involvement of observation and social environment in the development of smoking behaviour (Bandura, 1986). The dynamic effects of nicotine usage in different domains of life (e.g., social, cultural) are observed and create beliefs and expectancies about the substance (e.g., smoking relieves stress, reduces anxiety, makes me feel calm, helps produce weight loss). As such, personal experiences with nicotine confirm or disconfirm beliefs and expectancies, and also allow for new ones to immerge. Social learning models also posit that nicotine dependence occurs in both physical and psychological domains (Shadel & Mermelstein, 1993). For example, physical dependence occurs in part when an individual acts to avoid the experience of withdrawal by repeated use of nicotine, whereas psychological dependence occurs because of expectancies related to the positive benefits of nicotine use (e.g., smoking will relieve stress) and decreased self-efficacy or belief in one’s ability to manage or cope without the perceived effects of nicotine usage. Many of the key tenets of cognitive social learning models have received empirical support (e.g., expectancies, self-efficacy, stress coping; see Brandon, Herzog, Irvin, & Gwaltney, 2004); however, the models themselves continue to be influenced and refined by advancements in cognitive science (Brandon et al., 2004).
In summary, nicotine dependence can be explained using several conceptual perspectives, each of which has received support from empirical research (see Brandon et al., 2004; Field & Cox, 2008; Watkins, 2002). Consideration of the different perspectives provides a more comprehensive understanding of the complex interactions between the biological, psychological, behavioural, cognitive, and social mechanisms involved in nicotine dependence phenomena.

1.3 Pain

Pain is a common and ubiquitous experience that has been defined as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage…” (International Association for the Study of Pain Subcommittee on Taxonomy, 1994, p. S212). This definition highlights the emotionally aversive nature of pain. Understanding pain in such a way acknowledges that self-reported pain severity does not necessarily coincide with the severity of tissue damage or pathology; instead, the experience of pain is a complex subjective experience affected by sensory-physiological, motivational affective, and cognitive-evaluative components (Melzack & Wall, 1965; Turk & Melzack, 2001).

The experience of acute pain has a number of adaptive and protective functions (Millan, 1999; Wieseler-Frank, Maier, & Watkins, 2004). It calls attention to potentially dangerous stimuli and motivates action to remove oneself from danger. Indeed, the very experience of pain can facilitate the learning necessary to avoid potentially painful or injurious behaviours and circumstances in the future (Melzack & Wall, 1982). Pain from an injury can also prevent a person from engaging in activity that would interrupt the body’s natural healing processes or lead to further injury. The adaptive function of pain,
however, ceases when it persists beyond the time necessary to facilitate healing (International Association for the Study of Pain Subcommittee on Taxonomy, 1994); that is, when it becomes chronic.

Chronic pain is an outcome that occurs in a context with many facets including biological (e.g., genetics), psychological (e.g., interpretation of pain), and social factors (e.g., living conditions, income, stressors; see Butler & Moseley, 2003). Anxiety constructs are among the factors posited to play a role in the transition from acute to chronic pain. Anxiety has long been associated with certain pain conditions, including chronic back pain, arthritis, migraine headaches, and medically unexplained pain (Beesdo et al., 2009; McWilliams, Goodwin, & Cox, 2004; Von Korff et al., 2005). Individuals with musculoskeletal pain are frequently found to have co-occurring anxiety disorders (McWilliams, Cox, & Enns, 2003; McWilliams et al., 2004; Von Korff et al., 2005) at considerably higher rates than the general population (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Many dispositional characteristics that act as risk factors for the development of anxiety disorders have also been linked to pain perception and pain experience (e.g., anxiety sensitivity, Asmundson & Norton, 1995; Asmundson & Taylor, 1996; Ocanez, McHugh, & Otto, 2010; pain catastrophizing, Sullivan, Bishop, & Pivnik, 1995; for a review see Asmundson & Katz, 2009).

Several models contribute to our conceptualization of how acute pain becomes chronic (for a review see Asmundson & Wright, 2004). The fear-avoidance model of pain (Asmundson, Norton, & Norton, 1999; Asmundson, Norton, & Vlaeyen, 2004; Vlaeyen & Linton, 2000) specifically addresses the involvement of psychological processes and anxiety constructs in this progression. Following injury, the normal
healing process generally requires a progressive increase in activity to challenge soft tissue and regain function. For a small percentage of people, catastrophic interpretation of pain (e.g., worrying that pain is a sign of something more serious than it is; Sullivan et al., 1995) and other broad pain-related vulnerabilities can lead to avoidance of activities that might elicit further pain but otherwise promote healing. Ultimately, avoidance of such activities is posited to contribute to further disuse, deconditioning, and chronic pain (Asmundson et al., 2004; Vlaeyen & Linton, 2000). A brief discussion of the involvement of broad pain-related vulnerabilities will follow.

Pain catastrophizing, fear of pain, and pain-related anxiety are central to fear-avoidance models of pain. Pain catastrophizing is a construct describing a cognitive and emotional schema that results in negative responses to actual or anticipated pain (Quartana, Campbell, & Edwards, 2009; Sullivan, Thorn, Keefe, Martin, Bradley, & Lefebvre, 2001). The construct is characterized by the tendency to exaggerate the threat value of pain, feelings of helplessness in response to pain, and an inability to suppress or inhibit pain-related thoughts in anticipation of, during, or following pain (Sullivan et al., 2001). Fear of pain (McNeil & Vowles, 2004) and pain-related anxiety (McCracken & Dhingra, 2002) are constructs that are closely related, yet conceptually distinct. Fear has been conceptualized as a reaction to a current, specific, identifiable threat which prompts a behaviour intended to reduce proximity to danger; in contrast, anxiety is more anticipatory in nature, promoting arousal and vigilance in order to avoid contact with potential future threats (Barlow, 2002). Together, catastrophic interpretations of pain, fear of pain, and pain-related anxiety have been thought to promote hypervigilance to pain sensations and avoidance of behaviours that could potentially elicit further pain.
sensations but could also promote healing (Asmundson et al., 2004; Vlaeyen & Linton, 2000).

Fear-avoidance models also purport that there are certain predispositional differences that make it more likely for pain to be interpreted catastrophically. For example, anxiety sensitivity is a dispositional propensity to fear the physical sensations associated with anxiety (e.g., increased heart rate, sweating, dizziness) due to the belief that these sensations are a signal of harmful physical, social, or psychological consequences (Reiss & McNally, 1985; Taylor, 1999). Anxiety sensitivity has been implicated as a vulnerability factor for the development of anxiety disorders and may underlie individual differences in general fearfulness (Reiss & McNally, 1985; Taylor, 1999). Anxiety sensitivity has also been thought of as a predisposing factor for pain catastrophizing (e.g., Norton & Asmundson, 2003). Intolerance of uncertainty is another dispositional tendency, characterized by a propensity to react negatively to uncertain situations. This construct is also believed to underlie anxiety disorders (Carleton, Mulvogue, Thibodeau, McCabe, Antony, & Asmundson, 2012) and has been associated with anxiety sensitivity and pain-related anxiety (Carleton et al., 2007b). Lastly, distress tolerance has been defined as the ability (or perceived ability) to tolerate negative emotional, psychological, or physical states (Bernstein, Trafton, Ilgen, & Zvolensky, 2008; Simons & Gaher, 2005). Distress tolerance involves both a psychological and behavioural capacity to withstand or endure such negative states without attempts at experiential avoidance (Lynch & Mizon, 2011). Researchers have suggested that individuals who tolerate the most (over-tolerance) and least (intolerance) distress exhibit more maladaptive behaviours and experience less well-being (see Lynch & Mizon,
Distress tolerance has been implicated in the development and aggravation of numerous psychopathologies (e.g., anxiety disorders, depression, substance use, chronic pain; see Zvolensky, Bernstein, & Vujanovic, 2011). Although yet to be tested empirically within fear-avoidance pain models, exploration of distress tolerance in this context appears well-warranted (Asmundson et al., 2011).

1.4 The Co-Occurrence of Pain and Smoking

Epidemiological research has identified cigarette smoking as a risk factor for specific pain conditions (e.g., low back pain; Zvolensky, McMillan, Gonzalez, & Asmundson, 2009, 2010; Palmer, Syddall, Cooper, & Coggon, 2003). Indeed, a survey of the Canadian population indicated that the prevalence of back pain appears to be significantly higher for smokers (23%) than for non-smokers (16%; Alkherayf et al., 2010). Former smokers also evidence a higher prevalence rate of chronic pain than individuals who have never smoked, with tobacco smoking having been implicated in the development of acute and chronic pain conditions (John et al., 2006). The question remains whether the described co-occurrence is the result of a causal effect, and if so, in which direction does it occur (e.g., does smoking cause pain or does pain cause smoking, or both)?

1.4.1 The effect of smoking on pain. To date, much research has been directed at investigating the effects of smoking on pain. Indeed, smoking has been conceptualized as an indirect causal factor in the development of acute and chronic pain conditions. For example, smoking has been implicated in the development of chronic back pain via vascular disease, which can cause malnourishment of spinal disc cells, disk degeneration, and related back pain (see Wei & Hui, 2010). As a result, smokers are more likely to
suffer from lumbar disc herniation than non-smokers, even after adjusting for age, body mass index, exercise tendencies, and other variables that might affect blood perfusion (Jhawar, Fuchs, Colditz, & Stampfer, 2006; Wei & Hui, 2010). Twin studies have also shown that smoking may be predictive of disc height reduction and degeneration (Videman, Battie, Parent, Gibbons, Vainio, & Kaprio, 2000; Wei & Hui, 2010), both of which can contribute to the development of low back pain.

**1.4.2 Pain as a motivator to smoke.** More recently, research has been directed at investigating how pain may act as a source of motivation to increase smoking behaviours (Ditre & Brandon, 2008). Specifically, evidence suggests smoking may produce a temporary analgesic effect, providing temporary relief of pain (see Shi, Weingarten, Mantilla, Hooten, & Warner, 2010), thereby increasing smoking urge and reinforcing smoking behaviour as a means to cope with pain (Ditre & Brandon, 2008). Several studies using experimental pain have provided evidence for the involvement of nicotine in altered pain perception (see Ditre, Brandon, Zale, & Meagher, 2011; Shi et al., 2010). Jamner and colleagues (1998) found that administration of nicotine by nasal spray or transdermal patch significantly increased the electrical stimulation required to evoke a pain response in male participants. Smoking a cigarette has also been found to increase tolerance to experimental pain (see Ditre, Brandon, Zale, & Meagher, 2011; Shi et al., 2010). An investigation using a two-factor repeated measures design assessed the effects of cigarette smoking on pain tolerance in response to thermal pain stimuli and found that smoking cigarettes produced significant increases in pain tolerance compared with pain induction after a 12-hour abstinence from smoking (Lane, Lefebvre, Rose, & Keefe, 1995). Higher pain tolerance was also observed when participants smoked normal
cigarettes versus denicotinized cigarettes; however, these increases were not significant, suggesting the involvement of other factors beyond nicotine analgesia in the differences observed in pain perception. Indeed, a recent review of the literature assessing the effects of nicotine on pain perception revealed mixed results with only 10 out of 18 studies evidencing analgesic effects of tobacco on pain inhibition (Ditre et al., 2011). The authors concluded that differences in nicotine dosing, pain stimuli (e.g., cold pressor, heat, electrical stimulation), and sex are factors likely responsible for the mixed evidence of smoking-related analgesia in experimentally induced pain.

A review by Shi and colleagues (2010) provides clarification regarding which experimental conditions in conjunction with nicotine resulted in the described analgesic effect. In terms of pain stimuli, they found nicotine consistently increased pain threshold in studies using cold pressor, whereas heat and electrical stimulation provided mixed results. In terms of sex, studies using male participants consistently reported that smoking produced analgesia, whereas studies with mostly or only females did not. On the contrary, there were three studies that found no sex differences, one of which used transdermal nicotine and electrical stimulation (Jamner, Girdler, Shapiro, & Jarvik, 1998) and two of which used a smoking task and cold pressor pain induction (Kanarek & Carrington, 2004; Nastase, Ioan, Braga, Zagrean, Moldovan, 2007). Evidence for the analgesic effect of nicotine in response to experimental pain appears to be growing; however, the standardization of procedures used to assess such effects is becoming necessary to clarify the mechanisms underlying the observed relationship.

Post-operatively, nicotine has been found to have an analgesic effect for both male and female patients. In a double blind, randomized, placebo-controlled study, female
patients were given either nicotine nasal spray or a placebo following uterine surgery (Flood & Daniel, 2004). Women treated with the nicotine nasal spray reported lower pain levels and used only half the amount of morphine within the 24-hour time period following surgery when compared to the control group. The analgesic effect of nicotine has also been observed with both male and female participants following dental surgery. Yagoubian and colleagues (2011) conducted a single-center, prospective, randomized, double-blind, crossover trial in which 20 patients (11 female) had all 4 third molars removed. Patients received either nicotine nasal spray (3mg) or placebo prior to extraction of third molars on one side of their jaw. One week later, the alternate treatment was administered and the contralateral third molars were removed. The nicotine treatment was associated with a significant decrease in pain following surgery; however, there were no significant differences between conditions regarding the amount of hydrocodone and acetaminophen used by the patients. The authors concluded that the nicotine was acting as an adjuvant analgesic with the opioids.

1.4.3 The involvement of mood and context. The analgesic effect of nicotine may be one factor involved in reinforcing smoking behaviour as a means of managing pain (Ditre & Brandon, 2008); but, there is evidence to suggest that other pain-related mechanisms mediate and moderate this relationship. Smoking has been conceptualized as a coping mechanism used to reduce negative affective states in general (Shadel & Mermelstein, 1993), as well as those experienced as a result of pain (Ditre & Brandon, 2008). As previously discussed, nicotine stimulates nAChRs and dopamine receptors, which floods the body with endorphins and provides a sense of relaxation and pleasure (Nestler, 1999). From a behavioural perspective, this process could be considered a form
of negative reinforcement when used to relieve negative affective states (Eissenberg, 2004). However, a cognitive social learning perspective would highlight the importance of the situational context and smoking expectancies (i.e., smoking-related outcome expectations) in the mood regulatory effects of nicotine (Shadel & Mermelstein, 1993). Indeed, there is evidence to suggest that a reduction in negative affect as a result of smoking is most pronounced when individuals expect that smoking will aid in mood regulation (Ditre, Heckman, Butts, & Brandon, 2010; Juliano & Brandon, 2002; Wetter et al., 2004), as well as when smoking is combined with distraction (Kassel, 1997).

1.4.4 The involvement of predispositional and pain-related anxiety constructs.

Individual differences in predispositional and pain-related anxiety constructs have also been found to play a role in the effect that smoking has as a mood regulator. When in high stress situations, individuals with high levels of trait anxiety or anxiety sensitivity are more likely to report a reduction in anxiety due to smoking than individuals with low trait anxiety or anxiety sensitivity (Evatt & Kassel, 2010). Evidence also exists to suggest that anxiety-related constructs may contribute to maladaptive beliefs about the effect that smoking will have on pain, as well as the use of maladaptive smoking behaviours in response to pain (Gonzalez, Hogan, McLeish, & Zvolensky, 2010; Zvolensky, Stewart, Vujanovic, Gavric, & Steeves, 2009). Individuals with pain-related anxiety are more likely to report higher expectancies that smoking would reduce negative affect (Gonzalez et al., 2010). Further, individuals with high anxiety sensitivity and pain-related anxiety have been found less likely to use adaptive coping skills to deal with pain and negative affect, and more likely to use tobacco instead (Zvolensky et al., 2009).

Such findings are further supported by a recent investigation which found that
experimental manipulations designed to challenge expectations that smoking will reduce pain and provided adaptive pain smoking strategies was effective in reducing smoking urge and immediate smoking behaviour in response to experimental pain induction (Ditre et al., 2010). Using smoking as an analgesic may be particularly problematic because it is also associated with reports of increased pain intensity and reduced pain-related function (Patterson et al., 2012).

1.4.5 Experimental evidence. The available research evidence demonstrates associations between pain, predispositional and pain-related anxiety constructs, and smoking expectancies that could potentially increase smoking usage as a means to avoid pain and negative affective states. Research designs assessing these relationships have been mostly correlational (e.g., Davidson, Davidson, Tripp, & Borshch, 2005; Ditre et al., 2010) and cross-sectional demonstrating that individuals experiencing pain are more likely to be nicotine dependent (e.g., Zvolensky et al., 2009). Despite the previous research, causal relationships between pain – chronic or otherwise – and smoking have only been assessed in one experimental investigation (Ditre & Brandon, 2008).

Ditre and Brandon (2008) assessed the effects of experimentally-induced pain on smoking urge and behaviour using a between-subjects design in which participants comprised 132 smokers who were randomly assigned to a pain or no-pain condition. Pain condition participants were asked to place their arm in a cold pressor with water between 0 degrees and 1 degree Celsius, whereas those assigned to the no-pain condition submerged their arm in a bath of room temperature water. Following the pain manipulation task, participants completed measures assessing mood and urge to smoke. An unlit cigarette was then placed in front of participants and they were given the
opportunity to smoke. Results indicated that cold pressor pain resulted in greater reports of smoking urges and increases in immediate smoking behaviour, suggesting that experimental pain may be sufficient to increase smoking motivations.

1.5 Current Investigation: Purpose and Hypotheses

Findings from Ditre and Brandon (2008) were the first to empirically demonstrate the effects of experimental pain on smoking urge and smoking behaviour and, therefore, represent a unique and important contribution to the extant literature; however, the findings were novel and replication is necessary. Furthermore, the investigation had several methodological limitations that warranted further attention. Limitations are described below in order to frame the rationale for the current investigation.

The proposed investigation sought to replicate and extend the work of Ditre and Brandon (2008) assessing the effects of experimental pain on smoking urge and behaviour in three ways. First, Ditre and Brandon employed the cold pressor task, performed by immersing the hand into ice water until pain was no longer tolerable, as their method of pain induction; however, maintaining precise temperatures using the cold pressor can be difficult and can affect the reliability of results (Mitchell, MacDonald, & Brodie, 2004). In addition, cold pressor pain is thought to mimic some subjective qualities observed in chronic pain patients (Keogh, Hatton, & Ellery, 2000; Rainville, Feigne, Bushnell, & Duncan, 1992) but has been posited to fall outside the normal range of pain experiences, perhaps due to activation of more brain regions associated with affective pain processing than would thermal heat pain or cold pain (Duerden & Albanese, 2011). As such, the generalizability of results across pain modalities (i.e., thermal heat pain) remains unknown.
The proposed investigation was designed for three purposes. The first purpose was to assess the direct effects of experimental heat pain on smoking urge and behaviour, as well as to explore the indirect effects of experimental heat pain on smoking behaviour via smoking urge using state-of-the-art thermal heat pain-induction technology. The second purpose was to assess the direct and indirect effects of smoking expectancies on smoking urge and behaviour. The third purpose was to assess the effects of anxiety vulnerabilities as well as pain-induced anxiety and negative affect on the relationship between pain and smoking urge and behaviour. Accordingly, the proposed investigation had 5 main hypotheses:

1. Experimental heat pain induction will increase self-reported urge to smoke (Figure 1; Ditre & Brandon, 2008).

2. Experimental pain will increase immediate smoking behaviour (Figure 1; Ditre & Brandon, 2008).

3. The effect of pain on immediate smoking behaviour will be mediated by urge to smoke (Figure 1; Ditre & Brandon, 2008).

4. The effect of pain on smoking urge will be mediated by pain-induced negative affect (Ditre & Brandon) and state anxiety (Figure 2; Evatt & Kassel, 2010).

5. Predispositional anxiety vulnerabilities (anxiety sensitivity, distress tolerance, intolerance of uncertainty) will account for statistically significant variance in measures of smoking urge (and, therein, immediate smoking behaviours) through their effect on pain, pain-related anxiety constructs (pain-related anxiety, pain catastrophizing), state anxiety and negative affect, and smoking expectancies (Figure 3; Gonzalez et al., 2010; Zvolensky et al., 2009).
2.0 Methods and Materials

2.1 Participants

Ethical approval was obtained through the University of Regina Ethics Board. Participants comprised smokers from the University of Regina and the general community. Participants from the university were recruited through the Psychology Department research participant pool as well as poster advertisement. Participants from the general community were recruited through the local newspaper and online advertisements, which described the research as an inquiry into the relationship between anxiety and smoking behaviours and outlined the need for participants. Participants recruited through the research participant pool were given the option of receiving $20 cash or being entered into three cash draws of $100 for their participation, as well as an additional option of receiving bonus credits for their participation. Participants recruited from the general and university communities were given the options of receiving cash compensation or being entered into the cash draws for their participation. Eligible participants were individuals 18-65 years of age, who were fluent in English, and nicotine dependent (a score of 1 or greater on the Fagerström Test for Nicotine Dependence; FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991). Individuals with contraindicated medical conditions (e.g., medicated pain conditions or injury, diabetes, epilepsy) and those taking medication for heart or circulatory problems were excluded from participation as these factors could have an effect on pain perception. Participant characteristics will follow in the Results section.
Figure 1. Diagram of structural equation model to be tested as outlined in hypotheses 1, 2, and 3.

Note. Smoking urge as measured by the Brief Questionnaire of Smoking Urges; Experimental pain as measured by the Numerical Rating Scale pain ratings.
Figure 2. Diagram of structural equation model to be tested as outlined in hypothesis 4.

Note. State negative affect as measured by the Positive and Negative Affect Schedule negative affect subscale; State anxiety as measured by the State Trait Anxiety Index state anxiety subscale; Experimental pain as measured by the Numerical Rating Scale pain ratings; Smoking urge as measured by the Brief Questionnaire of Smoking Urges.
Figure 3. Diagram of structural equation model to be tested as outlined in hypothesis 5.

Note. * State negative affect as measured by the Positive and Negative Affect Schedule negative affect subscale; State anxiety as measured by the State Trait Anxiety Index state anxiety subscale; DTS - Distress Tolerance Scale; ASI-3 - Anxiety Sensitivity Index; IUS-12 - Intolerance of Uncertainty Scale, Short Form; PASS-20 - Pain Anxiety Symptoms Scale; PCS - Pain Catastrophizing Scale; Smoking expectancies as measured the Brief Smoking Consequences Questionnaire - Adult.
2.2 Design

The primary analytical procedure for this investigation was a 2 (pain manipulation) x 2 (sex) between-subjects design to assess the relative effects of pain induction on smoking urge and behaviour. Participants were stratified by sex and randomly assigned to one of two conditions (heat pain, no pain) using an online random assignment generator. For replication and consistency, this investigation employed the same measures as Ditre and Brandon (2008), as well as additional measures assessing predispositional and pain-related anxiety constructs. Though measures and subscales are detailed below for descriptive purposes, only full scales scores were calculated, unless otherwise specified. Cronbach’s alpha for measures based on the present sample are reported with other descriptive statistics in Table 1.

2.3 Measures and Equipment

_The Fagerström Test for Nicotine Dependence_ (FTND; Heatherton et al., 1991) is 6-item self-report measure that assesses nicotine dependence. Three items are multiple choice, scored from 0 to 3, and three items are yes/no, scored 0 (no) and 1 (yes). Despite questions related to reliability, the FTND is a widely used measure of nicotine dependence (Heatherton et al., 1991). A score of 1 or greater was used as an inclusion criterion prior to acceptance into the investigation.

_Anxiety Sensitivity Index-3_ (ASI-3; Taylor et al., 2007). The ASI-3 is an 18-item self-report measure that assesses the extent to which a person fears anxiety-related arousal symptoms due to the belief that such sensations will lead to physical, social, or psychological harm (Reiss, Peterson, Gursky, & McNally, 1986; Taylor, 1999). The ASI-3 has three factorially derived subscales that assess theorized dimensions of fear of cognitive dyscontrol (e.g., _It scares me when I am unable to keep my mind on a task_), fear of somatic
sensations (e.g., *When my stomach is upset, I worry that I might be seriously ill*), and 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*). Items are rated on a 5-point Likert scale ranging from 0 (very little) to 4 (very much). The ASI-3 has demonstrated strong reliability and validity in previous studies (Taylor et al., 2007). The ASI-3 was employed as a baseline measure to assess individual differences in anxiety sensitivity.

*Distress Tolerance Scale* (DTS; Simons & Gaher, 2005). The DTS is a 15-item self-report measure designed to assess a person’s capacity to experience and withstand negative psychological and emotional states. The DTS has four subscales: tolerance (e.g., *Feeling distressed or upset is unbearable to me*), appraisal (e.g., *My feelings of distress or being upset are not acceptable*), absorption (e.g., *My feelings of distress are so intense that they completely take over*), and regulation (e.g., *When I feel distressed or upset, I must do something about it immediately*). Items are responded to on a 5-point scale ranging from 1 (strongly agree) to 5 (strongly disagree). The DTS has demonstrated strong internal consistency (α = .91; Leyro, Bernstein, Vujanovic, McLeish, & Zvolensky, 2011) and was used as one of the baseline measures.

*Intolerance of Uncertainty, Short Form* (IUS-12; Carleton, Norton, & Asmundson, 2007a). The IUS-12 is a 12-item measure designed to assess the level of distress an individual may experience due to uncertainty and its potential consequences for the future. The measure is comprised of two subscales: prospective anxiety (e.g., *Unforeseen events upset me greatly*) and inhibitory anxiety (e.g., *When it’s time to act, uncertainty paralyses me*). Items are responded to on a 5-point Likert scale ranging from 1 (*not at all characteristic of me*) to 5 (*entirely characteristic of me*). The IUS-12 total and subscale scores have demonstrated good internal, convergent, and discriminant validity, as well as
good internal consistency (Carleton et al., 2007a; McEvoy & Mahoney, 2011). The IUS-12 was used as one of the baseline measures.

*Pain Anxiety Symptoms Scale-20* (PASS-20; McCracken & Dhingra, 2002). The PASS-20 is a 20-item self-report measure designed to assess pain-related anxiety across four subscales: escape and avoidance (e.g., *I try to avoid activities that cause pain*), cognitive anxiety (e.g., *During painful episodes it is difficult for me to think of anything else besides the pain*), pain-related fear (e.g., *Pain sensations are terrifying*), and physiological anxiety (e.g., *I find it difficult to calm my body down after periods of pain*). Items are responded to on a 6-point scale ranging from 0 (never) to 6 (always). The PASS-20 total and subscale scores have demonstrated factorial validity in both clinical (e.g., Coons, Hadjistavropoulos, & Asmundson, 2004) and non-clinical (Abrams, Carleton, & Asmundson, 2007) samples, as well as strong internal consistency and construct validity (McCracken & Dhingra, 2002). The PASS-20 was employed as one of the baseline measures.

*Pain Catastrophizing Scale* (PCS; Sullivan et al., 1995). The PCS is a 13-item scale that assesses the extent to which individuals engage in catastrophic thoughts and feelings when in pain. It is comprised of three subscales: rumination (e.g., *I can’t seem to keep it out of my mind*), magnification (e.g., *I become afraid that the pain will get worse*), and helplessness (e.g., *It’s awful and I feel that it overwhelms me*). Items are responded to on a 5-point scale ranging from 0 (not at all) to 4 (all the time). The PCS has demonstrated strong reliability and good construct validity (Osman, Barrios, Gutierrez, Kopper, & Grittmann, 2000). The PCS was used as one of the baseline measures.

*Positive and Negative Affect Schedule* (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS is a 20-item questionnaire designed to assess both state and trait positive and
negative affect. Items are responded to on a 5-point scale ranging from 1 (very slightly or not at all) to 5 (extremely). The two subscales have demonstrated strong internal consistency (Crawford & Henry, 2004; Watson, et al., 1988). The PANAS negative affect subscale was used as a baseline measure to assess trait and state negative affect, and to assess state negative affect upon completion of pain manipulations.

**State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Luschene, Vagg, & Jacobs, 1983).** The STAI is a 40-item self-report measure designed to assess state anxiety (e.g., *I feel nervous*) and trait anxiety (e.g., *I feel like a failure*). Items are responded to on a 4-point Likert scale with options ranging from 1 (not at all) to 4 (very much). The trait anxiety subscale has demonstrated good internal consistency ($\alpha = .89 – .96$) and high test-retest reliability ($r = .65 – .86$; Spielberger et al., 1983). The state anxiety subscale also possesses good internal consistency ($\alpha = .86 – .95$) and low to moderate test-retest reliability ($r = .16 – .62$) when compared with the trait anxiety scale, suggesting appropriate receptivity to situational influences (Spielberger et al., 1983). The STAI was used as a baseline measure and to assess state anxiety upon completion of pain manipulation trials.

**The Brief Smoking Consequences Questionnaire-Adult (BSCQ-A; Rash & Copeland, 2008)** is a 25-item questionnaire adapted from the 50-item Smoking Consequences Questionnaire (Copeland, Brandon, & Quinn, 1995), designed to assess smoking-related outcome expectancies. Items are responded to on a Likert scale with options ranging from 0 (completely unlikely) to 9 (completely likely). Only the following 5 subscales will be utilized, as they have particular relevance for the current investigation: (a) a 3-item measure assessing expectancies for negative affect reduction (e.g., *When I'm angry, a cigarette can calm me down*); (b) a 2-item measure of expectancies for state enhancement
(e.g., *Smoking a cigarette energizes me*); (c) a 2-item measure of expectancies related to craving and addiction (e.g. *Nicotine “fits” can be controlled by smoking*); (d) a 3-item measure of expectancies for social facilitation (e.g., *I feel more at ease with other people if I have a cigarette*); and (e) a 2-item measure of expectancies for boredom reduction (e.g., *If I have nothing to do, a smoke can help kill time*). Additionally, a 6-item scale (the Pain and Smoking Expectancies scale) will be employed to assess expectancies that smoking will help cope with pain (Ditre & Brandon, 2008). Each of these subscales have demonstrated adequate internal consistency and were utilized as baseline measures.

*Numerical rating scale* (NRS; Dworkin et al., 2005). The NRS is a measure used to numerically assess pain intensity. At baseline and following each trial of pain manipulation, participants were asked to rate their pain intensity on an 11-point scale ranging from “0 = no pain” to “10 = worst pain imaginable.”

*Questionnaire of Smoking Urges-Brief* (QSU-brief; Cox, Tiffany, & Christen, 2001). The QSU-brief is a 10-item measure assessing urge to smoke. It has two subscales that will comprise the primary dependent measures (desire/urge to smoke with anticipation for pleasure/reward; desire/urge to smoke with anticipation for relief of negative affect). Items are responded to on a Likert scale with options ranging from 1 (strongly disagree) to 7 (strongly agree). The QSU-brief total scale and subscales have demonstrated good internal consistency (Cox et al., 2001). These constructs were assessed at baseline and upon completion of all pain manipulation trials.

*Latency to smoke.* Experimental investigation has found that increased negative affect and anxiety are associated with decreased latency to smoke (time elapsed until cigarette is lit; Conklin & Perkins, 2005). As a behavioural index of smoking urge, latency to smoke was assessed in terms of the time from when the cigarette and lighter were first placed on
the counter beside the participant until participant first attempted to light the cigarette. For those who asked a clarifying question (e.g., I can smoke in here?), the time it took to ask the question and receive an answer was subtracted from their latency time. This was recorded by discrete video camera and scored independently by two trained raters (correlation between raters was $r = .996, p < .001$). Since there was no numerical value for participants that did not attempt to smoke, the longest demonstrated latency value was recorded for these participants (39.2 seconds).

*Medoc Pathway Pain and Sensory System.* Pain manipulations consisted of thermal heat pain delivered by the Medoc Pathway Pain and Sensory Evaluation System – ATS model (Ramat Yishay, Israel), which allowed for precise, programmable delivery of heat stimulation using the Advanced Thermal Stimulator (ATS) thermode. The ATS thermode has a 30mm x 30mm contact pad that produces temperatures between 0°C and 55°C at rate of change of up to 8°C per second. The Pathway system comes equipped with several safety measures to ensure the safety of participants. Each time the equipment is turned on, hardware self-test procedures are executed by the system to ensure all sensors are functioning properly. If a malfunction were to be detected, the system would be prevented from operating. If the system were to operate beyond the set temperature parameters, power would automatically be disconnected from the thermode. A physical trigger mechanism was also available to participants and operators, which allowed for manual termination of pain stimuli at any time.

### 2.4 Procedure

All participants were asked to smoke one cigarette approximately one hour prior to their appointment in order to standardize smoking behaviour prior to the experiment. Participants were asked to bring one of their own cigarettes and told they may have a
chance to smoke during the experiment. Cigarettes were provided for the six participants who either did not bring their own cigarettes or who brought unrolled tobacco. Upon arrival, participants were informed that the investigation comprised two phases with a two-tiered consent process. The first phase was described as an inquiry into the relationship between anxiety and smoking behaviours. After obtaining initial consent, participants were asked the time of their last cigarette. They were also asked to temporarily turn in all cigarettes and smoking paraphernalia (e.g., lighters, matches). They were assured that these items would be stored safely, and asked to remove one cigarette from the package. Participants were told that if they decided to participate in the second phase of the study, there would be a planned smoke break and that the cigarette that was removed from their pack would be brought to them at that time. The rationale for allowing participants to smoke was that we did not want people leaving the lab halfway through the investigation for a smoke break. The experimental room was set up to simulate a smoking lab (e.g., ‘smoking lab’ and ‘smoking permitted’ signage, ashtrays) and participants were informed that smoking was permitted in the experimental room. All cigarettes (including the cigarette that was removed from the pack) and smoking paraphernalia were stored in a separate room. Participants were then asked to complete the baseline measures (i.e., ASI-3, DTS, FTND, IUS-12, PCS, PASS-20, STAI1, QSU-brief1, PANAS1) on a computer located in the lab. For measures that were administered more than once, subscripts were used to clarify which administration of the measure was being referenced.

During the second phase of consent, participants were informed that the purpose of phase two was to assess pain tolerance and the Medoc system was introduced as the method used to induce pain. The experimenter also explained the purpose of using a two-tiered consent process, which was to ensure that baseline measures would not be affected
by anticipatory anxiety related to undergoing a pain induction. Participants were reminded that withdrawal from the investigation would not result in any negative consequences; however, all participants consented to participation in the second phase. At this point, a second measurement of smoking urge (i.e., QSU-brief2) was administered to assess for an effect of anticipatory anxiety.

Participants were grouped by sex and then randomly assigned to a heat pain or no-pain condition using an online random assignment generator. Participants in the heat pain condition underwent four trials of heat pain induction delivered by the Medoc ATS system, to the dorsal surface of their non-dominant hand. A 90-second delay followed each trial. Participants were asked to click a button when the task became too intense to tolerate, at which time the trial ended. All trials began at a baseline temperature of 32°C and increased at a rate of 1°C per second with a maximum temperature of 51 °C. The NRS(1,2,3,4) were administered after each pain induction trial. Upon completion of all trials, the QSU-brief3, STAI2, and the PANAS2, were administered. Participants in the no-pain condition followed the same procedure, except detection of mildly cool (minimum of 20°C) and mildly warm stimuli (maximum of 40°C) were assessed as opposed to heat pain tolerance.

Upon completion of pain induction trials and the second battery of questionnaires, participants were informed that there would be a 5 minute break before moving on to the next phase of the investigation and that they could use the experiment room to smoke if they wished. Participants were provided with a cigarette (the one that was earlier removed from their pack of cigarettes or one from the experimenter if they did not bring a pre-rolled cigarette) and a lighter; however, the lighter provided was not functional (i.e., containing no lighter fluid). Before leaving the room, the experimenter informed the participant that he/she would return in 5 minutes. The experimenter then left the room, and latency to
smoke was recorded by a discrete video camera. Once the participant attempted to light the
cigarette, or if the participant had not attempted to light the cigarette within 5 minutes, the
experimenter returned to debrief the participant and gave back any cigarettes and smoking
 paraphernalia that had been previously stored.

3.0 Results

3.1 Preliminary Analyses

3.1.1 Descriptive statistics. Data were collected from students from the University
of Regina (n = 5; \( M_{age} = 19.80, SD = 1.48 \) years; 80% male) and adults from the
surrounding community (\( M_{age} = 37.69, SD = 13.68 \) years; 58.6% female). Recruitment of
smokers without exclusion criteria (medicated pain conditions, medicated injury, diabetes,
epilepsy, medicated heart or circulatory problems) within the allotted time frame was a
particular challenge of the current investigation and resulted in a sample that was smaller
than originally proposed (i.e., a sample of 96); therefore, power was insufficient to conduct
all mediation analyses as proposed. Participants had an average FTND score of \( 5.59 (SD =
2) \), indicating that participants’ nicotine dependence level was moderate; however, this
result should be interpreted with caution as the FTND demonstrated a less than satisfactory
reliability (\( \alpha = .58 \)).

To characterize the sample, descriptive statistics (e.g., means, standard deviations)
and internal consistencies were calculated and reported for all measures and subscales
(Table 1). All total scale alphas, with the exception of the FTND, were within an
acceptable range (Bland & Altman, 1997). A series of independent \( t \)-tests were conducted
to assess differences between groups on baseline measures (i.e., ASI-3, DTS, FTND, IUS-
12, PCS, PASS-20, STAI\(_1\), QSU-brief\(_{1,2}\), PANAS\(_1\)). A large number of \( t \)-tests were
performed; nevertheless, alpha was set liberally at .05 in order to explore the effect of any
Table 1: *Descriptive statistics*

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<td>33.00</td>
<td>13.88</td>
<td>5.71</td>
<td>0.901</td>
</tr>
<tr>
<td>SCQ Negative Affect Reduction</td>
<td>3.00</td>
<td>27.00</td>
<td>21.72</td>
<td>6.52</td>
<td>0.954</td>
</tr>
<tr>
<td>SCQ Stimulation</td>
<td>0.00</td>
<td>18.00</td>
<td>9.72</td>
<td>5.53</td>
<td>0.930</td>
</tr>
<tr>
<td>SCQ Social facilitation</td>
<td>0.00</td>
<td>27.00</td>
<td>15.19</td>
<td>7.20</td>
<td>0.765</td>
</tr>
<tr>
<td>SCQ Craving Addiction</td>
<td>5.00</td>
<td>18.00</td>
<td>13.63</td>
<td>4.31</td>
<td>0.762</td>
</tr>
<tr>
<td>SCQ Boredom Reduction</td>
<td>6.00</td>
<td>18.00</td>
<td>14.41</td>
<td>4.13</td>
<td>0.779</td>
</tr>
<tr>
<td>SCQ Pain Relief</td>
<td>4.00</td>
<td>53.00</td>
<td>25.09</td>
<td>14.45</td>
<td>0.886</td>
</tr>
<tr>
<td>State Trait Anxiety Inventory&lt;sub&gt;1&lt;/sub&gt; State</td>
<td>20.00</td>
<td>56.00</td>
<td>35.22</td>
<td>9.49</td>
<td>0.905</td>
</tr>
<tr>
<td>State Trait Anxiety Inventory&lt;sub&gt;2&lt;/sub&gt; State</td>
<td>29.00</td>
<td>57.00</td>
<td>41.25</td>
<td>9.35</td>
<td>0.912</td>
</tr>
</tbody>
</table>

*Note.* QSU-brief refers to the Brief Questionnaire of Smoking Urges; for measures that were administered more than once, subscript denotes which administration is being referenced.
Table 2: *Baseline independent t-tests*

<table>
<thead>
<tr>
<th>Measure</th>
<th>$t$</th>
<th>$Df$</th>
<th>$p$ (2-tailed)</th>
<th>$r^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety Sensitivity Index-3</td>
<td>2.15</td>
<td>32</td>
<td>.039</td>
<td>0.13</td>
</tr>
<tr>
<td>Brief Questionnaire of Smoking Urges$_1$</td>
<td>1.53</td>
<td>32</td>
<td>.135</td>
<td>0.07</td>
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<tr>
<td>Brief Questionnaire of Smoking Urges$_2$</td>
<td>1.96</td>
<td>32</td>
<td>.059</td>
<td>0.11</td>
</tr>
<tr>
<td>Distress Tolerance Scale</td>
<td>0.57</td>
<td>32</td>
<td>.570</td>
<td>0.01</td>
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<tr>
<td>Fagerström Test for Nicotine Dependence</td>
<td>1.06</td>
<td>32</td>
<td>.296</td>
<td>0.03</td>
</tr>
<tr>
<td>Intolerance of Uncertainty</td>
<td>0.88</td>
<td>32</td>
<td>.386</td>
<td>0.02</td>
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<tr>
<td>Pain Anxiety Symptoms Scale</td>
<td>0.44</td>
<td>32</td>
<td>.666</td>
<td>0.01</td>
</tr>
<tr>
<td>Pain Catastrophizing Scale</td>
<td>0.68</td>
<td>32</td>
<td>.499</td>
<td>0.01</td>
</tr>
<tr>
<td>PANAS Trait Negative Affect$_1$</td>
<td>1.15</td>
<td>32</td>
<td>.257</td>
<td>0.04</td>
</tr>
<tr>
<td>PANAS State Negative Affect$_1$</td>
<td>0.28</td>
<td>32</td>
<td>.778</td>
<td>0.00</td>
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<tr>
<td>SCQ Negative Affect Reduction</td>
<td>0.32</td>
<td>32</td>
<td>.754</td>
<td>0.00</td>
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<td>SCQ Stimulation</td>
<td>0.60</td>
<td>32</td>
<td>.555</td>
<td>0.01</td>
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<td>SCQ Social facilitation</td>
<td>1.26</td>
<td>32</td>
<td>.216</td>
<td>0.05</td>
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<td>SCQ Craving Addiction</td>
<td>1.11</td>
<td>32</td>
<td>.277</td>
<td>0.04</td>
</tr>
<tr>
<td>SCQ Boredom Reduction</td>
<td>-0.46</td>
<td>32</td>
<td>.647</td>
<td>0.01</td>
</tr>
<tr>
<td>SCQ Pain Relief</td>
<td>0.63</td>
<td>32</td>
<td>.317</td>
<td>0.01</td>
</tr>
<tr>
<td>State Trait Anxiety Inventory$_1$ State</td>
<td>0.05</td>
<td>32</td>
<td>.960</td>
<td>0.00</td>
</tr>
<tr>
<td>State Trait Anxiety Inventory$_2$ State</td>
<td>0.50</td>
<td>32</td>
<td>.621</td>
<td>0.01</td>
</tr>
</tbody>
</table>

*Note.* QSU-brief refers to the Brief Questionnaire of Smoking Urges; for measures that were administered more than once, subscript denotes which administration is being referenced.
possible between groups baseline differences, especially given increased risk for type II error inherent within analyses for small samples. Results revealed no significant differences between groups on baseline measures (see Table 2), except for the ASI-3, $t(32) = 2.147, p < .04, r^2 = .13$. Because of this, between-group differences on ASI-3 subscales were assessed individually using independent $t$-tests. Only the ASI-3 social subscale demonstrated significant between groups differences, $t(32) = 2.98, p < .01, r^2 = .22$.

Accordingly, ANCOVAs were conducted in the primary analyses to statistically control for group differences that occurred prior to randomization.

3.1.2 Manipulation checks. Manipulation checks were conducted to assess between groups differences in post-manipulation pain intensity (NRS$_2$), state negative affect (PANAS$_2$), and state anxiety (STAI$_2$). As expected, participants in the pain group reported higher pain intensity ($M_{pain} = 6.82, SD_{pain} = 1.70$) than those in the control group ($M_{control} = .29, SD_{control} = .77$), $t(32) = 14.39, p < .001, r^2 = .87$. Expected between groups differences did not immerge in post manipulation state negative affect ($M_{pain} = 37.37, SD_{pain} = 8.23; M_{control} = 35.06, SD_{control} = 2.18$), $t(32) = 1.40, p = .17, r^2 = .06$, or state anxiety ($M_{pain} = 14.5, SD_{pain} = 5.09; M_{control} = 13.06, SD_{control} = 3.19$), $t(32) = 1.51, p = .14, r^2 = .07$; therefore, these variables were excluded from the primary analyses.

3.1.3 Preliminary regression analyses. In order to inform subsequent mediation analyses, a preliminary regression was used to assess which, if any, of the proposed SCQ subscales were significant predictors of post-manipulation urge to smoke (QSU-brief$_3$). Results indicated only smoking expectancies related to craving and addiction significantly predicted post-manipulation urge to smoke, $\beta = 2.48, t(31) = 2.79, p < .001$, and explained a significant proportion of variance in post-manipulation urge to smoke, $R^2 = .45, F(1, 31)$.
As such, smoking expectancies were included in subsequent mediation analyses.

3.2 Primary Analyses

All $p$ values for the primary analyses were calculated as one-tailed. Analyses of covariance (ANCOVAs) using SPSS 20 were used to test hypothesis 1 and 2. Structural equation modeling (SEM) using AMOS 20.0 with bootstrapping and 95% confidence intervals was conducted to test all remaining hypotheses. Model fit was assessed using a variety of fit indices including $\chi^2$ (the model is acceptable if values are not significant), $\chi^2/df$ (values less than 2.0 are acceptable; Ullman, 2001), the comparative fit index (CFI; values should be less than .95; Hu & Bentler, 1999), the root mean square error of approximation (RMSEA; values of .01 or lower are considered excellent; MacCallum, Browne, & Sugawara, 1996), the Standardized Root Mean Square Residual (SRMR; values less than .06 are considered a good fit; Hu & Bentler, 1999). Effect sizes, as partial eta squared ($\eta_p^2$), were calculated and interpreted as suggested by Field (2005), wherein values of .01, .06, and .14 can be considered small, medium, and large, respectively.

3.2.1 Hypothesis 1: Smoking urge. To test the hypothesis that individuals in the experimental heat pain condition would report higher urge to smoke, a 2 x 2 ANCOVA was conducted using pain (i.e., heat pain, no pain) and sex (i.e., female, male) as the two independent variables, self-reported urge to smoke (QSU-brief3) as the dependent variable, and ASI-3 social as the covariate. The direct effect of ASI-3 social on urge to smoke was assessed. The interaction of pain condition and sex, as well as pain condition and ASI-3 social were also assessed.

Results indicated there was a significant main effect of pain condition on post-manipulation urge to smoke, $F(1,28) = 5.16, p = .03, \eta_p^2 = .17$, above and beyond the
effects of ASI-3 social, such that participants in the heat pain group reported significantly higher levels of smoking urge than those in the no pain group ($M_{pain} = 61.69$, $SD_{pain} = 18.45$; $M_{control} = 41.30$ $SD_{control} = 24.30$). There was no effect of sex on self-reported urge to smoke, $F(1,28) = .07$, $p = .80$. ASI-3 social had a significant main effect on smoking urge, $F(1,28) = 4.82$, $p < .04$, $\eta^2 = .16$, such that participants with higher ASI-social had significantly higher urge to smoke. The interaction of pain condition and sex on self-reported urge to smoke was not significant, $F(1,28) = .55$, $p = .47$, nor was the interaction of condition and ASI-3 social, $F(1,28) = 2.92$, $p = .10$.

### 3.2.2 Hypothesis 2: Smoking behaviour.

To test the hypothesis that individuals in the experimental heat pain condition would demonstrate shorter post-manipulation latency to smoke, a 2 x 2 ANCOVA was conducted using pain (i.e., heat pain, no pain) and sex (i.e., female, male) as the two independent variables, latency to smoke as the dependent variable, and the ASI-3 social subscale as the covariate. The direct effect of ASI-3 social on latency to smoke was assessed. The interaction of pain condition and sex, as well as pain condition and ASI-3 social on latency to smoke were also assessed for combined effects. Two participants were excluded from the remaining analyses because of experimenter error (i.e., they were cued to smoke too early in the procedure).

Results indicated that pain condition had a significant effect on latency to smoke, $F(1,26) = 5.168$, $p = .03$, $\eta^2 = .17$, above and beyond the effects of pre-manipulation group differences in ASI-3 social, such that participants in the heat pain group had significantly shorter latency times than those in the no pain group ($M_{pain} = 17.82$ seconds, $SD_{pain} = 10.47$; $M_{control} = 20.24$ seconds, $SD_{control} = 15.59$). Sex did not have an effect on latency to smoke, $F(1,26) = .04$, $p = .85$. ASI-3 social did have a significant effect on latency to smoke, $F(1,26) = 5.59$, $p < .03$, $\eta^2 = .18$, such that individuals with higher ASI-3 social
had shorter latency times. This was interpreted as a large effect size. The interaction between pain condition and sex was not significant $F(1,26) = .36, p = .55$. The interaction between pain condition and ASI-social did have a significant effect on latency to smoke, $F(1,26) = 7.22, p = .01, \eta^2_p = .22$, above and beyond the individual main effects of pain condition and ASI-social, such that as pain ratings and ASI-social increased, latency to smoke decreased. Since sex differences were not observed for smoking urge or latency to smoke, sex was not considered in subsequent analyses. A traditional chi-square difference test was conducted to assess whether there was a difference between groups in the number of participants who attempted to smoke. Results indicated that significantly more individuals in the pain group attempted to smoke (100%) than in the control group (62.5%), $\chi^2 (1, N = 32) = 7.39, p < .001$. Of the six participants who were provided with a cigarette from the experimenter (rather than their own cigarette), only one person from the control condition did not attempt to light up.

### 3.2.3 Mediation analyses: Hypothesis 3.
Mediation analyses were conducted to test the hypothesis that the causal effect of pain on latency to smoke would be mediated by urge to smoke (Figure 4). This was tested in AMOS using bootstrapping with 95% confidence intervals. The pain ratings from only the second pain trial (NRS$_2$) were used in the analyses because the control group experienced only two trials of warmth (along with two trials of cold), the first of which was considered a trial run. The direct path from NRS$_2$ pain ratings to latency to smoke was not significant, $\beta = .05, p = .37$; however, the indirect path from NRS$_2$ pain ratings to latency to smoke through smoking urge was significant, $\beta = -.23, p < .01$, suggesting the effects of pain on decreased latency to smoke were fully mediated by urge to smoke. The direct path from pain ratings to latency to smoke was, therefore, removed from the model.
Figure 4: Estimated model as outlined in hypothesis 3.

Note. All values represent standardized regression weights; ** is $p < .01$; *** is $p < .001$; Experimental pain as measured by the Numerical Rating Scale; pain ratings; Smoking urge as measured by the Brief Questionnaire of Smoking Urges.
3.2.4 Mediation analyses: All remaining hypotheses. The models proposed for the remaining hypotheses (Figures 2 and 3) were not tested as initially suggested. Since results of the ANCOVAs revealed the pain manipulation did not have an effect on post-manipulation state-emotionality or latency to smoke, these variables were removed from the analyses. A revised model (Figure 5), built upon the results of hypothesis 2 (Figure 1), was constructed and tested using a stepped approach. First, smoking expectancies related to cravings/addiction was included in the model as an independent variable. Due to the aforementioned involvement of the ASI-3 social subscale in the post-manipulation urge to smoke, it was also included in the model as an independent variable of urge to smoke, with a direct path as well as an indirect path through NRS2 pain ratings. Results indicated that all direct pathways were significant (see Figure 5) except for the path from NRS2 pain ratings to smoking urge, which trended towards significance ($p = .06$). The model was not extended to include anxiety constructs as proposed in hypotheses 4 and 5 (Figure 3) as statistical power became an issue in maintaining significance across pathways; therefore, hypotheses 4 and 5 were not assessed. The revised model demonstrated excellent fit to the data on most fit indices, $\chi^2 = 3.164$, $df = 5$, $p = .864$, CFI = 1.0, RMSEA = .000 (90% CIs: .000 - .195), EVCI = .747 (90% CIs: .806 - .997), with the exception being the SRMR = .0997.
Figure 5: Estimated revised model.

Note. All values represent standardized regression weights; * refers to $p = .06$; * is $p < .05$; ** is $p < .01$; *** is $p < .001$; ASI-3 Social as measured by the social subscale of the Anxiety Sensitivity Index 3; Experimental pain as measured by the Numerical Rating Scale pain ratings; Smoking urge as measured by the Brief Questionnaire of Smoking Urges; Craving/addiction smoking expectancies as measured the Brief Smoking Consequences Questionnaire - Adult.
4.0 Discussion

The current investigation was the second study to experimentally assess the effects of pain on smoking urge and behaviour. As previously discussed, there is a substantial literature investigating the mechanisms in which smoking contributes to the development of pain and chronic pain conditions (e.g., Jhawar et al., 2006; Videman et al., 2000; Wei & Hui, 2010) but few investigating the effects of pain on smoking. Ditre and Brandon (2008) conducted an empirical investigation demonstrating that experimental cold pressor pain was sufficient to increase both smoking urge and immediate smoking behaviour and, therein, proposed a cycle in which smoking not only leads to pain, but pain perpetuates smoking urge and behaviours.

The current investigation was designed, primarily, to replicate and extend the work of Ditre and Brandon (2008) assessing whether situational pain is sufficient to increase smoking urge and immediate smoking behaviour. Indeed, participants in the pain condition of the current investigation reported greater urge to smoke than those in the control group. As reviewed earlier, Ditre and Brandon employed the cold pressor task as their method of pain induction, whereas the current investigation assessed the effects of an alternate pain modality (heat) and alternate pain induction technique (electrical stimulation) and, thereby, extend the generalizability of past results. In terms of latency to smoke, Ditre and Brandon asked all participants to smoke at least one puff of a cigarette and measured the time it took for them to ‘light up.’ The current investigation did not require participants to ‘light up’ but, rather, gave them the option to do so. This design variant may provide a more ecologically valid metric for assessing smoking behaviour. There was a statistically significant difference between groups in
latency to smoke, a mean difference of 2.42 seconds, which was consistent with the mean difference of 2.39 seconds reported by Ditre and Brandon (2008). Though this difference in latency between groups represented a large effect size ($\eta^2_p = .18$), the latency to smoke index was utilized primarily as a metric of external validity for the main dependent variable of smoking urge (i.e., given the current results, we would expect smoking urge would lead to actual smoking behaviour). Further, significantly more participants in the pain group vs. the control group chose to ‘light up’ (100%) vs. not ‘light up’ (62.5%) following pain induction. Taken together, current results support and extend those of Ditre and Brandon, indicating that situational pain is sufficient to increase both smoking urge and immediate smoking behaviour.

As mentioned, latency to smoke provided a measure of ecological validity showing that smoking urge has an effect in terms of generating actual smoking behaviour. However, the cognitive behavioural approach of treatment for other mental health conditions like obsessive compulsive disorder highlight that urge is not equal to action (see Veale, 2007). Likewise, smoking urge does not equate smoking behaviour. Smoking urge was dictated by self-report indicating that participants’ cognitions about their urge were within the level of conscious awareness and clearly preceded smoking behaviour. Response prevention techniques like those used in cognitive behavioural treatment of obsessive compulsive disorder (see Veale, 2007) may be of clinical benefit in interrupting the classically conditioned response of smoking to manage nicotine urge or craving. Future research would be necessary to test this preliminary theoretical position.
The current investigation explored mechanisms and predispositional predictors for the causal relationship between pain and smoking. As hypothesized, urge to smoke fully mediated the relationship between pain and smoking latency. In other words, self-reported smoking urge, as assessed by the QSU-brief, provided a reliable metric for smoking behaviour. As previously mentioned, smoking urge does not equate smoking behaviour; however, it appears to be predictive of smoking behaviour when left unchallenged. There is a substantial amount of research using smoking urge as a dependent variable. A search of MEDLINE with full text (EBSCOhost) using search terms “urge to smoke” OR “urges to smoke” OR “smoking urge” OR “smoking urges” limited to abstracts in academic journals published between 2000 and 2013 (conducted June 8, 2013) yielded 279 relevant papers. For this reason, it is critical to have a reliable proxy for measuring actual smoking behaviour, which is something the 10-item QSU-brief seems to provide.

Pain-induced state emotionality was expected to mediate the relationship between pain and smoking urge; however, unlike results from cold pressor manipulation (Ditre & Brandon, 2008), thermal heat pain induction was not effective in increasing levels of state anxiety or negative affect. Participants who came into the laboratory with higher state emotionality had higher pre- and post-manipulation urge to smoke, but state emotionality itself did not change following the manipulation. Since pain is both a sensory and emotional experience (International Association for the Study of Pain Subcommittee on Taxonomy, 1994, p. S212), this was unexpected; however, recent research indicates that pain induction, though noxious, does not necessarily evoke a detectable affective component (Horn, Blischke, Kunz, & Lautenbacher, 2012). Specifically, when a painful
experience is predictable (i.e., the intensity and course of the stimuli is known), affective pain responses can decrease below the level of detection. It is possible that the thorough informed consent process and the repetition of trials created a ‘predictability’ in which the affective component was no longer detectable. As chronic pain flare-ups are often unpredictable, structuring a component of unpredictability into experimental pain paradigms that seek to assess the effect of pain on smoking would likely provide a truer proxy to a chronic pain experience.

Unexpected pre-manipulation differences between groups were identified in anxiety sensitivity and, most notably, for the ASI-3 social subscale. The reason for these differences despite randomization remains unclear, but may be related to the small sample size. Results of the current investigation indicated that ASI-social was a significant predictor of pain ratings and, thereby, urge to smoke. The involvement of anxiety sensitivity in pain perception and smoking urge and behaviour is consistent with past research (pain perception: Asmundson & Norton, 1995; Asmundson & Taylor, 1996; Ocanez, McHugh, & Otto, 2010; smoking behaviour: Zvolensky et al., 2009). The reason for the specific involvement of solely the social subscale (and not the somatic subscale for instance) is also unclear. Past research indicates that high levels of ASI social are associated with a fear of negative evaluation and a diagnosis of social anxiety disorder (Deacon & Abramowitz, 2006), which suggests social anxiety may be a factor in the observed relationship between pain and smoking. Research indicates that social anxiety, and even sub threshold social anxiety symptoms, are associated with the maintenance of smoking behaviours; specifically, the use of smoking to relieve anxiety and feel comfortable in social situations (Watson, VanderVeen, Cohen, DeMarree, &
Morrell, 2012). The higher the level of social anxiety, the more likely individuals will use smoking as a way to cope across many social domains (Watson et al., 2012). Previous research also indicates that social anxiety plays a role in compounding suffering experienced as a result of pain from injury (Asmundson, Jacobson, Allerdings, & Norton, 1996). Individuals with social anxiety or social anxiety symptoms may be particularly vulnerable to increased smoking urge and behaviour in response to a situation with both a pain induction task and stressful social demands (e.g., unknown environment, facing someone with relative authority, being video recorded). Future investigation is necessary to explore whether the interaction of being observed and also in pain has an additive effect on urge to smoke for those with social anxiety symptoms, as well as the mechanisms involved. As social anxiety was not specifically assessed in the current investigation, inferences to this regard cannot be drawn; however, there may be implications regarding vulnerability for increased dependence for smokers with high levels of ASI-3 social scores within a clinical pain context.

Smoking expectancies related to craving/addiction were a significant independent predictor of smoking urge. According to Goldman (1999), an expectancy is fundamentally a memory, or a template, stored in the nervous system that prepares an organism for future circumstances that are similar to those it has already encountered. Smoking expectancies related to craving reduction are likely founded in repeat experience with the processes involved in biochemical addiction and withdrawal. Therefore, the finding that expectancies related to craving reduction accounted for a significant percentage of variation in urge to smoke was expected. An unexpected finding was that none of the other smoking expectancies proposed were independent
predictors of urge to smoke. Specifically, we anticipated that smoking expectancies related to negative affect reduction and pain coping would have been particularly relevant in the current investigation. In hindsight, there were aspects of the current investigation that may have limited the involvement of the different smoking expectancies. For example, activation of smoking expectancies related to negative affect reduction would have been more likely to occur had the current investigation been successful in increasing state emotionality. Similarly, smoking expectancies related to pain coping may have been activated had the current investigation also been successful in activating affective pain.

4.1 Theoretical Framework

Ditre and Brandon (2008) proposed a simple conceptual framework for the reciprocal relationship between pain and smoking, wherein pain contributes to enhanced smoking motivation, which contributes to greater smoking behaviour, which contributes to more pain. This framework can be built upon by including mediators of pain and smoking urge as well as predictors of pain reactivity (Figure 6). For example, a mediated path was added from pain intensity to smoking urge via pain-induced negative affect (Ditre & Brandon, 2008). As per current findings, a path was added from smoking expectancies to smoking urge. A path from smoking expectancies to pain intensity was also included, as it has been suggested that smoking expectancies may indirectly influence pain reactivity (Ditre et al., 2010). Lastly, a broader category labelled ‘anxiety constructs’ was included in the model as a predictor of pain reactivity. Although, the current investigation measured many anxiety constructs, we were unable to include most in the current models due to limited sample size. As such, the current investigation only
Figure 6: A tentative conceptual framework for the reciprocal relationship between pain and smoking adapted from Ditre and Brandon (2008).
explored ASI-3 – specifically the ASI-3 social subscale – as a predictor of pain ratings; however, the broader category of anxiety constructs was included in the model due to existing literature suggesting the involvement of other predispositional and pain-related anxiety constructs in the pain experience (e.g., anxiety sensitivity, Asmundson & Norton, 1995; Asmundson & Taylor, 1996; Ocanez et al., 2010; pain catastrophizing, Sullivan et al., 1995; for a review see Asmundson & Katz, 2009). Future research is necessary to investigate the involvement of such factors using causal modeling.

Set within a broader context of how acute pain becomes chronic, the proposed cycle (Figure 6), based on current findings and past research, could also be considered from a fear-avoidance perspective (Asmundson et al., 1999; Asmundson et al., 2004; Vlaeyen & Linton, 2000). As previously described, the fear-avoidance models postulate that following injury, the normal healing process generally requires a progressive increase in activity to challenge soft tissue and regain function; however, catastrophic (mis)interpretation of pain and other broad pain-related vulnerabilities can lead to avoidance of activities that might elicit further pain but otherwise promote healing. The fear-avoidance models postulate that avoidance of activities contributes to further disuse, deconditioning, and chronic pain. The proposed cycle (Figure 6) may provide an additional or alternate avoidant coping mechanism beyond avoidance of activities. For example, individuals with predispositional and pain-related anxiety who interpret their pain as threatening may smoke to relieve anxiety and negative mood associated with the pain (Ditre & Brandon, 2008) instead of confronting fear and anxiety related to their pain. Individuals’ beliefs about how smoking will help within the context of pain (e.g., relief of cravings that cannot be managed along with pain, relief of pain itself, relief of anxiety
related to the possibility that others will notice the physically observable signs accompanying their pain) may also contribute to their smoking behaviour in the context of pain (Ditre et al., 2010). Ultimately, smoking leads to further pain (Wei & Hui, 2010) and the cycle continues. Although the described associations are supported within the literature, many were not tested within a causal model in the current investigation; therefore, future research using causal modeling (e.g., SEM with methods that support causal inferences; see Asmundson et al., 2012) is necessary in order to investigate the involvement of smoking within the context of a fear avoidance perspective. Future research is also necessary to investigate whether smoking in response to pain could be a mechanism directly involved with avoidance of activities (e.g., smoking as a means of distraction while engaging in avoidance of activities).

4.2 Limitations and Directions for Future Research

The current investigation had several limitations that provide directions for future research. First, a primary limitation of the current study was generalizability. Experimentally testing the effects of pain on smoking urge and behaviour limited the investigation to experimental/situational pain paradigms that do not capture the complexity of the chronic pain experience. Individuals with chronic pain use coping strategies from many dimensions of human functioning (i.e., cognitive, affective, behavioural, and physiological) to manage pain and its impact on life (Peres & Lucchetti, 2010). Participants in the current investigation comprised relatively healthy individuals who have likely not developed such patterns for coping with the experience of pain. Exploration of the effects of experimental pain on smoking urge and behaviour in a
chronic pain sample may provide a richer context for which to detect mediating and moderating variables.

A second limitation of the current investigation was lack of power due to the modest sample size. Recruitment of smokers who met inclusion criteria within the allotted time frame was a particular challenge of the current investigation and resulted in a sample that was smaller than originally proposed (i.e., a sample of 96); therefore, the power became an issue for planned mediation analyses. Most affected were the analyses that relied on SEM, which is best used with large samples (Loehlin, 2004). As such, we restricted the number of constructs involved in the current models to only a select few proposed in the original models (e.g., smoking expectancies, ASI-3 social). A much larger sample size would be required to test the original model, which was comprised of a greater number of dispositional anxiety and pain-related anxiety variables. One pathway in particular appeared to be impacted by insufficient power; specifically, the main effect of pain ratings on smoking urge (see Model 4 and 5) only trended towards significance after controlling for ASI-3 social and smoking expectancies.

Small sample size can also be problematic for some fit indices (e.g., chi-square test, CFI; Hu & Bentler, 1999). For this reason, multiple fit indices were used to offset issues associated with the use of any one fit index. In addition, bootstrapping with 2000 samples was used to calculate the effects outlined in the hypotheses, providing a way to account for distortions in the distribution that may not be representative of the population (Ader, Mellenbergh, & Hand, 2008).

Demand characteristics were a third potential limitation of the current investigation. Participants may have realized that their urge to smoke was hypothesized
to increase following pain manipulation. In response to these demands, participants may have over- or under-reported smoking urge. Behavioural smoking indices may also have been affected by certain social demands. For example, smoking indoors in public spaces has been banned across Canada since 2010. Participants were informed that smoking was permitted in the experimental room, verbally and visually (i.e., ‘smoking-permitted’ signage, ashtrays); nevertheless, participants may have been hesitant to ‘light up’ even with a substantial urge to smoke. Alternatively, smoking inside may have represented a novel and desirable experience, such that participants may have been eager to take the opportunity to ‘light up’ indoors regardless of their level of smoking urge. The impact of potentially smoking inside may have been compounded by the weather, which happened to be extremely cold during the months of data collection (i.e., average temperatures in January: -14.7°C; February: -10°C; March: -12°C; April: -3.6°C). Future research inquiries may benefit from assessing how individual differences in attitudes related to the social aspects of smoking may be impacting results.

Fourth, the current investigation, though experimental in design, has limitations related to the causal inferences that can be drawn. Relationships between variables, though tested as simple, direct, and uni-directional, likely constitute a complexity of multidirectional and mutually influencing interactions between variables. Longitudinal investigations will be a necessary next step to address questions related to causation.

A final limitation of the current investigation was that the measure of nicotine dependence (FTND) used to determine inclusion into the investigation demonstrated a less than satisfactory reliability (α = .57). Low reliability raises questions regarding the actual level of nicotine dependence within the sample and how this may have affected
results. Future investigations should consider employing more than one measure of nicotine dependence and, preferably, corroborated with an index that does not depend on self-report (i.e., expired breath carbon monoxide levels).

4.3 Conclusions

The current results support and extend those of Ditre and Brandon (2008), indicating that situational pain is sufficient to increase both smoking urge and immediate smoking behaviour. The mechanisms involved in the causal effect of pain on smoking urge and behaviour has been the topic of a vast amount of research. Empirically supported mechanisms include smoking to reduce negative affect induced by pain (Ditre & Brandon, 2008), as well as smoking as a way to relieve pain via the temporary analgesic effect of nicotine (see Ditre, Brandon, Zale, & Meagher, 2011; Shi et al., 2010). Results of the current investigation were interpreted to suggest that even in the absence of pain-induced negative affect and smoking expectancies related to pain reduction, experimental pain still increased smoking urge. Such results suggest that there are likely many reasons why people use cigarette smoking in the context of pain. There appeared to be a particular role for fearing socially observable symptoms of anxiety and smoking expectancies related to craving/addiction; accordingly, comprehensive assessments of anxiety-related vulnerabilities and smoking expectancies that may lead to increased nicotine dependence are necessary in individuals seeking treatment for injury as well as chronic pain. Future research investigating the different types of anxiety variables and smoking expectancies that may exacerbate this causal cycle is necessary. Likewise, inquiry into the types of circumstances (like pain) in which anxiety and smoking expectancies may exacerbate smoking urge and behaviour will likely be central in
smoking cessation success for individuals with these vulnerabilities. In the interim, the proposed model, based on existing research and current results, should direct future investigative inquiry into the causal cycle of nicotine dependence and pain.
5.0 References


doi: 10.3109/08990229209144776


Statistics Canada, Canadian Socio-Economic Information Management System. (2009). *Smokers, by age group and sex* (Table 105-0501, Catalogue Number 82-221-X). Retrieved from http://www40.statcan.ca/l01/cst01/health73a-eng.html


early smoking lapse and relapse during smoking cessation treatment. *Nicotine and Tobacco Research, 11*, 323-331. doi: 10.1093/ntr/ntn037
6.0 APPENDICES
APPENDIX A

University of Regina, Research Ethics Board, Ethics Approval
DATE: December 18, 2012

TO: Holly Parkerson
   Psychology

FROM: Dr. Larena Hoeber
      Chair, Research Ethics Board

Re: Smoking to Cope with Pain: The Motivating Effects of Pain on Smoking Urge and Behaviour (File # 35S1213)

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.

Dr. Larena Hoeber

cc: Dr. Gordon Asmundson - Psychology

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

Anxiety Sensitivity Index - 3
Anxiety Sensitivity Index-3 (ASI-3; Taylor et al., 2007)

Please select the number that best describes how much you agree with each statement. 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.

<table>
<thead>
<tr>
<th></th>
<th>Agree very little</th>
<th>Agree a little</th>
<th>Somewhat agree</th>
<th>Agree a lot</th>
<th>Agree very much</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>It is important to me not to appear nervous.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2.</td>
<td>When I cannot keep my mind on a task, I worry that I may be going crazy.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3.</td>
<td>It scares me when my heart beats rapidly.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4.</td>
<td>When my stomach is upset, I worry that I might be seriously ill.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5.</td>
<td>It scares me when I am unable to keep my mind on a task.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6.</td>
<td>When I tremble in the presence of others, I fear what people might think of me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7.</td>
<td>When my chest feels tight, I get scared that I won't be able to breathe properly.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8.</td>
<td>When I feel pain in my chest, I worry that I'm going to have a heart attack.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9.</td>
<td>I worry that other people will notice my anxiety.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10.</td>
<td>When I feel “spacey” or spaced out I worry that I may be mentally ill.</td>
<td>0</td>
<td>1</td>
<td>2</td>
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</tr>
<tr>
<td>11.</td>
<td>It scares me when I blush in front of people.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12.</td>
<td>When I notice my heart skipping a beat, I worry that there is something seriously wrong with me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>13.</td>
<td>When I begin to sweat in a social situation, I fear people will think negatively of me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>14.</td>
<td>When my thoughts seem to speed up, I worry that I might be going crazy.</td>
<td>0</td>
<td>1</td>
<td>2</td>
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<tr>
<td>15.</td>
<td>When my throat feels tight, I worry that I could choke to death.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<td>16.</td>
<td>When I have trouble thinking clearly, I worry that there is something wrong with me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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</tbody>
</table>
Appendix C

Brief Questionnaire of Smoking Urges
Brief Questionnaire of Smoking Urges (QSU-Brief, Toll, Kutuak, & McKee, 2006)
Indicate how much you agree or disagree with each of the following statements by making one of the circles between STRONGLY DISAGREE and STRONGLY AGREE. The closer you place your mark to one end or the other indicates the strength of your agreement or disagreement. We are interested in how you are thinking and feeling right now as you are filling out the questionnaire.

1. I have a desire for a cigarette right now
   STRONGLY DISAGREE
   O O O O O O O O STRONGLY AGREE

2. Nothing would be better than smoking a cigarette right now
   STRONGLY DISAGREE
   O O O O O O O O STRONGLY AGREE

3. If it were possible I would probably smoke now
   STRONGLY DISAGREE
   O O O O O O O O STRONGLY AGREE

4. I could control things better right now if I could smoke
   STRONGLY DISAGREE
   O O O O O O O O STRONGLY AGREE

5. All I want right now is a cigarette
   STRONGLY DISAGREE
   O O O O O O O O STRONGLY AGREE

6. I have an urge for a cigarette
   STRONGLY DISAGREE
   O O O O O O O O STRONGLY AGREE

7. A cigarette would taste good now
   STRONGLY DISAGREE
   O O O O O O O O STRONGLY AGREE

8. I would do almost anything for a cigarette now
   STRONGLY DISAGREE
   O O O O O O O O STRONGLY AGREE

9. Smoking would make me less depressed
   STRONGLY DISAGREE
   O O O O O O O O STRONGLY AGREE

10. I am going to smoke as soon as possible
    STRONGLY DISAGREE
    O O O O O O O O STRONGLY AGREE
Appendix D

Brief Smoking Consequences Questionnaire - Adult
**Brief Smoking Consequences Questionnaire – Adult** (SCQ-A; Copeland, Brandon, & Quinn, 1995)

Instructions: This questionnaire is designed to assess beliefs people have about the consequences of smoking a cigarette. Below is a list of statements about smoking. We would like you to rate how LIKELY or UNLIKELY you believe each consequence is for you when you smoke. If the consequence seems UNLIKELY to you, circle a number from 0-4. If the consequence seems LIKELY to you, circle a number from 5-9. That is if you believe the consequence would never happen, circle 0; if you believe a consequence would happen every time you smoke, circle 9. Use the guide below to aid you further. For example, if a consequence seems completely likely to you, you would circle 9. If it seems a little unlikely to you, you would circle 4.

<table>
<thead>
<tr>
<th>Statement</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
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<tr>
<td>When I'm angry, a cigarette can calm me down.</td>
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<td>Smoking a cigarette energizes me.</td>
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<td>If I were to experience pain, a cigarette would help reduce it.</td>
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<td>I enjoy the taste sensations while smoking.</td>
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<td>I feel more at ease with other people if I have a cigarette.</td>
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<td>Smoking keeps my weight down.</td>
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<td>Nicotine “fits” can be controlled by smoking.</td>
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<td>If I have nothing to do, a smoke can help kill time.</td>
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<td>Smoking would ease my pain if I were hurting.</td>
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<td>I look ridiculous while smoking.</td>
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<td>Smoking calms me down when I feel nervous.</td>
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<td>The more I smoke, the more I risk my health.</td>
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<td>When I smoke, the taste is pleasant.</td>
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<td>I feel like part of a group when I'm around other smokers.</td>
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<td>If I hurt myself, I would feel less pain if I could smoke.</td>
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<td>Cigarettes keep me from eating more than I should.</td>
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<td>Smoking irritates my mouth and throat.</td>
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<td>People think less of me if they see me smoking.</td>
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<td>If I'm feeling irritable, a smoke will help me relax.</td>
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<td>A cigarette can give me energy when I'm bored and tired.</td>
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<td>When I feel pain, a cigarette can really help.</td>
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<td>By smoking I risk heart disease and lung cancer.</td>
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<td>I will enjoy the flavor of a cigarette.</td>
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<td>Smoking helps me enjoy people more.</td>
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<td>Smoking helps me control my weight.</td>
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<td>Smoking will satisfy my nicotine cravings.</td>
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<tr>
<td>If I hurt myself, I could cope with the pain without smoking.</td>
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<td>My throat burns after smoking.</td>
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<tr>
<td>When I'm alone, a cigarette can help me pass the time.</td>
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<tr>
<td>Smoking makes me seem less attractive.</td>
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<td>I feel like smoking would help me cope with pain.</td>
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</tbody>
</table>

**Note:** The table provides a sample of statements and their corresponding ratings. The full questionnaire contains a wide range of statements reflecting different aspects of smoking consequences.
Appendix E

Distress Tolerance Scale
**Distress Tolerance Scale - Reverse Coded (Simons & Gahee, 2005)**

Think of times that you feel distressed or upset. Select the item from the menu that best describes your beliefs about feeling distressed or upset.

<table>
<thead>
<tr>
<th></th>
<th>Strongly Disagree</th>
<th>Mildly Disagree</th>
<th>Agree and Disagree Equally</th>
<th>Mildly Agree</th>
<th>Strongly Agree</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Feeling distressed or upset is unbearable to me.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>2. When I feel distressed or upset, all I can think about is how bad I feel.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>3. I can’t handle feeling distressed or upset.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>4. My feelings of distress are so intense that they completely take over.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>5. There’s nothing worse than feeling distressed or upset.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>6. I can tolerate being distressed or upset as well as most people.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>7. My feelings of distress or being upset are not acceptable.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>8. I’ll do anything to avoid feeling distressed or upset.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>9. Other people seem to be able to tolerate feeling distressed or upset better than I can.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>10. Being distressed or upset is always a major ordeal for me.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>11. I am ashamed of myself when I feel distressed.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>12. My feelings of distress or being upset scare me.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>13. I’ll do anything to stop feeling distressed or upset.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>14. When I feel distressed or upset, I must do something about it immediately.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>15. When I feel distressed or upset, I cannot help but concentrate on how bad the distress actually feels.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>
Appendix F

Intolerance of Uncertainty Scale, Short Form
**Intolerance of Uncertainty, Short Form** (IUS-12; Carleton, Norton, & Asmundson, 2007)

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

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Unforeseen events upset me greatly.</td>
<td>Not at all characteristic of me</td>
<td>A little characteristic of me</td>
<td>Somewhat characteristic of me</td>
<td>Very characteristic of me</td>
<td>Entirely characteristic of me</td>
</tr>
<tr>
<td>2.</td>
<td>It frustrates me not having all the information I need.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>3.</td>
<td>Uncertainty keeps me from living a full life.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>4.</td>
<td>One should always look ahead so as to avoid surprises.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>5.</td>
<td>A small unforeseen event can spoil everything, even with the best of planning.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>6.</td>
<td>When it’s time to act, uncertainty paralyses me.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>7.</td>
<td>When I am uncertain I can’t function very well.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>8.</td>
<td>I always want to know what the future has in store for me.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>9.</td>
<td>I can’t stand being taken by surprise.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>10.</td>
<td>The smallest doubt can stop me from acting.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>11.</td>
<td>I should be able to organize everything in advance.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>12.</td>
<td>I must get away from all uncertain situations.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>
Appendix G

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.

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

Pain Catastrophizing Scale
Pain Catastrophizing Scale (PCS; Sullivan et al., 1995)

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

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

0 – not at all 1 – to a slight degree 2 – to a moderate degree 3 – to a great degree 4 – all the time

1 ☐ I worry all the time about whether the pain will end.
2 ☐ I feel I can’t go on.
3 ☐ It’s terrible and I think it’s never going to get any better.
4 ☐ It’s awful and I feel that it overwhelms me.
5 ☐ I feel I can’t stand it anymore.
6 ☐ I become afraid that the pain will get worse.
7 ☐ I keep thinking of other painful events.
8 ☐ I anxiously want the pain to go away.
9 ☐ I can’t seem to keep it out of my mind.
10 ☐ I keep thinking about how much it hurts.
11 ☐ I keep thinking about how badly I want the pain to stop.
12 ☐ There’s nothing I can do to reduce the intensity of the pain.
13 ☐ I wonder whether something serious may happen.
Appendix I
Positive and Negative Affect Schedule
**Positive and Negative Affect Schedule** (PANAS; Watson, Clark, & Tellegen, 1988)

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you felt this way **during the past week**. Use the following scale to record your answers.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>very slightly or not at all</td>
<td>a little</td>
<td>moderately</td>
<td>quite a bit</td>
<td>extremely</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>interested</td>
<td>irritable</td>
<td>alert</td>
<td>__</td>
<td>___</td>
</tr>
<tr>
<td>distressed</td>
<td>__</td>
<td>____</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>excited</td>
<td>___</td>
<td>__</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>upset</td>
<td>excited</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>strong</td>
<td>strong</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>guilty</td>
<td>guilty</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>scared</td>
<td>scared</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>hostile</td>
<td>__</td>
<td>____</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>enthusiastic</td>
<td>____</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>proud</td>
<td>___</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
</tbody>
</table>

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you feel this way **at this moment**. Use the following scale to record your answers.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>very slightly or not at all</td>
<td>a little</td>
<td>moderately</td>
<td>quite a bit</td>
<td>extremely</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>interested</td>
<td>irritable</td>
<td>alert</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>distressed</td>
<td>___</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>excited</td>
<td>___</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>upset</td>
<td>___</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>strong</td>
<td>___</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>guilty</td>
<td>___</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>scared</td>
<td>___</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>hostile</td>
<td>___</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>enthusiastic</td>
<td>___</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>proud</td>
<td>___</td>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
</tbody>
</table>
Appendix J

State-Trait Anxiety Inventory
State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Luschene, Vagg, & Jacobs, 1983)

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Appendix K

Numerical Rating Scale
Numerical rating scale (NRS; Dworkin et al., 2005)

Please put a circle around the number that best describes your pain, at its worst, since beginning this pain manipulation trial.

*Note: 0 means 'No pain' and 10 means 'Pain as bad as you can imagine'

No pain  0  1  2  3  4  5  6  7  8  9  10  Pain as bad as you can imagine