VALIDATION OF THE SENSITIVITY TO PAIN TRAUMATIZATION SCALE

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Abstract

The present dissertation examines the psychometric properties of the Sensitivity to Pain Traumatization Scale (SPTS-12), a measure developed to assess the cognitive, emotional, behavioural, and somatic responses to pain that are similar to a traumatic stress response. The literature review provides a description of the definition, models, and burden of chronic pain and trauma, as well as a discussion of the high rates of comorbidity between chronic pain and trauma. Next, common pain-related anxiety measures are described followed by a summary of the development of the SPTS-12. Three studies are presented that examine the psychometric properties of the SPTS-12. Study 1 evaluates the factor structure, reliability, and validity of the SPTS-12 in a sample of 823 undergraduate students who were pain-free or reported experiencing ongoing pain. For both groups, the one-factor model demonstrated adequate overall fit and the SPTS-12 total score showed excellent reliability and good convergent validity with a measure of trauma symptoms, with mixed findings regarding the divergent validity of the SPTS-12 when examined against a measure of depressive symptoms. Study 2 explores the factor structure, reliability, and validity of the SPTS-12 in a clinical sample of 180 patients receiving care in an outpatient multidisciplinary service designed to help prevent the development of chronic postsurgical pain. Confirmatory factor analysis supported the one-factor model of the SPTS-12, with evidence of excellent internal consistency reliability. The SPTS-12 demonstrated good convergent validity, but divergent validity was not supported. Study 3 uses latent class mixed models to represent trajectories of SPTS-12 scores in a clinical sample of 361 patients after surgery. The optimally-fitting model consisted of five SPTS-12 trajectories, three of which were characterized by significantly decreasing scores over time. Analysis of pain-related outcomes predicted by SPTS-12 trajectories provide evidence of criterion validity of the SPTS-12. Across

all three studies, the results indicate that the SPTS-12 provides a way to more directly measure traumatization that individuals may experience in response to pain, which may contribute to our understanding of why trauma and pain co-occur so frequently. Given the high incidence of pain and trauma, as well as the established efficacy of psychotherapy in treating pain after surgery, the present results suggest that tailoring treatment to better address trauma-specific symptoms may help improve pain management treatment strategies. Limitations include several large residual correlations between some items of the SPTS-12 in Study 1. Furthermore, in all three studies, the samples were highly heterogeneous and may not have identified differences among distinct subsamples. Additionally, missing data may have contributed to a systematic bias that only captures participants who provided adequate responses. Possible future directions include developing alternate wording for the item with the poorest fit on the SPTS-12, evaluating the concurrent validity of the SPTS-12 by examining its relationship with clinically relevant mental health diagnoses, and validating the SPTS-12 in different patient and community populations. In summary, the present dissertation provides evidence of strong psychometric properties of the SPTS-12 and encourages ongoing refinement and validation of the scale.

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Chapter 1: Introduction & Literature Review

There is growing evidence that psychological variables are linked to the development, maintenance, and experience of pain (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). These associations are especially evident in the high rate of comorbidity between chronic pain and symptoms of posttraumatic stress, as the incidence of having both disorders consistently exceeds the likelihood of having one disorder alone (Asmundson & Katz, 2009). Numerous theoretical models have been developed to explain this co-occurrence, with some suggesting that they have mutually maintaining factors (i.e., the disorders may be maintained by the symptoms of the other disorder), shared vulnerabilities (i.e., individuals who develop both disorders share risk factors that increase their overall vulnerability), or a combination of these theories (Asmundson, Coons, Taylor, & Katz, 2002; Sharp & Harvey, 2001; Turk, 2002). Despite this established relationship, only recently have researchers started to investigate the psychometric properties of a measure that is designed to assess the traumatic responses that individuals may experience in relation to pain.

The present dissertation examines the validity of a new scale for assessing the propensity of individuals to develop a traumatic stress response to pain. In the introduction and literature review, pain is defined and the substantial burden and prevalence of pain are discussed. This discussion is followed by a description of historical and modern understandings of pain. Trauma is subsequently defined with the prevalence and burden of trauma discussed. Next, models to explain the high comorbidity rate between chronic pain and posttraumatic stress disorder are described. Methods of assessing both pain and trauma are described and the initial development of the new questionnaire, the Sensitivity to Pain Traumatization Scale, is discussed. Three

studies are then described that examined the psychometric properties of the Sensitivity to Pain Traumatization Scale.

Chronic Pain

Definition. The International Association for the Study of Pain (IASP) presently defines pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (Merskey & Bogduk, 1994, p. 210). This definition is accompanied by a note that emphasizes that pain is a subjective experience that can be present even if an individual is unable to communicate verbally. In order to be considered pain, the sensations must be experienced to be unpleasant. Pain can occur in the absence of tissue damage and should be treated as pain if the individual subjectively states it to be pain. In 2016, a revision to the definition of pain was suggested by Williams and Craig: “pain is a distressing experience associated with actual or potential tissue damage with sensory, emotional, cognitive, and social components” (p. 2420). This suggested change further acknowledges that pain is a biopsychosocial experience and reflects a more current understanding of pain. More recently, IASP proposed a new definition of pain: "an aversive sensory and emotional experience typically caused by, or resembling that caused by, actual or potential tissue injury" (2019). The accompanying note was also revised to be shorter and clearer. The new definition has not yet been adopted by IASP. In August 2019, the IASP task force working on the definition of pain published the new definition online, welcoming the feedback of the public and scientific community before providing recommendations that incorporate the public’s feedback.

As of 2018, chronic pain is now also more comprehensively described in the newest edition of the International Classification of Diseases (ICD-11) published by the World Health Organization. The challenges in defining and classifying chronic pain were reflected in the lack

of a systematic and adequate representation of chronic pain diagnoses in previous editions of the ICD (Treede et al., 2019). This led to inadequate epidemiological estimates of the prevalence and impact of pain, which have hindered pain management efforts. To address this gap, a Classification of Pain Diseases Task Force was created by IASP that contributed to the pain diagnoses developed by the World Health Organization for the newest working draft of the ICD-11 released in June 2018 (World Health Organisation, 2018). The definition of chronic pain in the newest ICD-11 describes pain as being considered chronic if it re-occurs or persists for three or more months (Treede et al., 2019). The over-arching definition of chronic pain is divided into two subcategories: chronic primary pain and chronic secondary pain syndromes. Chronic primary pain conditions are considered to be a disease in their own right, with five types described: chronic widespread pain, complex regional pain syndrome, chronic primary headache or orofacial pain, chronic primary visceral pain, and chronic primary musculoskeletal pain. Chronic secondary pain syndromes are described as pain that develops as a symptom that follows another disease or condition, with six types described: chronic cancer-related pain, chronic postsurgical or posttraumatic pain, chronic neuropathic pain, chronic secondary headache or orofacial pain, chronic secondary visceral pain, and chronic secondary musculoskeletal pain. The development of new definitions and classifications of pain reflect the evolving understanding of the complexity of chronic pain.

More generally, pain has also been defined according to its physiology, time course, and syndrome, although it can be difficult to capture the complexities of a painful experience in a single category. The purpose of pain classification is to improve communication between patients and healthcare professionals in order to facilitate the best possible diagnosis and management of pain.

Pathophysiological classification. Pain has been described as having three major components: nociceptive, neuropathic, and inflammatory inputs. Pain arising from predominantly nociceptive inputs refers to the immediate sensory response to an unpleasant environmental stimulus, such as a pinprick, biting one's tongue, or scraping one's knee. This input triggers electrical activity in myelinated Aδ-fibers and unmyelinated C-fibers that travel along peripheral pathways to the central nervous system (Costigan, Scholz, & Woolf, 2009). The signal then travels up the spinal cord to the brain stem and thalamus, where it is processed and perceived as pain in the cerebral cortex. Pain arising from nociceptive inputs is generally considered adaptive: if a noxious stimulus is encountered, it is quickly perceived to be unpleasant and therefore provokes a behavioural response to withdraw from the stimulus, which can help prevent or worsen tissue damage (Woolf, 2010).

Pain arising from predominantly inflammatory inputs occurs when swelling is evident following tissue injury, such as when the skin around a mild burn or a cut becomes inflamed, red, and sensitive. As a result, nociceptive neurons become sensitized through the release of cytokines, chemokines, and other chemical mediators causing peripheral amplification, which triggers central amplification that travels through the spinal cord to the brain (Costigan et al., 2009). The inflamed region and the surrounding area consequently become sensitized, with sensations that were not previously painful becoming painful or becoming more painful. This increased sensitivity may facilitate healing and repair by discouraging over-use that may continue to damage the area. Although pain arising from inflammation inputs often subsides when the tissue heals, pain can persist with some conditions where tissue healing does not take place and inflammation continues to occur (e.g., rheumatoid arthritis; Michaud, Bombardier, & Emery, 2007).

Pain arising from predominantly neuropathic inputs has been defined by IASP as “pain caused by a lesion or disease of the peripheral somatosensory nervous system” (International Association for the Study of Pain, 2012). Lesions may be acquired peripherally (e.g., due to mechanical trauma, metabolic lesions, or disease) or centrally (e.g., due to stroke or spinal cord injury). The damage disrupts the signals that the nerve fibers send to pain centers, which may cause increased neural firing, reduced inhibitory circuits at the dorsal horn or brain stem, and increased sensitivity of spinal neurons (Yaksh, 1999). The hallmark feature of pain arising from neuropathic inputs is that it is caused by structural and functional cortical and neuronal changes through plasticity, with pain enduring after tissue damage has healed (Costigan et al., 2009). In contrast to pain that arises from nociceptive and inflammatory inputs, neuropathic pain is not considered adaptive, but rather it is associated with the maintenance of abnormal amplification (Yaksh, 1999).

Pain duration. Pain can be further classified according to its duration. Pain is defined as acute if it occurs for under 1 to 6 months (Merskey & Bogduk, 1994), which is considered the range of time when normal tissue healing should take place. This time range is broad in order to accommodate different expected healing times associated with different types of injuries, although there is no consensus on what is considered to be "normal healing" for a given injury. Nevertheless, it is the most common way of classifying and contrasting acute pain from chronic pain. The onset of acute pain may be more sudden and it is frequently easier to identify the source of the pain, although it can be of any pathological origin (i.e., nociceptive, inflammatory, or neuropathic; Carr & Goudas, 1999). Causes of acute pain may include cuts and bruises, broken bones, or surgery.

Pain is considered chronic when it "persists past the normal time of healing" (Merskey & Bogduk, 1994, p. xi). It is recognized that various pain conditions and injuries have different estimates for the duration of healing, although the IASP and World Health Organization consider pain lasting longer than three months to be an appropriate distinction between acute and chronic pain for many types of non-malignant pain (Merskey & Bogduk, 1994; World Health Organisation, 2018). After three months, tissue damage has had time to heal and inflammation to subside. When pain persists beyond this time, it is considered more pathological and maladaptive, with the source of pain more attributable to increased sensitization and brain plasticity, showing that chronic pain is a "disease of the nervous system" (Woolf, 2010, p. 3743). For this reason, it can become more difficult to identify a specific pathophysiological origin for chronic pain and it is important that the enduring symptoms continue to be recognized as pain, rather than associated with psychopathology (Gagliese & Katz, 2000). If pain is associated with psychopathology, it can promote distrust in the patient-provider relationship, with the implicit assumption that the pain the patient is experiencing is "all in their head" with no organic cause (Katz, Rosenbloom, & Fashler, 2015). As our understanding of pain becomes more advanced, many previously unexplained pain syndromes can now be explained with changes related to malfunctions in central nervous system pain mechanisms, supporting the position that pain should be treated as pain even without an identifiable pathophysiological origin rather than assuming that the pain has a psychological origin. Additionally, it is noteworthy that for some chronic pain conditions, "normal healing" does not take place: for example, in the case of the recurrent and persistent inflammation associated with rheumatoid arthritis (Costigan et al., 2009).

In summary, pain duration is frequently used as a method for classifying pain as either acute or chronic, although this definition can be problematic due to the large range of healing

time for injuries, the inability to identify a specific pathophysiological origin for pain, and the absence of an expected range of normal healing for chronic conditions.

Pain syndromes. Pain can be further classified by syndrome, distinguished by a set of co-occurring symptoms that includes the site, duration, quality, and pattern of pain (Merskey & Bogduk, 1994). The Task Force on Taxonomy of the IASP (1994) established one of the first comprehensive classification systems for pain syndromes, which was organized according to site of pain and then further described the main features of the syndrome, including the average age of onset, duration, severity, quality, typical course, pathology, diagnostic criteria, and treatment. This form of classification facilitates communication between pain sufferers and their healthcare providers and assists with research and knowledge translation of various conditions. Due to the complexity of pain, IASP emphasizes that definitions of syndromes must not be held too rigidly as an exhaustive description of all conditions and to permit for ongoing advances in clinical practice and research.

Postsurgical pain. Pain can be further classified according to the cause of pain when this information is available, such as related to a specific trauma (e.g., car accident) or illness (e.g., cancer pain). In the last 20 years, there has been increasing recognition that pain may develop following surgery (Crombie, Davies, & Macrae, 1998; Macrae, 2008) and has been aptly referred to as “chronic postsurgical pain”. Since that time, chronic postsurgical pain has been generally described as pain that persists after surgery for at least two months, with other possible causes of pain ruled out (including pre-existing painful conditions; Katz & Seltzer, 2009; Macrae & Davies, 1999). More recently, Woolf (2010) and Weinrib et al. (2017) have suggested several changes to the criteria for pain to be considered chronic post-surgical pain. They describe chronic post-surgical pain as the pain that can develop following a surgical procedure or that can

increase following a procedure that lasts for at least 2 months and negatively impacts an individual's health quality of life. This time range was proposed so that it better captures typical post-surgical healing and permits a variable healing time to better account for complications such as infections that may be related to surgical procedures. That is, “normal” post-surgical healing may vary for different surgical procedures. The authors also caution against using only time as the primary criterion to define postsurgical pain, since time alone does not capture the complex multidimensional risk and protective factors that contribute to the development of postsurgical pain. The authors furthermore propose that the pain must be continuous from surgery or it may develop after a pain-free period, and that the location of the pain is the site of the surgery, projected pain related to the surgery site, or projected pain related to a dermatome. Finally, the new definition maintains that the pain cannot have another cause, such as cancer pain, infection, or another condition.

In 2018, the World Health Organization published a working draft of the newest edition of the ICD-11 that included a new definition of chronic postsurgical and posttraumatic pain similar to those described above (Schug et al., 2019). It defines chronic postsurgical and posttraumatic pain as "pain developing or increasing in intensity after a surgical procedure or a tissue injury (involving any trauma including burns) and persisting beyond the healing process, i.e. at least 3 months after surgery or tissue trauma. The pain is either localized to the surgical field or area of injury, projected to the innervation territory of a nerve situated in this area, or referred to a dermatome” (World Health Organisation, 2018). Similar to the definitions described above, a further requirement is that other potential causes of pain must be excluded. The definition acknowledges that pain may be neuropathic in nature, noting that it should be highly probable that the cause of injury was the surgery itself.

Pain after surgery is common: approximately 10 to 50% of patients who undergo surgical procedures go on to develop chronic postsurgical pain (Kehlet, Jensen, & Woolf, 2006). The transition from acute to chronic pain is a complex process involving several psychosocial risk factors, including catastrophizing, state anxiety, and social support (Katz & Seltzer, 2009). The pathophysiological origin of chronic postsurgical pain is frequently considered neuropathic, and is characterized by sensations of burning, shooting, or stabbing pain that can be experienced as hyperalgesia, where an individual reports a disproportionate amount of pain intensity or that the pain persists for a disproportionate amount of time in response to mild or even no stimuli (Gottrup, Andersen, Arendt-Nielsen, & Jensen, 2000).

Burden of pain. Pain is a major public health concern. It is estimated that nearly 20% of Canadian adults and 15% of American adults experience chronic pain (Hardt, Jacobsen, Goldberg, Nickel, & Buchwald, 2008; Schopflocher, Taenzer, & Jovey, 2011). Pain is a commonly cited reason for seeking medical attention, being the primary complaint for 40% of individuals in a primary care setting (Mäntyselkä et al., 2001) and for 52.2% of visits to the emergency room (Cordell et al., 2002). Even when pain itself is not the primary complaint, patients report that two out of the top reasons to visit their doctor (back problems and osteoarthritis/joint disorders) involve pain (Sauver et al., 2013). Pain contributes to diminished quality of life (Dillie, Fleming, Mundt, & French, 2008) and is associated with poorer psychological functioning, with higher rates of comorbid depression (Arnow et al., 2006; Bair, Robinson, Katon, & Kroenke, 2003) and anxiety (Asmundson & Katz, 2009; Bair et al., 2003). Pain can negatively impact social relationships (Turk et al., 2008) and marital relationships (Cano, Gillis, Heinz, Geisser, & Foran, 2004). Chronic pain contributes to poorer general health

and is associated with higher rates of disability and longer healing time (Tripp, VanDenKerkhof, & McAlister, 2006).

In addition to the burden of pain on the individual and their families, pain places substantial strain on the healthcare system. In the United States alone, it is estimated that direct costs of medical services are between $560 to $635 billion each year (Gaskin & Richard, 2012), exceeding the costs for other chronic health conditions such as cancer, diabetes, and heart disease. Furthermore, it is estimated that in the United States, an additional $61 billion is lost each year for active workers due to absenteeism and reduced productivity (Stewart, Ricci, Chee, Morganstein, & Lipton, 2003). To better understand the economic burden of pain in Ontario, Hogan, Taddio, Katz, Shah, and Krahn (2016) estimated the annual per-person cost of medical expenses related to pain. The annual incremental cost of managing chronic pain was estimated to be $1742 per person when compared to matched controls that did not experience pain. The incremental cost was even higher for matched controls with severe pain and with activity limitations. Furthermore, individuals with chronic pain matched to controls demonstrated that those with pain have a higher burden of disease as measured with an index of health utilities compared to many other chronic conditions, including heart disease, diabetes, and cancer (Hogan et al., 2017).

Taken together, these findings point to the substantial costs of pain at the individual, social, and societal levels and speak to the disabling effects of pain and the challenges of effective treatment (Gatchel et al., 2007).

Models of pain. How we understand pain has changed over time, growing more complex to better incorporate the multidimensional aspects of the pain experience. Descartes (1644) developed one of the earliest theories of pain which is known as specificity theory. He proposed

that pain was the direct result of contact with a noxious stimulus; for example, if a hand accidentally made contact with a flame, this would trigger a pain signal that would travel up to the brain and trigger a reflexive response, such as the withdrawal of the hand from the fire. Descartes proposed that pain intensity has a one-to-one correspondence to pain stimulus and the brain is not actively involved in the experience of pain. This model became popular because it is intuitive and it is supported by daily experiences with pain. However, this theory did not account for how painful experiences can vary as a result of experience, context, affect, or culture. Furthermore, it did not offer an explanation for how some individuals can experience pain in a part of the body that has been removed, as with phantom limb pain, or how others report no subjective discomfort when engaging in an otherwise painful activity, such as walking across coals.

To address these limitations, Melzack and Wall (1965) developed the gate control theory of pain. According to the gate control theory, three spinal cord systems, the substantia gelatinosa in the dorsal horn, the first central transmission (T) cells in the dorsal horn, and the dorsal-column fibers projecting to brain, are imperative for the transmission of nociceptive inputs throughout the body. They proposed that the substantia gelatinosa serves as a "gate control system" that can disrupt neuronal signals from transmission in the dorsal column. Then, the neural mechanisms are activated by T cells to determine if the noxious inputs continue to be transmitted to the brain, triggering the perception and response to the stimulus, or inhibited in the spinal cord. Lastly, the afferent patterns are processed by the brain and can modulate the gate control system based on a variety of factors, including mood, experience, and context (Melzack & Katz, 2013). In contrast to the pain specificity model, this model shows that the brain is not a passive recipient of neuronal messaging, but rather contributes actively to the experience of pain.

In this way, the brain, not the stimulation, can determine the degree of pain experienced. Although controversial at first, the gate control theory of pain is now widely accepted and subsequent research has found support for many of the propositions made in the theory (e.g., Dickenson, 2002; Guo & Hu, 2014; Katz & Rosenbloom, 2015; Mendell, 2014; Werner & Kongsgaard, 2014).

Measurement of pain. The experience of pain is subjective – it can occur in the absence of an identifiable pathophysiological cause and an experience with a pathophysiological cause should not be considered painful unless it is accompanied by the subjective appraisal of unpleasantness (Merskey & Bogduk, 1994). Therefore, as the definition of pain states that it is always a "psychological state" (Merskey & Bogduk, 1994), it is not surprising that the most common method of assessing pain is through asking the individual to self-report on the level, degree, and nature of their pain. To measure intensity, numeric rating scales and visual analogue scales are two of the most common methods (Williamson & Hoggart, 2005). The numerical rating scale involves asking the individual to rate the severity of their pain on a scale, with the lowest number (e.g., "0") indicating that the absence of pain and the highest number (e.g., "10" or "100") being the worst possible pain imaginable. Visual analogue scales ask individuals to rate pain in the same manner, although pain severity is marked along a line (frequently 100 mm long), with similar verbal anchors (e.g., "no pain" to "worst pain imaginable"; Williamson & Hoggart, 2005).

Although the numeric rating scale and the visual analogue scale are widely used, they are limited in that they assume that pain is unidimensional and they do not capture other sensory, temporal, cognitive, emotional, and social qualities of the pain experience (Katz & Melzack, 1999). Many of these limitations are addressed in other pain assessment measures, including the

McGill Pain Questionnaire (Katz & Melzack, 2011; Melzack, 1975), Leeds Assessment of Neuropathic Symptoms and Signs (LANSS) Pain Scale (Bennett, 2001), and Brief Pain Inventory (Cleeland & Ryan, 1994). Importantly, some individuals are unable to use self-report due to a limited ability to communicate, such as infants, individuals with developmental disabilities, and individuals suffering from dementia. In these cases, observational pain measures can be used to estimate pain severity to help provide improved pain management for these populations (Hadjistavropoulos & Craig, 2002).

Trauma

Definition. Exposure to a traumatic event is common across the lifespan. In a large, global examination of traumatic event exposure conducted across 24 countries with 68,864 adults, over 70% of respondents endorsed exposure (Benjet et al., 2016). The most common traumatic events included witnessing the death of another individual, the unexpected death of a family member or friend, being robbed, being in a serious motor vehicle accident, and experiencing an injury or illness that is life-threatening (Benjet et al., 2016). The exposure rate was reported to be even higher in a sample of 2181 individuals living in the Detroit Area, which showed that nearly 90% of respondents endorsed exposure to a trauma (Breslau et al., 1998). In response to experiencing a traumatic event, some individuals go on to develop adverse psychological effects. The most well-investigated resultant syndrome is Posttraumatic Stress Disorder (PTSD).

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) describes PTSD as the development of specific symptoms following a traumatic event. The first criterion is that the individual was exposed to trauma that includes witnessing death, threatened death, or sexual violence. This criterion may

occur through direct exposure to a trauma, witnessing a trauma, indirectly experiencing a trauma (i.e., learning of a trauma that a close other endured if it was violent or accidental), or through repeated or severe indirect exposure related to trauma (e.g., as with first responders). Furthermore, the person must experience one or more intrusive or reexperiencing symptoms related to the trauma (e.g., distressing memories, dreams, dissociative reactions of reliving the trauma, marked distress following exposure to cues related to the trauma, or significant physiological reaction to cues related to the trauma), evidence of persistent avoidance of reminders of the trauma (e.g., of specific cognitions or of specific cues), two or more negative changes in cognitions and affect related to the trauma (e.g., inability to remember aspects of the trauma, persistent negative beliefs, blaming self for trauma, negative emotional state, anhedonia, feeling detached from others, or inability to experience emotions), and two or more changes in arousal or reactivity related to the trauma (e.g., persistent irritable behaviour, reckless behaviour, hypervigilance, excessive startle response, difficulty concentrating, sleep disturbance). To be diagnosed as PTSD, the above symptoms are required to be present for at least one month and to cause clinically significant distress or impairment in functioning. Furthermore, the symptoms may not be attributable to another medical condition or the effects of a substance.

Burden of trauma. Not everyone who is exposed to a trauma goes on to develop PTSD. As a part of the 2012–2013 National Epidemiologic Survey on Alcohol and Related Conditions-III, the Alcohol Use Disorder and Associated Disabilities Interview Schedule-5 was administered to a sample of 36,309 adults to measure rates of PTSD (Goldstein et al., 2016). The authors found that 6.1% of adults endorsed criteria meeting lifetime prevalence of PTSD. This estimate increases when considering the lifetime prevalence for individuals who have engaged in military service. For example, 30.9% of male and 26.9% of female veterans of the Vietnam War met

criteria for PTSD (Kulka et al., 1990). Comorbidities with other psychiatric disorders are common with PTSD: in the early 1990's, the National Comorbidity Survey (NCS) showed that in a sample of individuals with PTSD, 44% of women and 59% of men met criteria for three or more additional psychiatric disorders (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Unsurprisingly, PTSD negatively impacts quality of life (Rapaport, Clary, Fayyad, & Endicott, 2005) and can lead to avoidance of activities (Nemeroff et al., 2006), social isolation, and impairment in interpersonal functioning and relationships (Beck, Grant, Clapp, & Palyo, 2009). Greenberg et al. (1999) estimated that the annual cost of anxiety disorders, including PTSD, in the United States in 1990 was $42.3 billion dollars. Furthermore, PTSD was estimated to have the highest service health service use of all anxiety disorders (Greenberg et al., 1999).

Comorbidity of PTSD and chronic pain. Individuals with PTSD are more likely than individuals without a diagnosis of PTSD to report physical health difficulties (Beckham et al., 1997), disability related to pain, chronic pain, and reduced functional ability. Pain is one of the most reported comorbid symptoms for individuals with PTSD (McFarlane, Atchison, Rafalowicz, & Papay, 1994). In a sample of individuals suffering from PTSD six months following a traffic accident, 37% reported continuing to experience pain compared to 8% of individuals who were not suffering from PTSD (Chossegros et al., 2011). Chronic pain is even more commonly reported in war veterans. Shipherd, Keyes, Jovanovic, and Ready (2007) showed that in a group of 90 male war veterans who had previously received treatment for PTSD, 66% endorsed chronic pain. In a sample of 129 combat veterans of the Vietnam War with PTSD, 80% reported experiencing chronic pain (Beckham et al., 1997).

Similarly, individuals with chronic pain report higher levels of PTSD (DeCarvalho & Whealin, 2006). It is estimated that in pain clinics, rates of PTSD are between 9.5 and 33%

(Macfarlane et al., 1999; Meltzer-Brody et al., 2007). The Canadian Community Health Survey was conducted with 36,984 individuals in 2002 (Sareen et al., 2007) and found higher incidence of PTSD among individuals with back problems (20.6% of individuals without PTSD and 46.0% of individuals with PTSD), arthritis (17.3% of individuals without PTSD and 42.6% of individuals with PTSD), migraine headaches (10.5% of individuals without PTSD and 33.8% of individuals with PTSD), and fibromyalgia (1.4% of individuals without PTSD and 7.7% of individuals with PTSD). Furthermore, among individuals reporting pain, symptoms of PTSD are reported to be worse than if comorbid pain is not present (Villano, Rosenblum, Magura, & Fong, 2007). When pain develops as a consequence of trauma (e.g., injuries sustained in military combat, motor vehicle accidents, or work-related accidents), it is more likely that the individual will suffer from PTSD (Otis, Keane, & Kerns, 2003). However, it is important to note that the consistent link between trauma and pain exists regardless of whether pain and/or injury are part of the traumatic event. Evidence of a link between chronic pain and traumatic exposure in the absence of bodily harm was provided by Asmundson et al (2002) who highlighted the importance of discerning the mechanisms by which this link is established and maintained. Taken together, these findings support the strong relationship between chronic pain and PTSD.

Models of pain and PTSD. Due to the high comorbidity between trauma and chronic pain, many models have been developed to try to explain this relationship. They include models that propose that the symptoms of both pain and anxiety maintain each other and that pre-existing individual differences make certain individuals more vulnerable to both pain and anxiety, as well as models that combine both of these considerations (Katz, Pagé, Fashler, Rosenbloom, & Asmundson, 2014).

Mutual Maintenance Model. Based on the high level of comorbidity between PTSD and chronic musculoskeletal pain, Sharp and Harvey (2001) developed the Mutual Maintenance model according to the premise that these two conditions are connected rather than being distinct disorders. They suggest that the behavioural, affective, and cognitive features of each condition can exacerbate and maintain the other. They specify seven possible mechanisms through which this process might occur: (1) attentional and reasoning biases, where more focus is paid to threatening and pain-related stimuli and subsequently the likelihood of danger or pain is considered to have a higher probability of occurrence, (2) sensitivity to feelings of anxiety, whereby individuals are more fearful of the symptoms associated with anxiety, such as physiological arousal, (3) reminders of the trauma, especially if pain serves as a reminder of the trauma, (4) avoidance of reminders related to trauma and of pain sensations, (5) depression and reduced activity levels, possibly leading to physical deconditioning and contributing to avoidance, (6) an elevated level of anxiety that is linked to increased pain perception, and (7) high cognitive load, where thoughts related to the trauma and pain place greater demands on cognition. This model presents several mechanisms to help understand how symptoms of chronic pain and PTSD may maintain each other, although it does not necessarily represent a specific causal relationship among the seven suggested mechanisms.

Perpetual Avoidance Model. Liedl and Knaevelsrud (2008) developed the Perpetual Avoidance Model that suggests that the high comorbidity between chronic pain and PTSD is caused by avoidance. They indicated that this mechanism is both causal and maintaining. It was developed out of the cognitive model of PTSD suggested by Ehlers and Clark (2000) and the fear avoidance model of chronic musculoskeletal pain developed by Vlaeyen and Linton (2000). They propose that following a trauma, individuals with PTSD engage in dysfunctional thinking

patterns and experience intrusive thoughts and memories. This situation causes hyperarousal and awareness of pain sensations and avoidance of activities, where the avoidance can then cause pain sensations due to lack of use, causing deconditioning and subsequently more pain. In addition, pain sensations are predicted to contribute to catastrophizing and fear-avoidance beliefs related to pain, which can similarly cause avoidance of activities. In summary, Liedl and Knaevelsrud (2008) suggest that avoidance is the central mechanism contributing to the cause and maintenance of symptoms of chronic and PTSD.

Diathesis-Stress Model of Chronic Pain and Disability. Turk (2002) developed the Diathesis-Stress Model of chronic pain and disability to explain why the degree of chronic pain and disability experienced is not well correlated with underlying physical pathology. The model suggests that individuals are more likely to develop chronic pain if they possess certain predisposing qualities (i.e., diathesis) and experience a physical trauma (i.e., stress). It builds on the findings of work by Asmundson and Taylor (1996) which demonstrates that anxiety sensitivity can exacerbate fear of pain, which can subsequently lead to more pain-related avoidance. In his expanded model, Turk (2002) proposed that important predisposing factors include anxiety sensitivity, fear of pain or injury, catastrophizing ideation, cognitive attributions about the cause and consequence of physical symptoms, and poor self-efficacy. These vulnerability factors can contribute to the avoidance of behaviours that are related to pain, which may then contribute to the development of pain through disuse. It is suggested that learning plays an active role in this cycle, where individuals learn to predict events and how to react to them. The manner in which information is processed can lead to subsequent avoidance and emotional responses, such as anxiety. Taken together, these risk factors can increase the likelihood of an individual developing disability in response to a physical traumatic stressor.

Martin, Halket, Asmundson, Flora, and Katz (2010) tested the Diathesis-Stress Model in a population of 208 patients undergoing major surgery. They examined the scores on questionnaires assessing anxiety sensitivity, pain catastrophizing, pain anxiety, pain disability, and symptoms of posttraumatic stress. The results showed that anxiety sensitivity predicts both pain catastrophizing and fear of pain, which then subsequently predicted escape and avoidance behaviour. Furthermore, escape and avoidance behaviour predicted pain disability. When symptoms of posttraumatic stress were added to the model, it was found that they accounted for a degree of variance in pain disability. The authors concluded that experiencing pain, fear of pain, and avoidance of pain can make individuals more vulnerable to developing posttraumatic stress symptoms and that pain itself may serve as a traumatic stressor that can independently lead to posttraumatic symptoms.

Shared Vulnerability Model. The Shared Vulnerability Model was developed by Asmundson et al. (2002) to explain the high degree of comorbidity between PTSD and musculoskeletal pain. It expands on the Mutual Maintenance Model by suggesting that not only do the behavioural, affective, cognitive, and physiological features of both conditions maintain each other, but they also can serve as vulnerability factors, making individuals with these characteristics more likely to develop PTSD and chronic musculoskeletal pain to begin with. In this model, the vulnerability factors include life stressors, such as a traumatic event or injury, low threshold for alarm, such as sympathetic dysregulation, and psychological vulnerability, such as being high in anxiety sensitivity. These factors contribute to an emotional response to a life stressor, such as anxiety, worry, and fear. The emotional response may be accompanied by physiological, behavioural, and cognitive patterns that cause and maintain the emotional reactivity and levels of disability. In addition to offering an explanation of potential cause and

maintenance of PTSD and chronic pain, this model may apply more broadly to other anxiety disorders, given that these core features are present in other conditions.

Triple Vulnerability Model of PTSD and Chronic Pain. Keane and Barlow (2002) developed the Triple Vulnerability Model to propose the mechanisms that contribute to the development of PTSD. They suggested that there are three integral predisposing factors: biological vulnerability, general psychological vulnerability (e.g., learned helplessness in response to life events), and specific psychological vulnerability (e.g., when anxiety is present in certain contexts). The model proposes that learning is central to the development of PTSD: following an alarm response to a traumatic event, an association is learned that both of these elements are uncontrollable and unpredictable, which can make individuals more susceptible to develop PTSD. Otis et al. (2003) expanded this model to apply to chronic pain. They suggested that individuals who go on to develop pain similarly possess vulnerabilities that are biological (e.g., a genetic predisposition to develop chronic pain), general psychological vulnerabilities (e.g., the global belief that pain is uncontrollable and unpredictable), and specific psychological vulnerabilities (e.g., informed by particular previous experiences with pain). As with PTSD, when pain is perceived as uncontrollable, this perception can contribute to negative affect and poor self-efficacy, in turn promoting fear of pain and avoidance of activities that may cause pain, which can contribute to further negative affect, feeling a lack of control, and poor self-efficacy. Importantly, social support and coping skills are also predicted to have a mediating effect between these vulnerabilities and the development and maintenance of PTSD and chronic pain.

Combined Shared Vulnerability and Mutual Maintenance Model of PTSD and Chronic Pain. Further research examining the relationship between PTSD and chronic pain led Rosenbloom, Khan, McCartney, and Katz (2013) to develop the Combined Shared Vulnerability

and Mutual Maintenance Model of Disability for traumatic injury, which comprehensively combines and extends purported mechanisms of both conditions. It builds on Turk's (2002) Diathesis-Stress Model, suggesting that predisposing factors include biological vulnerabilities in addition to psychological vulnerabilities. Biological vulnerabilities include an over-responsive startle reflex, sensitive hypothalamic-pituitary-adrenal axis, and concurrent disease or injury. Following a traumatic injury, fear avoidance factors (including the pain experience, fear of pain, pain catastrophizing, and poor self-efficacy) may interact with posttraumatic stress symptoms (including reexperiencing, hyperarousal, avoidance, and emotional numbing) in a manner that is bi-directionally mutually maintaining for both symptoms of PTSD and chronic pain.

Measurement of trauma. The assessment of trauma symptoms is most commonly conducted using structured interviews and self-report questionnaires. Structured interviews are administered by trained interviewers and involve asking a participant a series of questions regarding specific symptoms. One of the most commonly administered clinical interviews is the Clinician-Administered PTSD Scale (CAPS) structured interview, which was developed by the National Center for PTSD (Blake et al., 1995). The CAPS assesses all diagnostic criteria of PTSD as described in the DSM, starting with the DSM-III-R (Blake et al., 1995), and later revised to match the revisions to diagnosis in the DSM-IV (Weathers, Keane, & Davidson, 2001) and the DSM-5 (Weathers et al., 2017). Its use has been validated in several populations, including combat veterans, victims of torture, and individuals who have been in a motor vehicle accident (Weathers et al., 2001). It is frequently used as the primary diagnostic tool to establish the severity of symptoms or the diagnosis of PTSD. The Structured Clinical Interview for DSM (SCID) is also a popular interview used in the diagnosis of PTSD (Spitzer, First, Gibbon, & Williams, 1990; Spitzer, Williams, Gibbon, & First, 1992). It was developed to assess major

psychiatric disorders and a special module was developed to specifically measure symptoms of PTSD (Spitzer & Williams, 1985). Similar to the CAPS, the SCID has been modified to meet the changing criteria of the DSM-III-R (Spitzer et al., 1990; Spitzer & Williams, 1985), DSM-IV (First & Gibbon, 1995), and DSM-5 (First, Williams, Karg, & Spitzer, 2015). Other popular clinical interviews include the PTSD Symptom Scale Interview (Foa, Riggs, Dancu, & Rothbaum, 1993), the Structured Interview for PTSD (Davidson, Smith, & Kudler, 1989), and the Anxiety Disorders Interview Schedule - Revised (Brown, Di Nardo, & Barlow, 1994). Numerous self-report measures have been developed to assess trauma symptoms. The PTSD Checklist (PCL) was developed by the National Center for PTSD (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996). It has three versions to assess PTSD in a military population (PCL-M), civilian population (PCL-C; Weathers, Litz, Herman, Huska, & Keane, 1994), and in response to a specific stressful experience (PCL-S; Blanchard et al., 1996). Questions correspond to the diagnostic criteria of PTSD described in the DSM and the number of items for the scale varies depending on the version of the DSM (Blanchard et al., 1996; Blevins, Weathers, Davis, Witte, & Domino, 2015; Weathers et al., 1994). The Impact of Events Scale (IES; Horowitz, Wilner, & Alvarez, 1979) was developed to measure the frequency of cognitive intrusions (including dreams, memories, and thoughts) and avoidance behaviours (including of specific people, situations, and thoughts) related to a traumatic experience. To better match the diagnostic criteria of PTSD in the DSM, the IES was revised by Weiss and Marmar (1997) who added six items to assess hyperarousal. Other popular self-report measures include the Mississippi Scale for Combat-Related PTSD (Keane, Caddell, & Taylor, 1988), Distressing Event Questionnaire (Kubany, Leisen, Kaplan, & Kelly, 2000), and the Posttraumatic Diagnostic Scale (Foa, Cashman, Jaycox, & Perry, 1997).

Measuring Pain-Related Anxiety

The development of pain is complex and it is influenced by various biopsychosocial factors, including anxiety- and trauma-related symptoms. Although these links are well-established, only a few measures have been developed to examine pain-related anxiety and physiological responsiveness. They are described in more detail below.

Anxiety Sensitivity Index (ASI)**.** The ASI was developed to assess an individual's tendency to be worried about the harmful consequences of the symptoms of anxiety (Reiss, Peterson, Gursky, & McNally, 1986). The questionnaire has three subscales, including anxiety sensitivity related to physical concerns (i.e., worry about the negative consequences of physical symptoms such as tightness in the chest, stomach ache), cognitive concerns (i.e., worry about the negative consequences of thinking patterns such as one's mind going blank, difficulty concentrating), and social concerns (i.e., worry about the negative consequences of other people witnessing anxiety-related symptoms). Anxiety sensitivity has been implicated as a risk factor in various models (e.g., Diathesis-Stress Model of Chronic Pain and Disability and Shared Vulnerability Model) and is related to pain-related fear and avoidance in individuals suffering from recurrent headaches (Norton & Asmundson, 2004), chronic back pain (Asmundson & Norton, 1995), and other pain-related conditions (Asmundson, Wright, & Hadjistavropoulos, 2000).

Illness Sensitivity Index (ISI). The ISI was developed to assess an individual's tendency to fear illness or injury (Taylor, 1993). The questionnaire has two subscales to separately assess fear of illness and injury (Carleton, Asmundson, & Taylor, 2005), which is in contrast to the proposition that it was a singular fear. Although closely related, illness or injury sensitivity has been found to be factorially distinct from anxiety sensitivity (Taylor, 1993), although closely

related to a physical concerns subscale (Carleton et al., 2005; Carleton, Park, & Asmundson, 2006). It has been proposed that illness or injury sensitivity is a vulnerability factor which can maintain or exacerbate health concerns such as chronic pain (Asmundson et al., 2002). This conjecture has been supported by the finding that illness or injury sensitivity can predict avoidance behaviours better than anxiety sensitivity can (Vancleef, Peters, Roelofs, & Asmundson, 2006).

Pain Anxiety Symptoms Scale (PASS). The PASS was developed to assess the degree an individual fears pain and pain-related sensations (McCracken & Dhingra, 2002). The questionnaire has four subscales, including cognitive anxiety (i.e., worry about how pain impacts thinking), pain-related fear (i.e., worry about the experience of painful sensations), escape and avoidance (i.e., the avoidance of activities that may induce pain), and physiological anxiety (i.e., worry about the physical symptoms associated with experiencing pain, such as nausea). Pain anxiety is related to ratings of pain severity and disability (McCracken, Gross, Aikens, & Carnrike, 1996). Although PASS is related to anxiety sensitivity, research has found that scores on the PASS reflect more of a continuum of symptoms, whereas anxiety sensitivity can provide a measure that corresponds to when pain becomes more pathological (Bernstein, Zvolensky, Vujanovic, & Moos, 2009).

Fear of Pain Questionnaire (FPQ). The FPQ was developed to assess the amount of fear that an individual experiences in response to different painful experiences (McNeil & Rainwater, 1998). The questionnaire has three subscales to examine fear of different types of pain: minor pain (e.g., biting your tongue), severe pain (e.g., being in a motor vehicle accident), and medical pain (e.g., receiving an injection in your arm). As described in the Diathesis-Stress Model of chronic pain (Turk, 2002), the Triple Vulnerability Model of PTSD and Chronic Pain

(Keane & Barlow, 2002), and the Combined Shared Vulnerability and Mutual Maintenance Model of PTSD and Chronic Pain (Rosenbloom et al., 2013), fear of pain is suggested to be an important contributor to the avoidance of activities that may cause pain, which can subsequently contribute to deconditioning from underuse. Moreover, it is more likely that individuals with traumatic symptoms and social anxiety will be fearful of pain (Asmundson & Carleton, 2005).

Pain Catastrophizing Scale (PCS). The PCS was developed to assess the degree to which individuals have strong, negative reactions to the experience or anticipation of pain (Sullivan, Bishop, & Pivik, 1995). The questionnaire has three subscales related to different aspects of pain catastrophizing, specifically rumination (i.e. over-thinking about pain, worrying about the pain, an inability to stop thinking about pain), magnification (i.e., the tendency to interpret pain as more serious, unpleasant, and negative than it is), and helplessness (i.e., belief that the individual will be unable to cope with the pain). The PCS measures an anxious response to pain and is considered a risk factor in several models of pain and PTSD (Rosenbloom et al., 2013; Turk, 2002)

Development of the Sensitivity to Pain Traumatization Scale

The idea behind the construct known as Sensitivity to Pain Traumatization was first conceived to address the high comorbidity rate of PTSD and chronic pain and was defined as "the propensity to develop anxiety related somatic, cognitive, emotional and behavioural responses to pain that resemble the features of a traumatic stress reaction" (Kleiman, Clarke, & Katz, 2011, p. 175). The Sensitivity to Pain Traumatization Scale was developed in two studies using different methodology, although they both used item reduction analysis to identify a common factor from items of several pain-related anxiety scales (Kleiman et al., 2011; Roosen,

2009). Both studies are described below with the items from final versions of the scales displayed in Table 1.1.

The first study was conducted by Roosen (2009) and described the development of a 12-item Sensitivity to Pain Traumatization Scale (SPTS-12; Katz et al., 2017). First, a list of 79 items was generated from existing scales measuring pain-related anxiety constructs, consulting with experts, and a literature review. The five scales that were evaluated included the ASI, PCS, FPQ-III, PASS-20, PCL-C, and IES. To ensure clinically relevant scale items, specific criteria for inclusion in the final scale were based on diagnostic criteria for Post-Traumatic Stress Disorder (PTSD) from the DSM-IV-TR (American Psychiatric Association, 2000) and a literature review. The item domains were labeled "pain and avoidance", "pain and emotional numbing", "pain and hyperarousal", "pain experiencing", "fear of pain", and "pain sensitivity". Next, all scale items were combined into a questionnaire that was completed by a sample of 116 undergraduate students at York University. The list of items was first reduced using nonparametric item response theory (IRT), whereby items with almost identical wording were examined and the item with the least variance was removed. Next, a kernel-smoothing procedure was used to estimate the unidimensional item response function. Items were then chosen for inclusion in the final scale based on the item characteristic curves (ICCs), option characteristic curves (OCCs), and the representation of items according to diagnostic criteria for PTSD determined prior to analysis. Only two items from each diagnostic criteria category were selected for inclusion for the final 12-item scale. Next, a parametric IRT graded response model was fitted to data for the newly developed 12-item scale. An exploratory factor analysis supported a one-factor model for SPTS-12, providing evidence that it is a unidimensional construct. The scale properties were then examined using coefficient alpha ($\alpha = 0.92$) and an inter-item

correlation matrix, leading the author to report that the scale shows "good preliminary reliability" (p. 41). The resulting SPTS-12 uses a 5-point Likert-type scale with item anchors 0 = "not at all true" and 4 = "entirely true". The score range is 0-48 with higher scores indicating more traumatic responses to pain. The SPTS-12 has been used in subsequent research and has demonstrated excellent internal consistency (coefficient alpha = .91; Fashler & Katz, 2014).

The second study was conducted by Kleiman et al. (2011), who generated a list of items from the PASS-20, PCS, and ASI to construct a questionnaire to assess the theoretical construct of Sensitivity to Pain Traumatization. The questionnaire items were then completed by a sample of 444 patients prior to undergoing major surgery. Six months and 12 months after surgery, patients were followed up by phone to assess their level of postsurgical pain. The factor structure of the three measures of pain-related anxiety was examined using exploratory factor analysis. Based on these findings, 20 items were retained for the longer Sensitivity to Pain Traumatization Scale (SPTS-20). Like the SPTS-12, the scale's items use a 5-point Likert-type scale with item anchors ranging from 0 = "not at all true" to 4 "entirely true". The score range is 0-80, with higher scores reflecting more traumatic responses to pain. Criterion validity of the SPTS-20 was evaluated by correlating the total SPTS-20 score with the total score for the PCL-C, as it is well-established and commonly used scale to measure trauma, and a strong positive relationship was found. Furthermore, the total SPTS-20 score was significantly higher for individuals reporting ongoing pain problems compared to those not reporting ongoing pain problems showing some evidence of known-groups validation, as we would expect the total score on the SPTS-20 to be higher for individuals reporting pain than those not reporting pain (Kleiman et al., 2011). The internal consistency reliability of the SPTS-20 has not yet been evaluated.

In summary, the Sensitivity to Pain Traumatization Scale was recently developed to assess the traumatic response that an individual may exhibit in response to pain. Two versions of the scale have been developed using difference methodologies. The scales show theoretical promise in better understanding and addressing the high comorbidity rates between posttraumatic stress disorder and chronic pain. The purpose of the present dissertation is to conduct further psychometric evaluation of the reliability and validity of the SPTS-12.

Table 1.1. Items on the 12- and 20-item version of the Sensitivity to Pain Traumatization Scale.

	SPTS-20 (Kleiman, 2011)	SPTS-12 (Roosen, 2009)
1	When I feel pain, I worry all the time about whether it will end	Pain keeps me awake at night[a]
2	It scares me when I tremble or feel shaky.	When I am in pain, everything I see or do reminds me of the pain
3	I can't think straight when in pain.	I try to avoid activities that cause pain
4	Other people notice when I tremble or feel shaky.	When I feel pain, I'm scared that it's the beginning of a terrible problem
5	When I feel pain, it is difficult for me to think of anything else.	Pain seems to bother me more than it does other people
6	When I feel pain, I keep thinking of other painful events.	When I feel pain, I think about it even when I don't mean to
7	When I feel pain, I am afraid that something terrible will happen.	I can't stand pain
8	When pain comes on strong, I think that I might become paralyzed or more disabled.	When I'm in pain, I feel distant from people even when I'm talking to them
9	When pain gets too severe, I think it will never decrease.	As soon as the pain comes on, I take medications to reduce it
10	I go immediately to bed when I feel severe pain.	Pain sensations terrify me
11	When I feel pain, I feel I can’t go on.	When I'm in pain, things don't feel real
12	As soon as pain comes on I take medication to reduce it.	I feel sick to my stomach when I am in pain
13	When I feel pain, I wonder whether something serious may happen.	
14	It scares me when I feel faint.	
15	When I feel pain, I think that I might be seriously ill.	
16	When I feel pain, I think about it constantly.	
17	I try to avoid activities that cause pain.	
18	I find it hard to concentrate when I hurt.	
19	It embarrasses me when my stomach growls.	
20	I worry when I am in pain.	

Note. SPTS-20 – 20-item Sensitivity to Pain Traumatization Scale, SPTS-12 – 12-item Sensitivity to Pain Traumatization Scale

[a] This item was modified to “when I am in pain, it keeps me awake at night” in the final published version of the SPTS-12 (Katz et al., 2017).

Chapter 2: Study 1

Disclosure Notes

A portion of the research reported in Study 1 has been published. The data were presented as a poster presentation at the Canadian Pain Society conference in May 2013, and an abstract of the poster and presentation was published in *Pain Research & Management* (vol. 18, issue 2, pp. e1-e47, 2013). Some of the text below was published in the article "Sensitivity to Pain Traumatization Scale: development, validation, and preliminary findings" that appeared in the *Journal of Pain Research* (vol. 10, pp. 1297-1316, 2017) as a part of a larger study validating the STPS. All content included in the present study was authored by Samantha Fashler.

Introduction

In recent years, the significant symptom overlap between chronic pain and Posttraumatic Stress Disorder (PTSD) has led researchers to propose several theoretical models to explain the relationship between chronic pain and PTSD. These include mutual maintenance models (e.g., the Mutual Maintenance Model; Sharp & Harvey, 2001, Perpetual Avoidance Model; Liedl & Knaevelsrud, 2008), which suggest that the psychological, behavioural, and physical symptoms of PTSD can maintain the symptoms of both pain and PTSD. Mutual maintenance models emphasize the role of seven core mechanisms which can be mutually reinforcing: attentional/reasoning biases, anxiety sensitivity, reminders of the trauma, avoidance, depressive symptoms, anxiety of pain perceptions, and high cognitive load. Vulnerability models (e.g., Diathesis-Stress Model of Chronic Pain and Disability; Turk, 2002, Shared Vulnerability Model; Asmundson, Coons, Taylor, & Katz, 2002), in contrast, suggest that similar vulnerabilities make individuals more susceptible to developing both PTSD and chronic pain. These models hypothesize that these risk factors include the interplay between various individual

characteristics (such as anxiety sensitivity, fear of pain or re-injury, catastrophizing, and self-efficacy), behavioural responses to trauma (such as avoidance/escape), and physiological factors (such as low threshold for alarm). Moreover, the Shared Vulnerability Model further suggests that higher levels of anxiety can reduce the threshold for pain that can in turn increase distress and pain disability (Asmundson et al., 2002).

When considered together, there is a wealth of theoretical and empirical evidence suggesting that psychological and behavioural constructs are important elements contributing to the high co-occurrence of chronic pain and PTSD. To better understand these overlapping concepts, Kleiman et al. (2011) proposed that there may be a unitary psychological construct that underlies the pain-related anxiety symptoms proposed in mutual maintenance and shared vulnerability models. The resulting construct was termed "Sensitivity to Pain Traumatization", and it was defined as the propensity to develop anxiety-related somatic, emotional, cognitive, and behavioural responses to pain which resemble features of a traumatic stress reaction.

The primary aims of Study 1 were to evaluate the psychometric properties of the 12-item Sensitivity to Pain Traumatization Scale (SPTS-12), including its factor structure, reliability, convergent validity, and divergent validity in a sample of undergraduate students who are pain-free or are reporting experiencing ongoing pain. This aim will be achieved with a factor analysis and evaluation of the correlations between the SPTS-12 and measures of theoretically related constructs. For both undergraduate students reporting and not reporting ongoing pain, the following is predicted: (1) the associations among the 12 items of the SPTS-12 will suggest that a one-factor model will be ideal with strong factor loadings for each item; (2) the SPTS-12 items will have good internal consistency; (3) the SPTS-12 will be highly correlated with the total score of a scale assessing a similar construct (i.e., symptoms of post-traumatic stress scores as

measured by the PCL-C); and (4) the SPTS-12 will be less strongly correlated with a scale assessing a related, but distinct construct, specifically depressive symptoms as measured by the Beck Depression Inventory-II.

Methods

Procedure. The study protocol was reviewed and approved by the York University Research Ethics Board (Human Participants Review Subcommittee, certificate approval #s: 2009-008 and 2013-018). Approval letters for this study are listed in the Appendix A. Participants were recruited online through the Undergraduate Research Participant Pool at York University in 2009, 2012, and 2013. Participants provided informed consent electronically through an online consent form prior to participation. Next, they were asked to complete an online survey consisting of basic demographic questions and nine questionnaires to assess anxiety, traumatic responses, perception of painful experiences, and depressive symptoms: SPTS-12, ASI, BDI-II, FPQ-III, ISI-R, PASS-20, PCS, PCL-C, and STAI-T. After completing the study, participants were presented with a debriefing form that explained the purpose of the investigation and provided contact information of the investigators for any follow-up questions. Students received course credit for participating.

Measures.

Demographic information and pain history. Participants provided information about their gender, ethnic background, and age. Pain history questions included the question "do you experience pain on an ongoing basis?" as well as follow-up questions regarding the diagnosis, duration, frequency, average intensity, and degree of pain interference in their daily life.

Sensitivity to Pain Traumatization Scale (SPTS-12). The SPTS-12 assesses the propensity to develop a traumatic response to pain. The 12-item version developed by Roosen

(SPTS-12; 2009) and subsequently published by Katz et al. (2017) as well as the 20-item version developed by Kleiman et al. (SPTS-20; 2011) were included. For both the SPTS-12 and SPTS-20, the items use a 5-point Likert-type response scale with anchors 0 = *not at all true* and 4 = *entirely true*. The total score range is 0 to 48 for the SPTS-12 and 0 to 80 for the SPTS-20, with higher scores indicating a more traumatic response to pain. Item 1 of the original SPTS-12 was re-worded from "Pain keeps me awake at night" to "When I am in pain, it keeps me awake at night" so that it is applicable to people who do not have pain at the time of questionnaire completion.

Anxiety Sensitivity Index (ASI; Reiss et al., 1986). The ASI assesses fear of the physical symptoms of anxiety, such as pounding heart and shortness of breath. It consists of 16 items using a 5-point Likert-type response scale, with item anchors 0 = *very little* and 4 = *very much*. The total score range is 0 to 64, with higher scores indicating greater fear of anxiety symptoms. The ASI has excellent internal consistency (α = .93; Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012). The ASI showed very good internal consistency in the present study (α = .89).

Beck Depression Inventory – II (BDI-II; Beck, Steer, & Brown, 1996)**.** The BDI-II assesses cognitive, emotional, and physical symptoms of depression. It consists of 21 items using a 4-point Likert-type response scale. Each item has a 4-point response scale with anchors pertaining to the symptom being measured; for example, the first item assesses sadness with anchors 0 = *I do not feel sad*, 1 = *I feel sad much of the time*, 2 = *I am sad all the time*, and 3 = *I am so sad or unhappy that I can't stand it*. The total score range is 0 to 63, with higher scores reflecting greater depressive symptoms. The BDI-II has excellent internal consistency (α = .90), shows good test-retest reliability over a range of times (r = 0.73 to 0.93), and has established

high construct validity (Beck et al., 1996; Wang & Gorenstein, 2013). The BDI-II showed excellent internal consistency in the present study (α = .93).

Fear of Pain Questionnaire - III (FPQ-III; McNeil & Rainwater, 1998)**.** The FPQ-III assesses the degree of fear associated with a variety of painful experiences. It consists of 30 items using a 5-point Likert-type response scale, with item anchors 0 = *not at all* to 4 = *extreme*. The total score range is 0 to 120, with higher scores indicating higher levels of fear of experiencing different types of pain. The FPQ-III has good internal consistency and test-retest reliability, with the scale demonstrating strong construct validity (McNeil & Rainwater, 1998). The FPQ-III showed excellent internal consistency in the present study (α = .94).

Illness Sensitivity Index – Revised (ISI-R; Carleton et al., 2006). The ISI-R is revised version of the 11-item scale that assesses fear of illness and injury (Taylor, 1993). It consists of nine items using a 5-point Likert-type response scale, with item anchors 0 = *agree very little* to 4 = *agree very much*. The total score range is 0 to 36 with higher scores indicating greater levels of fear of illness and injury. The ISI-R has very good internal consistency (α = .86), good convergent validity ($r > .65$) with other injury- and illness- related measures, and correlates very highly with the original scale ($r = .96$; Carleton et al., 2006). The ISI-R showed excellent internal consistency in the present study (α = .92).

Pain Anxiety Symptoms Scale – Short Form (PASS-20; McCracken & Dhingra, 2002). The PASS-20 assesses pain-related anxiety and is a shorter, revised version of the 40-item Pain Anxiety Sensitivity Scale (McCracken, Zayfert, & Gross, 1992). It consists of 20 items using a 6-point Likert-type response scale, with item anchors 0 = *never* to 5 = *always*. The total score range is 0 to 100, with higher scores indicating higher levels of anxiety related to pain. The PASS-20 has excellent internal consistency (α = .91), convergence with the original 40-item

scale is very strong (r = .97; McCracken & Dhingra, 2002), and it has demonstrated good construct validity (McCracken & Dhingra, 2002). The PASS-20 showed excellent internal consistency in the present study (α = .94).

Pain Catastrophizing Scale (PCS; Sullivan et al., 1995). The PCS assesses ruminative thinking, perception of threat, and feelings of helplessness in relation to painful experiences and sensations. It consists of 13 items using a 5-point Likert-type response scale, with item anchors 0 = *not at all* and 4 = *all the time*. The total score range is 0 to 52 with higher scores indicating greater levels of pain catastrophizing. The PCS has good internal consistency (α = .87; Sullivan et al., 1995) and test-retest reliability over a six-week period (r = .75; Sullivan et al., 1995). The PCS has demonstrated strong construct validity (Osman et al., 1997; Sullivan et al., 1995). The PCS showed excellent internal consistency in the present study (α = .94).

PTSD Checklist – Civilian Version (PCL-C; Weathers et al., 1994). The PCL-C assesses the traumatic response that individuals may have in response to stressful events. It consists of 17 items using a 5-point Likert-type response scale, with item anchors 1 = *not at all* to 5 = *extremely*. The total score range is 17 to 85, with higher scores reflecting greater traumatic responses. The PCL-C has excellent internal consistency (α = .92-.94), the test-retest reliability over a two-week period is good (r = .66), and the scale demonstrates good convergent and discriminant validity (Conybeare, Behar, Solomon, Newman, & Borkovec, 2012). The PCL-C had excellent internal consistency in the present study (α = .93).

State-Trait Anxiety Index – Trait Version (STAI-T; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). The STAI assesses both temporary "state" levels of anxiety and enduring "trait" characteristics of anxiety. The present study only assessed trait anxiety with the trait subscale (STAI-T), which consists of 20 items using a 4-point Likert-type response scale, with

item anchors 1 = *almost never* to 4 = *almost always*. The total score range is 20 to 80 with higher scores indicating greater levels of trait anxiety. The STAI-T has good internal consistency (Cronbach's α = .89; Bieling, Antony, & Swinson, 1998), high test–retest reliability (r = 0.73–0.86; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983), and strong convergent validity with related anxiety measures . The STAI-T showed excellent internal consistency in the present study (α = .92).

Statistical analysis. See Appendix B for the syntax used in the final analysis.

Exploratory factor analysis. Exploratory factor analysis (EFA) was favored over confirmatory factor analysis (CFA) because the SPTS-12 had not been previously examined in clinical and non-clinical settings. Analyses were conducted in R (Version 3.5.0) using the "psych" package (Revelle, 2018). Because the item responses of the SPTS-12 are given on a 5-point Likert-type scale, the items were considered categorical rather than continuous. Therefore, polychoric correlations were used to account for the discrete nature of the variables (see Flora, LaBrish, & Chalmers, 2012). EFA was used to examine the dimensional structure of the SPTS-12 and to assess the degree to which each item is influenced by the underlying factor(s). Ordinary least squares estimation was selected due to its robust statistical properties with polychoric correlations (Lee, Zhang, & Edwards, 2012). For models with two or more predicted factors, multiple oblimin rotations were conducted (oblimin weights: 0, .25, .50, and .75) to allow greater approximation to simple structure (Browne, 2001).

Five methods were used to determine the optimal number of factors: examination of the scree plot (Cattell, 1966), parallel analysis (Longman, Cota, Holden, & Fekken, 1989), the root mean square residual (RMR), root mean square error of approximation (RMSEA) index, and Tucker-Lewis index (TLI).

Reliability. Internal consistency reliability was calculated using McDonald's omega (McDonald, 1970, 2013). Omega was favoured over coefficient alpha because it provides a more accurate estimate of reliability if multidimensionality is present, factor loadings differ across items, or measurement errors are correlated across items (Raykov & Marcoulides, 2016).

Validity. Spearman's correlations were used for comparisons because they do not require a linear relationship between variables. Convergent validity was evaluated with the correlation between the SPTS-12 and a scale measuring a theoretically similar construct, the PCL-C. Divergent validity was evaluated from the correlation between the SPTS-12 and the BDI-II, a measure of a related but theoretically distinct construct. The magnitude of the difference between correlation coefficients for SPTS-12 and PCL-C and for SPTS-12 and BDI-II was evaluated (Lee & Preacher, September, 2013). A significantly larger correlation for the former than the latter would suggest good convergent and divergent validity.

Results

A total of 860 participants were recruited. Data from participants who failed to respond to all questions of the SPTS-12 (n = 26) or did not indicate whether they experience ongoing pain (n = 11) were excluded from the analysis. For the remaining questionnaires, total scores were prorated by calculating the mean score for available items and then multiplying this by the total number of items in the scale to create a new adjusted total score (Enders, 2010). Prorating was only conducted if 80% or more of questions were completed. Missing data ranged from a low of 1.19% for the ISI-R to a high of 2.35% for BDI-II.

Participant characteristics. Of the 823 participants, 268 (32.56%) endorsed experiencing pain on an ongoing basis whereas 555 (67.44%) did not. For participants not endorsing pain, the age range was 17 to 42 years (M = 19.34, SD = 3.43). The majority identified

as female (n = 388; n = 165 male, n = 2 decline to respond). The participants were ethnically diverse, identifying as of African descent (n = 30), Asian descent (n = 291), Hispanic/Latino (n = 10), Caucasian (n = 136), and other (n = 90), with eight individuals declining to respond.

The age range for those with ongoing pain was 16 to 45 years (M = 20.63, SD = 4.63). The majority identified as female (n = 202; n = 66 male, n = 1 decline to respond). The participants were ethnically diverse, identifying as of African descent (n = 11), Asian descent (n = 105), Hispanic/Latino (n = 5), Caucasian (n = 99), and other (n = 46), with two individuals declining to respond.

Further information was collected from participants reporting ongoing pain. For the length of time experiencing ongoing pain, 16.4% reported experiencing pain for less than 3 months, 11.6% between 3 and 6 months, 13.4% between 6 months and 1 year, and 55.6% for longer than 1 year (3% declined to respond). For frequency of pain, 38.1% reported experiencing pain daily, 37.7% weekly, and 16.4% monthly (5.2% described the frequency of pain as "other" and 1.9% declined to respond). For severity, 30.6% described their pain as mild, 57.5% as moderate, and 10.8% as severe (1.1% declined to respond). For degree of interference that their pain level has on daily functioning, 9.3% reported that it was not at all affected, 56.0% that it was slightly affected, 28.4% that it was moderately affected, and 5.2% that it was severely affected (1.1% declined to respond).

Participants endorsing ongoing pain were significantly older than those who did not endorse pain, $t(420.87) = 3.79$, $p < .001$ (M = 20.63, SD = 4.64 for participants endorsing pain vs. M = 19.35, SD = 3.43 for those not reporting pain). Presence or absence of ongoing pain did not differ significantly by gender, $\chi^2(1, N = 843) = 2.12$, $p = .137$.

Descriptive statistics. Descriptive statistics for the 12 items of the STPS are reported in Table 2.1 separately for participants reporting no pain and for those reporting ongoing pain. Tables 2.2 and 2.3 show the polychoric correlations for participants reporting no pain and those reporting ongoing pain, respectively.

Factor analysis.

Participants reporting no pain. For participants not reporting ongoing pain (n = 555), the scree plot suggested a one-factor model (see Figure 1) and parallel analysis suggested a three-factor model. For the one-factor model, RMR = .04, RMSEA = .08 (90% CI: .07-.09) and TLI = .92, all suggesting adequate model fit.

The factor loadings and communality estimates for the one-factor model are shown in Table 2.4. The two largest residual correlations were between item 4 and item 9 (r = -.11) and between item 9 and item 12 (r = .10). Overall, the relationships among items are adequately explained by the one-factor model, although there may be additional, minor dimensions underlying the STPS, particularly item 9, that are not represented by the one-factor model.

Two-factor, three-factor, and four-factor models were also estimated: for the two-factor model, RMR = .04, RMSEA = .08 (90% CI: .07-.09), and TLI = .93; for the three-factor model, RMR = .03, RMSEA = .06 (90% CI: .05-.08), and TLI = .96; and for the four-factor model, RMR = .02, RMSEA = .07 (90% CI: .05-.08), and TLI = .95. Despite having marginally improved fit statistics, these models did not better explain the dimensional structure of the sensitivity to pain traumatization for pain-free participants than the one-factor model because (1) certain items did not clearly load on additional factors and (2) the additional factors were not meaningfully interpretable. The factor loadings and communality estimates for the two-factor, three-factor, and four-factor models are shown in Table 2.5.

Participants reporting ongoing pain. For participants reporting ongoing pain (n = 268), the scree plot suggested a one-factor model (see Figure 2) and parallel analysis suggested a three-factor model. For the one-factor model, RMR = .06, RMSEA = .10 (90% CI: .08-.11), and TLI = .89; thus, there is mixed evidence for the fit of the one-factor model.

The factor loadings and communality estimates for the one-factor model are shown in Table 2.6. The largest residual correlations were between item 1 and item 2 (r = .16), between item 4 and item 9 (r = -.11), between item 8 and item 11 (r = .12), and between item 9 and item 12 (r = .16).

Two-factor, three-factor, and four-factor models were also estimated: for the two-factor model, RMR = .04, RMSEA = .08 (90% CI: .06-.10), and TLI = .92; for the three-factor model, RMR = .03, RMSEA = .07 (90% CI: .05-.09), and TLI = .94. The four-factor model obtained an improper solution and therefore is not reported. Compared with the one-factor model, these multi-factor models did not appear to better account for the dimensional structure of the SPTS-12 for participants reporting ongoing pain because (1) certain items did not clearly load on additional factors and (2) the additional factors were not meaningfully interpretable, despite the fit statistics and parallel analysis suggesting a three-factor model. The factor loadings and communality estimates for the two-factor and three-factor models are in Table 2.7.

Reliability. Internal consistency reliability for the STPS-12 total score (based on a one-factor model) was calculated using McDonald's omega, which equaled .89 both for pain-free participants and for participants reporting ongoing pain.

Validity. The correlations between SPTS-12 total scores and other measures for individuals reporting ongoing pain and reporting no pain are shown in Tables 8 and 9.

Convergent validity. The Spearman correlation between the SPTS-12 and the PCL-C total scores was moderate for both pain-free participants, $r(551) = .53$, $p < .001$, and for participants reporting ongoing pain, $r(268) = .50$, $p < .001$.

***Divergent validity*.** The Spearman correlation between the SPTS-12 and the BDI-II total scores was moderate for both pain-free participants, $r(552) = .44$, $p < .001$, and for participants reporting ongoing pain, $r(268) = .40$, $p < .001$.

For pain-free participants, the correlation between the SPTS-12 and PCL-C was significantly greater than the correlation between the SPTS-12 and the BDI-II ($z = 1.98$, $p = .048$; Lee & Preacher, September, 2013). For participants reporting ongoing pain, the correlation between the SPTS-12 and PCL-C did not significantly differ from the correlation between the SPTS-12 and the BDI-II ($z = 1.31$, $p = .19$).

Known-groups validity. The SPTS-12 did not differ significantly between individuals with no pain ($M = 14.25$, $SD = 9.19$) and those with pain ($M = 14.65$, $SD = 9.35$), $t(820) = -0.58$, $p = .56$.

Discussion

The present study was designed to evaluate the factor structure, reliability, and validity of the SPTS-12 in a sample of undergraduate students. The results indicate that a single factor model adequately represents the internal dimensional structure of the SPTS-12 for both individuals reporting ongoing pain and individuals that are pain-free, along with very good internal reliability estimates for both samples. As assessed with the correlation between the SPTS-12 and the PCL-C, the SPTS-12 showed good evidence for convergent validity for participants without pain; furthermore, because this correlation was significantly greater than the relationship between the SPTS-12 and the BDI-II, it demonstrated good divergent validity.

However, for participants with pain, the correlation between the SPTS-12 and the PCL-C did not significantly differ from the correlation between the SPTS-12 and the BDI-II. Taken together, these findings provide initial support for the overall validity the SPTS-12 as a measure of a distinct sensitivity to pain traumatization construct, in that validation is an ongoing process (e.g., Cizek, 2012).

The one-factor model for the SPTS-12 items demonstrated adequate fit among individuals without pain. The factor loadings were high, ranging from .46 to .78 with communality estimates ranging from .21 to .61. Notably, the high residual correlations between items 4 and 9 as well as item 9 and 12 suggests that these items may be influenced by minor factors above and beyond the single common factor. However, when models with more factors were estimated, the resulting factor loading pattern was interpretationally ambiguous. For example, for the three-factor model, six items had loadings above .25 on two or more factors, and it was unclear how items with stronger loadings on the second and third factors were related. For instance, items 8, 9, 11, and 12 had loadings above .42 on the second factor; although items 8 (“when I'm in pain, I feel distant from people even when I'm talking to them”) and 11 (“when I'm in pain, things don't feel real”) appear to be related to emotional numbing, it is unclear how these two items relate to item 9 (“as soon as the pain comes on, I take medications to reduce it”), which is a behavioural response to pain, and item 12 (“I feel sick to my stomach when I am in pain”), which is a heightened awareness of sensations. Consequently, it is likely that other, minor dimensions exist among the 12 items of the SPTS-12, but that they are not better described by the models with more than one common factor.

Similarly, the one-factor model for the SPTS-12 items demonstrated adequate fit among individuals reporting ongoing pain with factor loadings ranging from .55 and .77 and

communality estimates between .30 and .59. Despite this strong finding, it is important to note that four of the residual correlations between items exceeded .10. However, the two- and three-factor models had multiple items with cross-loadings and did not produce substantially improved communality estimates. This finding demonstrates that although there may be additional minor factors, the one-factor model remains the best explanation of the data. One such minor factor may be reflected in Factor 1 of the three-factor solution, on which items 4 (“When I feel pain, I'm scared that it's the beginning of a terrible problem”) and 10 (“Pain sensations terrify me”) had high loadings, reflecting a fear of pain factor. However, it was unclear how these items were conceptually related to other items with higher loadings on this same factor.

Support for a one-factor model for both individuals with and without pain is consistent with previous research on sensitivity to pain traumatization. In the initial development of the SPTS-12 (Roosen, 2009), the scale was completed by a sample of 105 university students. An EFA determined that a one-factor model was the best fit for the data. However, due to the small sample size, further analysis to determine if this factor structure would be similar for a sub-sample of individuals experiencing ongoing pain was not possible. Similarly, Kleiman et al. (2011) examined the hierarchical factor structure of pain-related anxiety measures, predicting that trauma symptoms were closely linked to pain. In a sample of 444 patients scheduled to undergo surgery, they conducted an EFA on 49 items of three anxiety and pain related scales: the PASS-20, the PCS, and the ASI. They found that a factor composed of 20 items accounted for 68.3% of shared variance among the items. The authors suggested that this factor represented sensitivity to pain traumatization factor and, similar to the current study, found that the total score of the 20 items correlated highly with the PCL-C for both a subset of patients with pain (*r*

= .49) and without pain (r = .48). Thus, there is compelling evidence from the present study and previous research that there is a single factor that encompasses a traumatic response to pain.

In contrast, the literature on the factor structure of PTSD symptoms independent of whether individuals also experience pain is mixed. The Diagnostic and Statistical Manual of Mental Disorders, 4th edition (American Psychiatric Association, 2000) defined PTSD as consisting of 17 symptoms represented on the PCL-C as belonging to three categories: reexperiencing, effortful avoidance and emotional numbing, and hyperarousal. More recent factor analyses do not support the three-factor model, instead favouring four-factor models (Asmundson, Stapleton, & Taylor, 2004; Elhai & Palmieri, 2011). A popular new model includes the emotional numbing PTSD model (King, Leskin, King, & Weathers, 1998), which suggests that avoidance and emotional numbing should be considered separate factors, with the four symptoms clusters including a reexperiencing factor, arousal factor, avoidance factor, and emotional numbing factor. Although this model has received support in a variety of populations (Elhai, Ford, Ruggiero, & Frueh, 2009; Mansfield, Williams, Hourani, & Babeu, 2010; Schinka, Brown, Borenstein, & Mortimer, 2007), it is not universally accepted. Simms, Watson, and Doebbelling (2002) propose a four-factor dysphoria model of PTSD which preserves factors for reexperiencing, avoidance, and arousal, but introduces a new dysphoria factor that includes symptoms of emotional numbing.

In response to recent research, the newest edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) revised the diagnostic criteria for PTSD to include four, rather than three, primary symptoms clusters: intrusion symptoms/reexperiencing, avoidance, alterations to mood and cognitions, and alterations to arousal and reactivity. The changes included omitting the term “emotional

numbing" in favour of more mood-oriented symptoms, such as "feelings of detachment or estrangement from others" and "persistent negative emotional state" (American Psychiatric Association, 2013). In an evaluation of the factor structure of the newly defined diagnostic criteria, Miller et al. (2013) found that the structural model demonstrated "adequate, albeit not excellent, fit to the data" (p. 10) in both a community sample of adults (n = 2,953) and in a clinical sample of military veterans (n = 345). Furthermore, based on a factor analysis of a sample of 1484 veterans using the new DSM-5 criteria, Tsai, Armour, Southwick, and Pietrzak (2015) proposed a six-factor model of PTSD. The authors suggested that six factors consisting of reexperiencing, avoidance, emotional numbing, externalizing behaviours, dysphoric arousal, and anxious arousal demonstrated the best fit to their data. Taken together, these studies reflect how our understanding of the factor structure and the specific diagnostic criteria for PTSD is not definite and evolves dynamically with emerging research.

When the PCL-C has been examined in a population of individuals with pain, the number of factors that best explain the data can vary. Pagé, Kleiman, Asmundson, and Katz (2009) investigated the factor structure of the PCL-C for a sample of individuals scheduled to undergo surgery. Two EFAs were conducted, one in a subsample of patients reporting pain (n = 175) and another in a sample that was pain-free (n = 272). For individuals with pain, a one-factor model had the best fit whereas a two-factor model had the best fit for individuals without pain, with a reexperiencing/avoidance factor and an emotional numbing/hyperarousal factor. As higher PCL-C scores were observed for participants with pain, the authors proposed that pain may contribute to a worsening of all trauma symptoms, suggesting that pain may serve as a higher-order factor for individuals experiencing pain. Because the SPTS-12 assesses pain-related traumatic symptoms, the findings in the present study showing an adequate fit for a one-factor

model is consistent with the Pagé et al. (2009) findings, suggesting that pain serves as a higher-order factor that affects cognitive, emotional, behavioural, and somatic dimensions of trauma.

It is noteworthy that the item with the poorest association with its common factors for individuals without pain (factor loading = .46, communality estimate = .21) and for individuals reporting ongoing pain (factor loading = .55, communality estimate = .30) was item 9, "as soon as the pain comes on, I take medications to reduce it". Although the communality estimates for this item improved for the two-, three-, and four-factor models, it only exceeded .4 for the four-factor model that did not provide a meaningfully interpretable factor structure. In the development of the SPTS-12, item 9 was included because it provides a measure of pain avoidance/escape that is an important theoretical dimension of a traumatic stress response: a diagnosis of PTSD in the DSM-5 requires that avoidance of thoughts, emotions, or other reminders of the trauma be present. Similarly, avoidance and pain-related escape is a fundamental characteristic in the Fear Avoidance Model of Pain (Asmundson, Norton, & Norton, 1999; Vlaeyen & Linton, 2000, 2012); in response to an injury, individuals may interpret their pain as being threatening, developing a fear of pain. This interpretation can subsequently lead to pain-related escape (e.g., medication use) and avoidance of activities which cause pain (e.g., lifting boxes), contributing to disuse, disability, and low mood. Avoidance is therefore implicated as a central mechanism contributing to pain as well as a diagnostic characteristic of a traumatic stress response. As such, even though this item showed the least robust association with the common factors, it represents an important theoretical dimension of the SPTS-12 that provides clinically meaningful information.

Internal consistency reliability for the SPTS-12 total score was very good for both individuals with pain (.89) and those without pain (.89), demonstrating that the total score

represents the construct hypothesized to be sensitivity to pain traumatization with minimal measurement error. This result is consistent with the initial validation of the SPTS-12, in which Roosen (2009) found that the scale had a high internal consistency (coefficient alpha = .91). Although it has been suggested that clinical measures should meet more stringent criteria for internal consistency, with Nunnally (1967) suggesting that .90 is the "minimally tolerable estimate" (p. 226), more recently Streiner (2003) stated that when internal consistency is too high, it may reflect redundancy in the scale items.

For participants without pain, the SPTS-12 has good convergent validity through a moderate correlation with the PCL-C (r = .53), a measure of trauma symptoms. This finding reflects the similar domains that are assessed with the items on both measures, including emotional numbing, hyperarousal, avoidance, and intrusive thoughts, although the items on the SPTS-12 are specific to the experience of pain. This strong relationship is supported by the Mutual Maintenance Model which suggests that the physiological, affective, and behavioural symptoms of PTSD can worsen pain, which can in turn mutually maintain traumatic symptoms (Sharp & Harvey, 2001). Further, the SPTS-12 showed good divergent validity with a significantly weaker correlation with the BDI-II (r = .44), a measure of a related but distinct construct from sensitivity to pain traumatization.

In contrast, for participants reporting ongoing pain, the correlation between the SPTS-12 and PCL-C (r = .50) was not significantly larger than the correlation between the SPTS-12 and BDI-II (r = .40). This result may reflect that the comorbidity rates between pain and depression tend to be both be higher when either is present – that is, rates of depression are higher in individuals who also experience pain (Fishbain, Cutler, Rosomoff, & Rosomoff, 1997). For example, Currie and Wang (2004) found that in a sample of 118,533 Canadians, depression rates

were much higher for individuals suffering from chronic back pain (19.8%) compared to the general population (5.9%). Furthermore, they found that the more severe the pain is, the higher the rate of major depression symptoms. In the present study, BDI-II scores were significantly higher for individuals reporting ongoing pain (M = 16.80, SD = 11.45) than those reporting no pain (M = 13.58, SD = 10.53), $t(819) = -3.99$, $p < .001$ (see Table 2.10). Furthermore, the suggested cut-off scores for the BDI-II are 0 to 13 for minimal depressive symptoms and 14 to 19 for moderate depressive symptoms (Beck et al., 1996). Thus, on average, the reported depressive symptoms of individuals with pain are in the moderate range in contrast to those without pain who tend to be closer to the minimal depressive symptoms range. Therefore, a measure of depression may be sufficiently disparate concept from sensitivity to pain traumatization when individuals are not experiencing ongoing pain, but when pain is present, symptoms of depression become more highly related to pain.

Because the SPTS-12 was developed to measure the traumatic response that an individual may experience in response to pain, it was anticipated that individuals with ongoing pain would report higher levels of sensitivity to pain traumatization. Yet, the total score on the SPTS-12 did not differ significantly between individuals with and without pain and therefore did not demonstrate known-groups validity in the present sample. This result is inconsistent with previous research on the sensitivity to pain traumatization construct: in their validation of a 20-item version of the scale, Kleiman et al. (2011) found that in a sample of 444 patients scheduled to undergo surgery, those that indicated that they experienced ongoing pain problems scored higher on the SPTS-20 than those without an ongoing pain problem. In the present study, it is possible that the difference was weak because the type of pain experienced by the majority of the pain group was sufficiently mild not to warrant a traumatic response: most of the pain group

reported experiencing mild to moderate pain with no to little pain interference. For individuals who experience more frequent, intense, and interfering pain, it is possible that the SPTS-12 will elicit higher scores. Notably, the groups also did not differ significantly on the ASI, FPQ-III, ISI-R, and PASS-20, although they did differ significantly on the PCL-C, PCS, STAI-T, and BDI-II (see Table 2.10). These findings may reflect the nature of the differences between the groups in that the questionnaires that did not detect a significant difference assess fear of pain. Specifically, the ASI measures a fear of bodily sensations related to anxiety (Reiss et al., 1986), the FPQ-III measures a fear of experiencing pain in response to different painful experiences (McNeil & Rainwater, 1998), the ISI-R measures a fear of illness and injury (Taylor, 1993), the PASS-20 measures fear of pain and pain-related sensations (McCracken & Dhingra, 2002), and the SPTS-12 include several items that assess fear of experiencing pain and pain sensitivity. In contrast, the questionnaires showing a difference between groups emphasize more emotional symptoms, with the BDI-II measuring symptoms of depression, the PCL-C measuring symptoms of traumatic stress, the PCS measuring negative reactions to the experience or anticipation of pain (Sullivan et al., 1995), and the STAI-T measuring trait characteristics of anxiety (Spielberger et al., 1983). In summary, for the present sample of young undergraduate students, the total score on the SPTS-12 did not differ significantly between individuals with and without pain.

The present study has several limitations. First, although the overall fit statistics for the one-factor model SPTS-12 items were strong, there were some fairly large residual correlations for participants both with and without pain. As the SPTS-12 was intentionally designed to assess a wide breadth of symptoms to capture the range of anxiety-related cognitive, emotional, behavioural, and somatic responses to pain, it includes items that assess six major categories of symptoms: pain and emotional numbing, pain and hyperarousal, pain avoidance, pain

experiencing/intrusive thoughts, sensitivity to pain, and fear of pain – importantly, this was considered acceptable to have an adequate range for content validity. It is noteworthy that overall, this breadth may contribute to a poorer fit for subsequent confirmatory factor analyses. For future research, it will be important to consider the possibility that additional minor dimensions may be relevant in other samples, as the validity of the SPTS-12 may be diminished if the SPTS-12 is incorrectly assumed to be a unidimensional construct.

Second, participants who reported experiencing ongoing pain comprised a heterogenous sample: it is possible that the high variability in the severity, duration, frequency, and functional impact of pain symptoms may have impacted the distribution of scores on the SPTS-12 (see Figures 3 to 6). For example, 34% of participants reported that their pain interfered moderately or severely with their ability to engage in everyday activities compared to 66% who indicated that they experience no or only slight interference. Similarly, 38.1% reported they experienced pain daily, 37.7% reported they experienced pain weekly, and 16.4% reported they experienced pain monthly. These differences are reflected in the responses to the type of pain that individuals reported experiencing, which ranged from occasional migraines that are mild in intensity to daily neck and back pain that are severe in intensity. For a subset of participants whose symptoms are more severe, persistent, frequent, and interfering, it is possible that a factor analysis would suggest the retention of more than one factor or strengthen the finding that pain acts as a higher-order factor across trauma symptoms when it is present (Pagé et al., 2009). Furthermore, it is possible that the SPTS-12 will demonstrate higher convergence with the PCL-C and worse divergent validity with measures such as the BDI-II due to the higher comorbidity when symptoms of trauma, depression, and pain are present (Chibnall & Duckro, 1994; Geisser, Roth, Bachman, & Eckert, 1996; Otis et al., 2003). In the current study, the sample size was

insufficient to explore if one or more of these qualities of experiencing pain affects the factor structure, validity, and reliability of the SPTS-12. Future research on the SPTS-12 should continue to evaluate the psychometric qualities of the scale to determine the efficacy of its use for other populations and samples. The present study examined a sample of undergraduate students some of which reported experiencing ongoing pain. It would be meaningful to examine the scale properties in a clinical sample of participants, especially for those with more significant pain symptoms with respect of severity, frequency, and disability.

Future studies should consider evaluating an alternate statement for item 9, “as soon as the pain comes on, I take medications to reduce it”. This item was included to assess pain avoidance, which is important to capturing the breadth of a traumatic response to pain. It demonstrated the poorest association with the sensitivity to pain traumatization factor and it is possible that another item could be written that also assesses pain avoidance and is more strongly related with the other items of the scale. The challenge of developing an alternative item is in part due to the lack of a clear understanding of what is meant by avoidance behaviour (Volders, Boddez, De Peuter, Meulders, & Vlaeyen, 2015) and that specific avoidant behaviours may vary from person to person. A possible source for an alternative item is the pain behaviour item bank that was developed as a part of the Patient-Reported Outcome Measurement Information System (PROMIS; Revicki et al., 2009). In particular, pain behaviours that have been classified as being more controlled and intentional may be appropriate, such as "Tried to stay very still" and "Isolated her/himself from others" (McCrystal, Craig, Versloot, Fashler, & Jones, 2011), with the wording modified to fit with the other questions of the SPTS-12.

Lastly, the clinical utility of the SPTS-12 should be evaluated in future research. In particular, it would be informative to determine the predictive validity of the SPTS-12 for related emotional and

physical symptoms, such as self-reported pain, opioid use, pain-related disability, and depression. If the SPTS-12 shows predictive utility, then it may provide the opportunity for interventions to be introduced to individuals with high scores of SPTS-12.

Conclusion

The present study examined the psychometric properties of the SPTS-12, a scale developed to assess the cognitive, emotional, behavioural, and somatic responses to pain that are similar to a traumatic stress response. The factor structure, reliability, and validity were evaluated in a sample of undergraduate students who were pain-free or reported experiencing ongoing pain. For both groups, the one-factor model demonstrated adequate overall fit and the SPTS-12 total score also showed excellent reliability and good convergent validity with the PCL-C. As assessed by the magnitude of the correlation between the SPTS-12 and a similar construct (PCL-C) compared to the magnitude of the correlation between the SPTS-12 and a measure of depression symptoms (BDI-II), the SPTS-12 showed good divergent validity for pain-free participants, but not for participants reporting ongoing pain.

Table 2.1. Descriptive statistics for the 12 items of the Sensitivity to Pain Traumatization Scale (SPTS-12) for participants with and without ongoing pain.

		No pain (n = 555)				Ongoing pain (n = 268)			
	SPTS-12 item	*M*	*SD*	*Skewness*	*Kurtosis*	*M*	*SD*	*Skewness*	*Kurtosis*
1.	When I am in pain, it keeps me awake at night.	1.41	1.06	.39	-.64	1.63	1.12	.27	-.67
2.	When I am in pain, everything I see or do reminds me of the pain.	.93	1.00	.86	-.15	1.00	1.05	.87	.01
3.	I try to avoid activities that cause pain.	1.77	1.24	.19	-1.00	1.61	1.27	.29	-1.03
4.	When I feel pain, I'm scared that it's the beginning of a terrible problem.	1.14	1.13	.68	-.53	1.23	1.17	.72	-.35
5.	Pain seems to bother me more than it does other people.	.84	1.09	1.16	.48	.96	1.09	.91	-.14
6.	When I feel pain, I think about it even when I don't mean to.	1.20	1.08	.61	-.49	1.24	1.09	.60	-.50
7.	I can't stand pain.	1.57	1.27	.42	-.92	1.55	1.26	.43	-.86
8.	When I'm in pain, I feel distant from people even when I'm talking to them.	1.19	1.14	.65	-.52	1.28	1.25	.69	-.62
9.	As soon as the pain comes on, I take medications to reduce it.	1.27	1.31	.73	-.67	1.07	1.23	.94	-.22
10.	Pain sensations terrify me.	1.18	1.15	.74	-.32	1.16	1.13	.66	-.45
11.	When I'm in pain, things don't feel real.	.81	1.06	1.22	.70	.86	1.09	1.03	-.04
12.	I feel sick to my stomach when I am in pain.	.94	1.06	1.05	.43	1.06	1.14	.86	-.19

Note. SPTS-12 item scores for Study 1 range from 0 to 4.

Table 2.2. Polychoric correlation matrix of the 12 items of the Sensitivity to Pain Traumatization Scale (SPTS-12) for participants reporting no ongoing pain (*n* = 555).

	1	2	3	4	5	6	7	8	9	10	11	12
1	-											
2	.54	-										
3	.39	.43	-									
4	.44	.56	.45	-								
5	.42	.50	.45	.54	-							
6	.45	.61	.48	.62	.63	-						
7	.42	.43	.45	.42	.57	.53	-					
8	.42	.51	.43	.50	.47	.59	.51	-				
9	.26	.32	.33	.22	.31	.29	.35	.33	-			
10	.42	.51	.43	.57	.56	.54	.62	.54	.37	-		
11	.48	.54	.36	.52	.50	.54	.43	.60	.36	.56	-	
12	.45	.55	.35	.49	.50	.53	.48	.55	.44	.56	.59	-

Table 2.3. Polychoric correlation matrix of the 12 items of the Sensitivity to Pain Traumatization Scale (SPTS-12) for participants reporting ongoing pain (*n* = 268).

	1	2	3	4	5	6	7	8	9	10	11	12
1	-											
2	.59	-										
3	.33	.37	-									
4	.43	.52	.48	-								
5	.39	.47	.41	.56	-							
6	.40	.55	.40	.58	.57	-						
7	.43	.45	.41	.43	.46	.47	-					
8	.34	.55	.33	.42	.42	.58	.46	-				
9	.29	.36	.34	.28	.38	.35	.45	.35	-			
10	.39	.52	.51	.61	.51	.55	.59	.50	.45	-		
11	.35	.60	.35	.45	.49	.51	.51	.64	.41	.53	-	
12	.33	.48	.27	.35	.40	.45	.44	.50	.51	.47	.56	-

Table 2.4. Factor loadings and communality estimates for the one-factor solution for the 12 items of the Sensitivity to Pain Traumatization Scale (SPTS-12) for participants without ongoing pain (n = 555).

	SPTS-12 item	Factor Loading	Communality Estimate
1.	When I am in pain, it keeps me awake at night.	.61	.38
2.	When I am in pain, everything I see or do reminds me of the pain.	.73	.53
3.	I try to avoid activities that cause pain.	.59	.35
4.	When I feel pain, I'm scared that it's the beginning of a terrible problem.	.71	.51
5.	Pain seems to bother me more than it does other people.	.73	.53
6.	When I feel pain, I think about it even when I don't mean to.	.78	.61
7.	I can't stand pain.	.69	.47
8.	When I'm in pain, I feel distant from people even when I'm talking to them.	.72	.52
9.	As soon as the pain comes on, I take medications to reduce it.	.46	.21
10.	Pain sensations terrify me.	.76	.58
11.	When I'm in pain, things don't feel real.	.73	.53
12.	I feel sick to my stomach when I am in pain.	.73	.53

Table 2.5. Factor loadings and communality estimates for the two-factor, three-factor, and four-factor solutions for the 12 items of the Sensitivity to Pain Traumatization Scale (SPTS-12) for participants without ongoing pain (n = 555).

		Two-factor solution			Three-factor solution				Four-factor solution				
	SPTS-12 item	factor 1	factor 2	h^2	factor 1	factor 2	factor 3	h^2	factor 1	factor 2	factor 3	factor 4	h^2
1.	When I am in pain, it keeps me awake at night.	0.61	0.03	0.38	0.34	0.29	0.04	0.38	0.53	-0.01	0.13	0.10	0.41
2.	When I am in pain, everything I see or do reminds me of the pain.	0.74	-0.02	0.54	0.52	0.34	-0.06	0.58	0.72	-0.11	0.18	0.06	0.63
3.	I try to avoid activities that cause pain.	0.59	0.01	0.35	0.33	0.05	0.30	0.36	0.54	0.27	-0.19	0.17	0.44
4.	When I feel pain, I'm scared that it's the beginning of a terrible problem.	0.80	-0.21	0.59	0.73	0.04	0.03	0.60	0.54	0.15	0.15	-0.20	0.60
5.	Pain seems to bother me more than it does other people.	0.74	-0.05	0.54	0.46	0.01	0.37	0.57	0.33	0.46	0.05	-0.06	0.57
6.	When I feel pain, I think about it even when I don't mean to.	0.85	-0.16	0.67	0.67	0.05	0.17	0.67	0.54	0.26	0.10	-0.12	0.66
7.	I can't stand pain.	0.64	0.14	0.48	0.04	0.04	0.79	0.71	0.02	0.74	0.05	0.09	0.64
8.	When I'm in pain, I feel	0.68	0.12	0.52	0.26	0.42	0.13	0.53	0.18	0.22	0.42	-0.01	0.53

	distant from people even when I'm talking to them.											
9.	As soon as the pain comes on, I take medications to reduce it.	0.33	0.42	0.35	-0.23	0.54	0.22	0.31	0.03	0.15	0.26	0.44	0.42
10.	Pain sensations terrify me.	0.71	0.14	0.58	0.21	0.28	0.39	0.59	0.03	0.52	0.34	-0.02	0.63
11.	When I'm in pain, things don't feel real.	0.67	0.16	0.54	0.23	0.63	-0.05	0.60	0.16	0.04	0.65	0.00	0.63
12.	I feel sick to my stomach when I am in pain.	0.63	0.30	0.59	0.02	0.73	0.06	0.63	0.09	0.12	0.60	0.14	0.62

Note. h^2 = communality estimate.

Table 2.6. Factor loadings and communality estimates for the one-factor solution for the 12 items of the Sensitivity to Pain Traumatization Scale (SPTS-12) for participants with ongoing pain (n = 268).

	SPTS-12 item	Factor Loading	Communality Estimate
1.	When I am in pain, it keeps me awake at night.	.57	.33
2.	When I am in pain, everything I see or do reminds me of the pain.	.74	.55
3.	I try to avoid activities that cause pain.	.56	.31
4.	When I feel pain, I'm scared that it's the beginning of a terrible problem.	.70	.48
5.	Pain seems to bother me more than it does other people.	.68	.47
6.	When I feel pain, I think about it even when I don't mean to.	.74	.55
7.	I can't stand pain.	.69	.47
8.	When I'm in pain, I feel distant from people even when I'm talking to them.	.70	.49
9.	As soon as the pain comes on, I take medications to reduce it.	.55	.30
10.	Pain sensations terrify me.	.77	.59
11.	When I'm in pain, things don't feel real.	.74	.54
12.	I feel sick to my stomach when I am in pain.	.64	.41

Table 2.7. Factor loadings and communality estimates for the two-factor and three-factor solutions for the 12 items of the Sensitivity to Pain Traumatization Scale (SPTS-12) for participants reporting ongoing pain (n = 268).

		Two-factor solution			Three-factor solution			
	SPTS-12 item	factor 1	factor 2	h^2	factor 1	factor 2	factor 3	h^2
1.	When I am in pain, it keeps me awake at night.	0.20	0.42	0.33	0.37	0.26	-0.01	0.34
2.	When I am in pain, everything I see or do reminds me of the pain.	0.45	0.36	0.55	0.24	0.63	-0.10	0.61
3.	I try to avoid activities that cause pain.	0.04	0.57	0.36	0.63	-0.11	0.18	0.39
4.	When I feel pain, I'm scared that it's the beginning of a terrible problem.	-0.11	0.90	0.67	0.83	0.03	-0.11	0.67
5.	Pain seems to bother me more than it does other people.	0.19	0.55	0.49	0.53	0.17	0.08	0.48
6.	When I feel pain, I think about it even when I don't mean to.	0.30	0.51	0.56	0.43	0.40	-0.04	0.57
7.	I can't stand pain.	0.41	0.33	0.47	0.38	0.18	0.30	0.50
8.	When I'm in pain, I feel distant from people even when I'm talking to them.	0.64	0.12	0.53	0.00	0.78	-0.02	0.59
9.	As soon as the pain comes on, I take medications to reduce it.	0.54	0.05	0.34	0.13	0.11	0.62	0.55
10.	Pain sensations terrify me.	0.27	0.56	0.60	0.62	0.08	0.24	0.63
11.	When I'm in pain, things don't feel real.	0.73	0.07	0.62	0.00	0.76	0.08	0.64
12.	I feel sick to my stomach when I am in pain.	0.81	-0.11	0.55	-0.07	0.58	0.32	0.54

Note. Results of the four-factor model is not reported because it obtained an improper solution. h^2 = communality estimate.

Table 2.8. Spearman correlation matrix for participants reporting no pain (*n* range: 547-554).

	SPTS-12	ASI	BDI-II	FPQ-III	ISI-R	PASS-20	PCL-C	PCS
ASI	.619							
BDI-II	.436	.500						
FPQ-III	.447	.362	.215					
ISI-R	.614	.519	.321	.482				
PASS-20	.752	.577	.397	.409	.546			
PCL-C	.532	.580	.694	.285	.363	.484		
PCS	.652	.508	.429	.418	.523	.666	.485	
STAI-T	.368	.463	.704	.184	.334	.375	.587	.378

Note. All correlations significant, $p < .001$. ASI - Anxiety Sensitivity Index, BDI-II - Beck Depression Inventory - II, FPQ-III - Fear of Pain Questionnaire - III, ISI-R - Illness Sensitivity Index- Revised, PASS-20 - Pain Anxiety Symptoms Scale - Short Form, PCS - Pain Catastrophizing Scale, PCL-C - PTSD Checklist - Civilian Version, SPTS-12 – 12-item Sensitivity to Pain Traumatization Scale, STAI-T – Spielberger State-Trait Anxiety Inventory (Trait Subscale).

Table 2.9. Spearman correlation matrix for participants reporting ongoing pain (*n* range: 263-268).

	SPTS-12	ASI	BDI-II	FPQ-III	ISI-R	PASS-20	PCL-C	PCS
ASI	.594							
BDI-II	.401	.484						
FPQ-III	.467	.380	.120§					
ISI-R	.601	.589	.305	.484				
PASS-20	.841	.570	.390	.454	.626			
PCL-C	.496	.488	.672	.160*	.324	.456		
PCS	.730	.515	.437	.442	.590	.729	.499	
STAI-T	.412	.470	.737	.170*	.311	.400	.646	.426

Note. All correlations significant, $p < .001$, unless otherwise indicated. *$p < .01$. §$p = .052$. ASI - Anxiety Sensitivity Index, BDI-II - Beck Depression Inventory - II, FPQ-III - Fear of Pain Questionnaire - III, ISI-R - Illness Sensitivity Index- Revised, PASS-20 - Pain Anxiety Symptoms Scale - Short Form, PCS - Pain Catastrophizing Scale, PCL-C - PTSD Checklist - Civilian Version, SPTS-12 – 12-item Sensitivity to Pain Traumatization Scale, STAI-T – Spielberger State-Trait Anxiety Inventory (Trait Subscale).

Table 2.10. Descriptive statistics for questionnaire data and independent-samples *t*-tests.

	No pain (*n* range = 547-554)		Ongoing pain (*n* = 263-268)				
	M	*SD*	*M*	*SD*	*t*	*df*	*p*
ASI	22.24	11.42	23.76	11.83	-1.76	812	.079
BDI-II	13.58	10.53	16.80	11.45	-3.99	819	< .001
FPQ-III	85.18	22.50	85.44	20.36	-.160	808	.873
ISI-R	15.39	9.24	15.91	8.93	-.761	818	.447
PASS-20	40.36	19.35	40.28	19.75	.054	820	.957
PCL-C	36.46	12.96	41.51	14.00	-5.10	818	< .001
PCS	15.74	11.17	18.22	10.98	-2.99	819	.003
SPTS-12	14.25	9.19	14.65	9.35	-.582	820	.561
STAI-T	43.47	10.10	46.55	11.02	-3.98	819	< .001

Note. ASI - Anxiety Sensitivity Index, BDI-II - Beck Depression Inventory - II, FPQ-III - Fear of Pain Questionnaire - III, ISI-R - Illness Sensitivity Index- Revised, PASS-20 - Pain Anxiety Symptoms Scale - Short Form, PCS - Pain Catastrophizing Scale, PCL-C - PTSD Checklist - Civilian Version, SPTS-12 – 12-item Sensitivity to Pain Traumatization Scale, STAI-T – Spielberger State-Trait Anxiety Inventory (Trait Subscale).

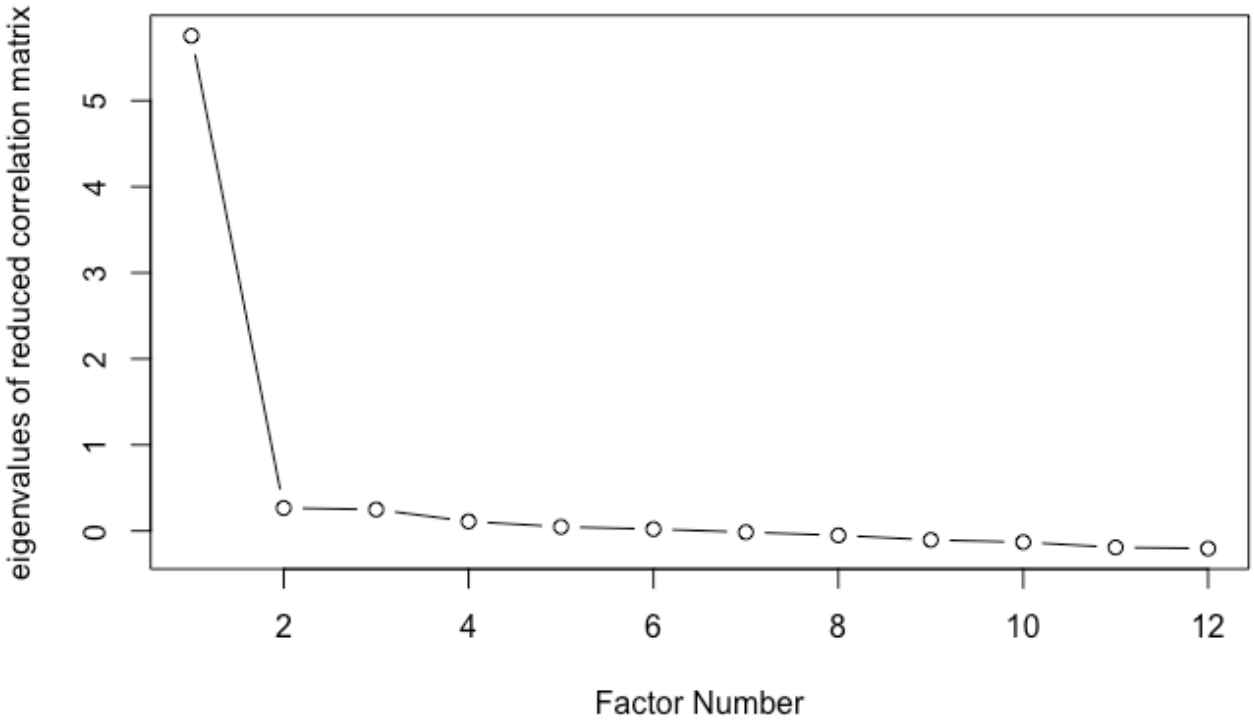

Figure 2.1. Scree plot of the eigenvalues of the reduced correlation matrix for participants reporting no pain (n = 555).

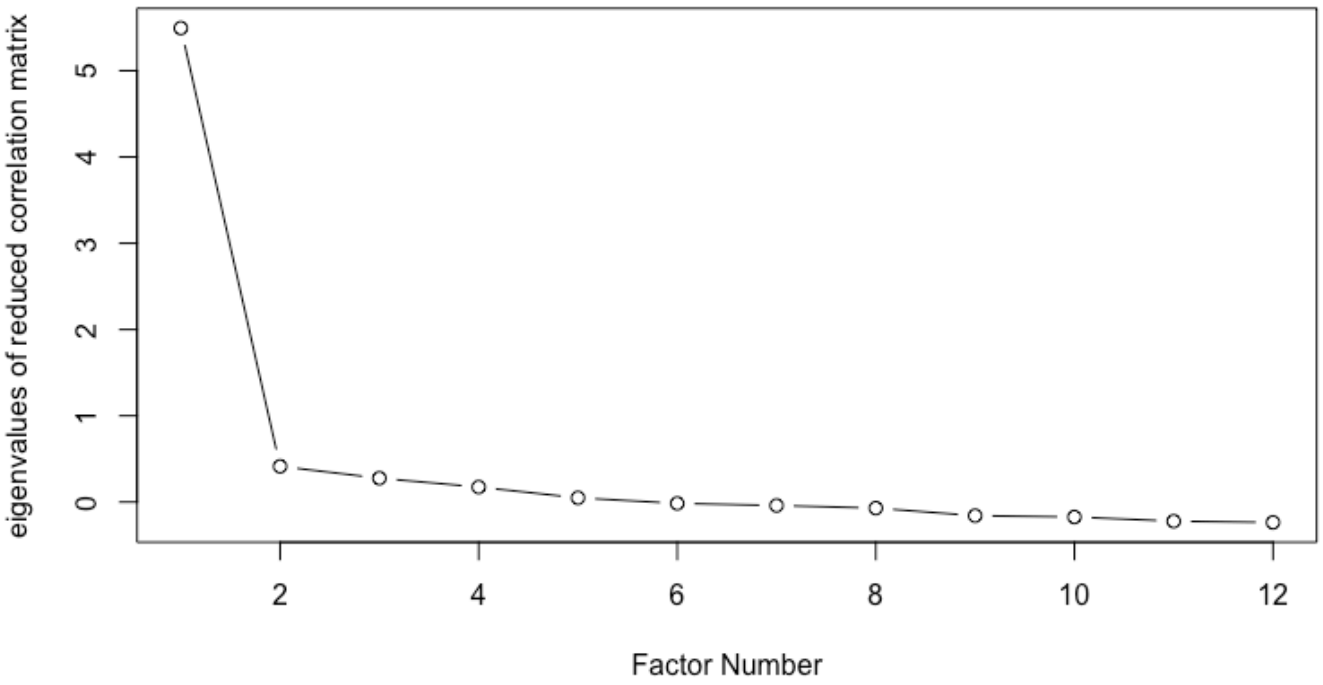

Figure 2.2. Scree plot of the eigenvalues of the reduced correlation matrix for participants reporting ongoing pain (n = 268).

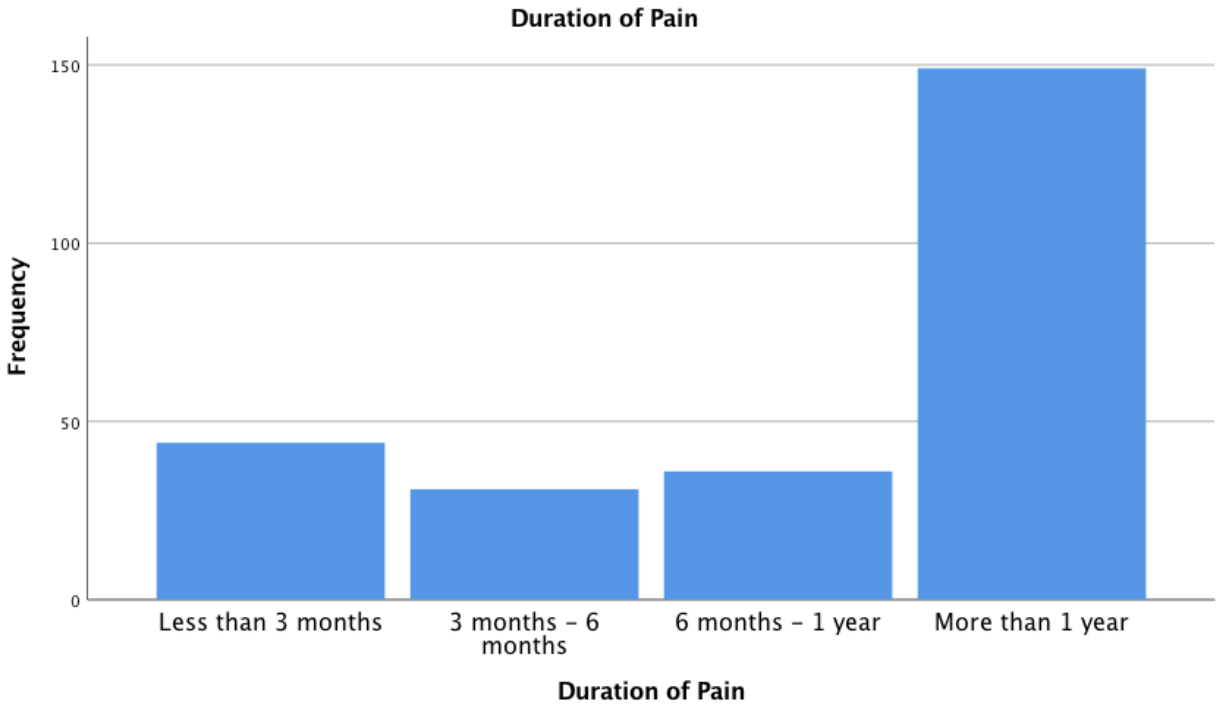

Figure 2.3. Bar chart of the frequency of responses for the duration that pain has been experienced by participants reporting ongoing pain (n = 260).

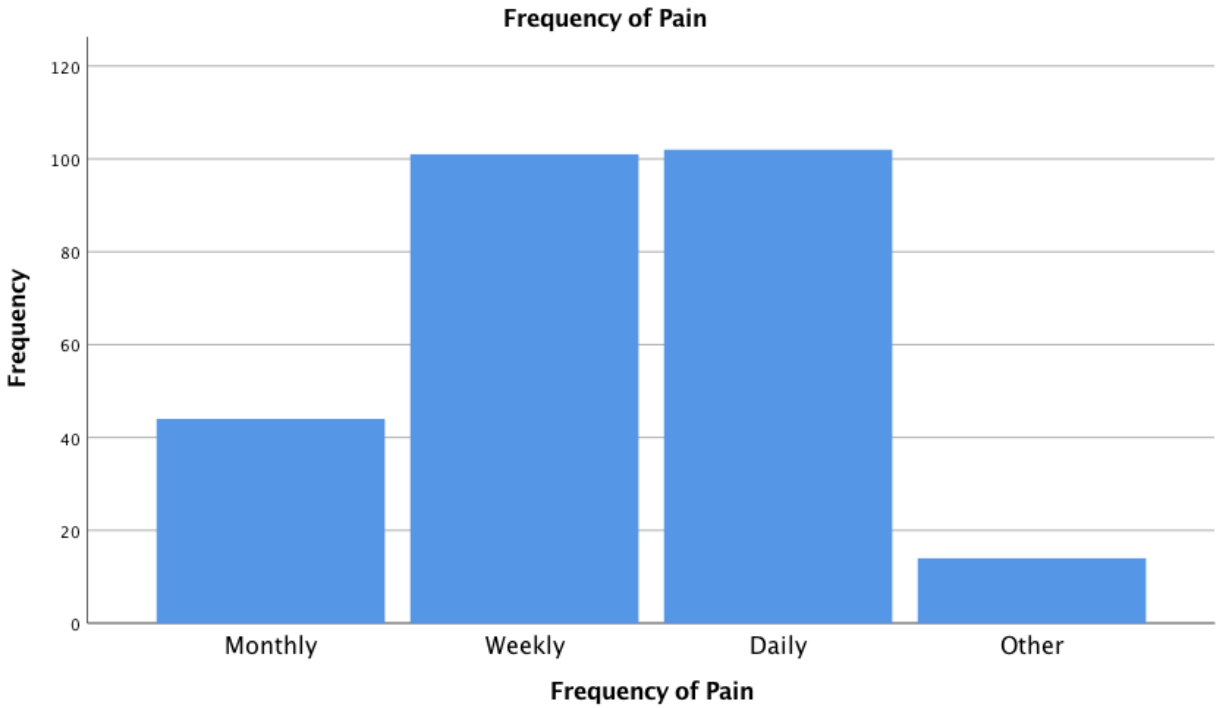

Figure 2.4. Bar chart of the frequency that pain is experienced by participants reporting ongoing pain (n = 261).

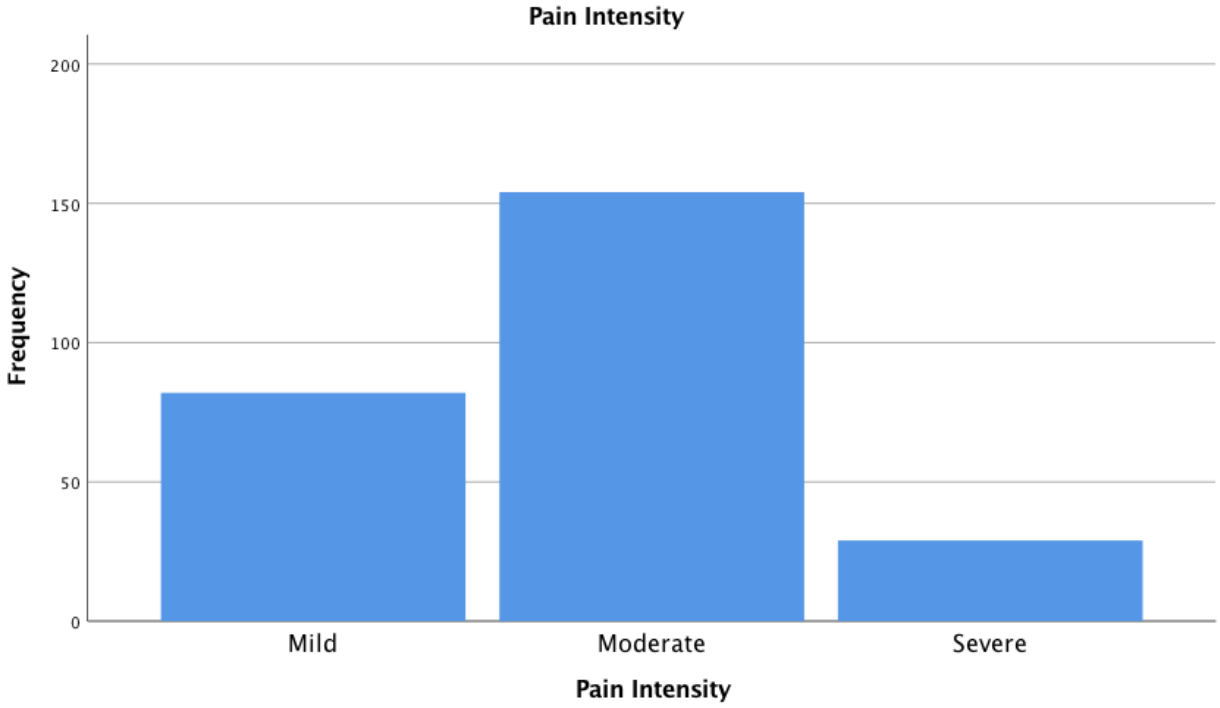

Figure 2.5. Bar chart of the frequency of responses for the pain intensity experienced by participants reporting ongoing pain (n = 265).

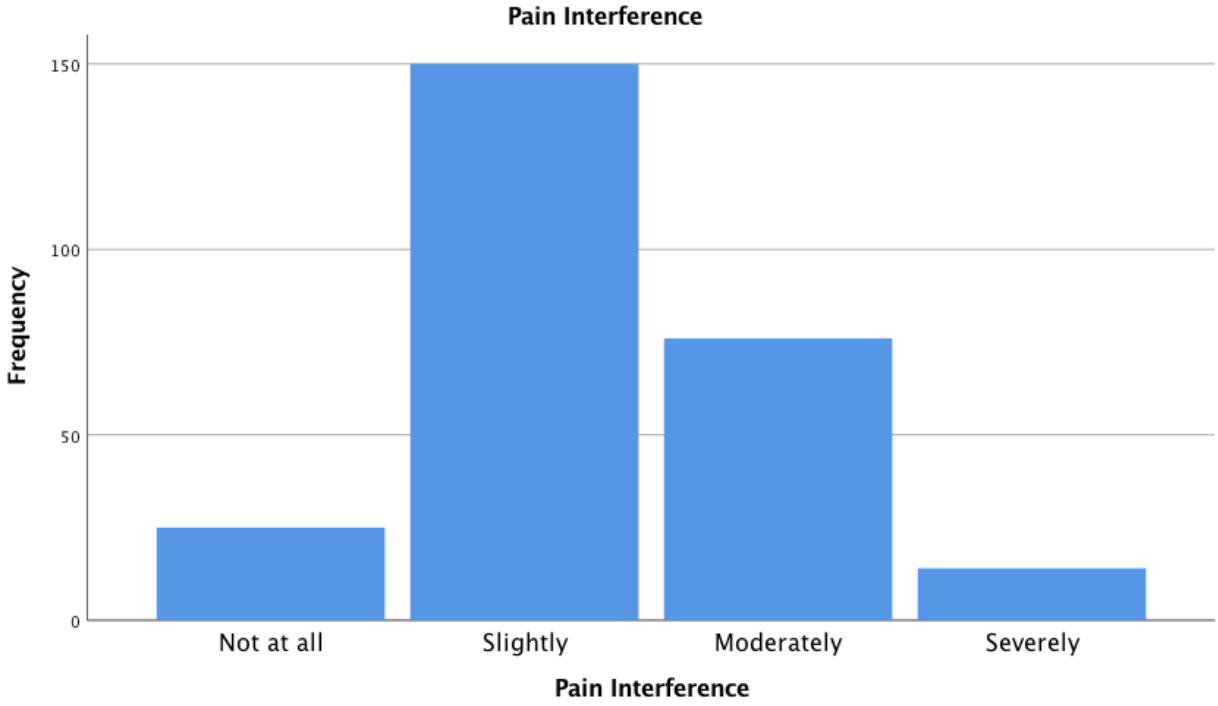

Figure 2.6. Bar chart of the frequency of responses for pain interference experienced by participants reporting ongoing pain (n = 265).

Chapter 3: Study 2

Disclosure Notes

A portion of the research reported in Study 2 has been published. The data were presented as a poster presentation at the Canadian Pain Society conference in May 2017 and an abstract of the poster presentation was published in the *Canadian Journal of Pain* (vol. 1, issue 1, pp. 93-94, 2017). All content included in the present study was authored by Samantha Fashler.

Introduction

There has been an increasing awareness in recent decades of the significant co-occurrence and symptom overlap of Posttraumatic Stress Disorder (PTSD) and pain (Asmundson et al., 2002; Asmundson & Katz, 2009; Katz et al., 2014). This overlap stems from the consistent finding that rates of PTSD are higher for individuals experiencing pain and that rates of pain are higher for individuals experiencing PTSD than would be expected in the general population (Cohen et al., 2002; Cox & McWilliams, 2002; Lew et al., 2009; McWilliams, Cox, & Enns, 2003; Roy-Byrne, Smith, Goldberg, Afari, & Buchwald, 2004; Sareen et al., 2007; Seedat & Stein, 2001; Siqveland, Hussain, Lindstrøm, Ruud, & Hauff, 2017). For example, a study using the Canadian Community Health Survey (Sareen et al., 2007) found that chronic pain conditions were frequently reported in a sample of 478 individuals with PTSD: 46% reported back problems, 43% reported arthritis, 34% reported migraine headaches, and 8% reported fibromyalgia. In contrast, the prevalence of pain problems in the general population is estimated to be 18.9% (Schopflocher et al., 2011). A recent meta-analysis looked at the prevalence of PTSD in individuals with chronic pain reported across 21 studies (Siqveland et al., 2017). They found a pooled estimate of comorbid PTSD in 57% of individuals with fibromyalgia, 20.5% of individuals reporting chronic widespread pain, and 11.2% of individuals reporting headache

pain. In the general population, the prevalence of PTSD is estimated to be between 7 to 12% (Seedat & Stein, 2001).

Building on these highly comorbidity prevalence estimates, several models have proposed mechanisms explaining the relationship between pain and PTSD. Shared vulnerability models suggest that particular symptoms may make individuals more susceptible to developing both conditions, most notably anxiety sensitivity (Asmundson et al., 2002). Anxiety sensitivity is defined as a fear that the symptoms of anxiety, such as a racing heart or light-headedness, will result in harmful consequences (Taylor, 2014). It is suggested that high levels of anxiety sensitivity can lead to more intense emotional reactions in response to a stressor and pain (Asmundson et al., 2002; Asmundson & Katz, 2009; Taylor, 2003). Mutual maintenance models suggest that various biopsychosocial variables maintain both chronic pain and PTSD; specifically, attentional and reasoning biases, anxiety sensitivity, reminders of the trauma, avoidance, depression and reduced activity levels, anxiety and pain perception, and cognitive demand from symptoms (Sharp & Harvey, 2001). Taken together, these models propose a complicated interplay between pre-existing factors (e.g., genetic predispositions), vulnerabilities (e.g., anxiety sensitivity), and ongoing mutual maintenance factors (e.g., attentional biases, anxiety, and depression).

Symptom overlap of PTSD and chronic pain as emphasized by high comorbidity rates and explanatory models has been assessed using various questionnaires measuring pain-related anxiety constructs. These include the Pain Catastrophizing Scale (PCS; Sullivan et al., 1995), developed to assess the degree to which individuals ruminate about, magnify, and feel helpless to manage their pain. The Pain Anxiety Symptoms Scale (PASS-20; McCracken & Dhingra, 2002) was developed to assess the degree an individual fears pain and pain-related sensations across

four domains: cognitive anxiety, pain-related fear, escape and avoidance, and physiological anxiety. The Fear of Pain Questionnaire (FPQ-III; McNeil & Rainwater, 1998) assesses the degree to which individuals are fearful of specific painful experiences, including severe pain (e.g., "breaking your neck"), minor pain (e.g., "biting your tongue while eating"), and medical pain (e.g., "receiving an injection in your arm"). While the Anxiety Sensitivity Index (ASI-3; S. Taylor et al., 2007) measures anxiety sensitivity, it does not specifically assess pain; however, as described above, anxiety sensitivity has been implicated as an important vulnerability factor for developing both chronic pain and PTSD. Higher levels of anxiety sensitivity are evident among various anxiety disorders, most notably PTSD and panic disorder (Taylor, Koch, & McNally, 1992). Moreover, anxiety sensitivity is associated with pain-related thoughts and behaviours. Individuals with high anxiety sensitivity are more likely to engage in pain-related avoidance as well as experience increased fearful interpretations of pain in those with recurrent headaches (Asmundson, Norton, & Veloso, 1999), musculoskeletal pain (Asmundson & Taylor, 1996), and chronic back pain (Asmundson & Norton, 1995). Taken together, this link with pain suggests that those high in anxiety sensitivity have a tendency to respond to physical symptoms like pain with hypervigilance, which reflects the overlap between anxiety and bodily sensations.

Although the PCS, PASS-20, FPQ-III, and ASI-3 are designed to assess pain-related anxiety, they were not designed to address the intersection between pain and traumatic symptoms. In many important ways, trauma-related disorders differ from anxiety disorders. As described by Resick and Miller (2009), this disparity is apparent because, unlike anxiety disorders (1) PTSD requires a causal link between an adverse event and the development of symptoms, (2) the development and maintenance of PTSD is related to emotions other than only fear and anxiety, (3) physiological reactivity of those with PTSD is associated with emotions

other than only fear and anxiety, and (4) the underlying latent factor class of trauma-related disorders suggests that they are discrete from other anxiety disorders. This dissimilarity is reflected in the newest edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013). While PTSD was previously included in the section describing anxiety disorders (American Psychiatric Association, 2000), it is now included in a new chapter describing trauma- and stressor-related disorders. The current definition of PTSD in the DSM-5 is that in conjunction with exposure to a stressor, the following symptom clusters must be present for diagnosis: intrusion symptoms (e.g., recurrent memories, nightmares), persistent avoidance (e.g., avoiding thoughts, feelings, or external reminders), negative alterations in cognitions and mood (e.g., persistent self-blame), and alterations in arousal and reactivity (e.g., sleep disturbance, hypervigilance). Due to the significant differences between trauma- and anxiety-related disorders, it is possible that the previously described pain-anxiety scales do not adequately capture the overlap between the breadth and nature of symptom clusters of PTSD and pain.

To address the gap in available assessment measures for pain and trauma, Katz et al. (2017) developed a 12-item measure, based on earlier work by Kleiman et al. (2011), that they called the Sensitivity to Pain Traumatization Scale (SPTS-12). Sensitivity to pain traumatization describes “the propensity to develop anxiety-related somatic, cognitive, emotional and behavioural responses to pain that resemble features of a traumatic stress reaction” (Kleiman et al., 2011, p. 169). In addition to directly including trauma-related symptoms (i.e., intrusion, avoidance, hyperarousal, emotional numbing), they aimed to capture the comorbid features of chronic pain and PTSD proposed in the shared vulnerability and mutual maintenance models (i.e., fear of pain and sensitivity to pain). As development of the SPTS-12 started prior to the

release of the newest edition of the DSM (American Psychiatric Association, 2013), the items included in the scale were based on the diagnostic criteria of PTSD as described in the DSM-IV-TR (American Psychiatric Association, 2000). Following exposure to a traumatic event, the DSM-IV-TR requires the presence of the following: (1) one or more symptoms of persistent reexperiencing the traumatic event, (2) three or more symptoms of persistent avoidance of stimuli related to the traumatic event, and (3) two or more symptoms of increased arousal following the traumatic event. In response to the criticism that the three-factor model of PTSD insufficiently captures symptom clusters of PTSD (Elhai, Grubaugh, Kashdan, & Frueh, 2008; Elhai & Palmieri, 2011; Simms et al., 2002; Yufik & Simms, 2010), the SPTS-12 included two items that assess emotional numbing, consistent with the "emotional numbing model" proposed by King et al. (1998). King et al. proposed that the avoidance symptom cluster should be further divided into two factors (i.e., avoidance and emotional numbing), for a total of four factors: reexperiencing, hyperarousal, effortful avoidance, and emotional numbing. The DSM-5 made several changes to the diagnostic criteria of PTSD, with one of the major modifications including the separation of the "persistent avoidance" symptom cluster into two: "avoidance" and "negative alternations in cognitions and mood", where the latter captures symptoms of emotional numbing. For these reasons, although the SPTS-12 was developed based on the criteria of PTSD as described in the DSM-IV-TR, the items correspond to the key symptoms of PTSD as described in the DSM-5.

The psychometric properties of the SPTS-12 were evaluated by Katz et al. (2017) in a series of three studies. Study 1 described the initial development and validation of the scale. The authors generated a list of 79 potential questionnaire items from existing scales (PCS; Sullivan et al., 1995, PASS-20; McCracken & Dhingra, 2002, ASI-3; Taylor et al., 2007, FPQ-III; McNeil

& Rainwater, 1998, PTSD Checklist – Civilian Version; McNeil & Rainwater, 1998, Impact of Events Scale; Horowitz, Wilner, & Alvarez, 1979) and created items based on a literature review to ensure breath of item content was included. Item responses were analyzed for 105 undergraduate student participants. Using item response theory, the preliminary 12-item STPS was developed. Study 2 and Study 3 examined the psychometric properties of the STPS-12 with a sample of 823 undergraduate student participants and a sample of 345 participants who had undergone coronary artery bypass graft surgery. Both studies found that the SPTS-12 discriminated between individuals reporting varying levels of sensitivity to pain traumatization, has very good internal reliability, good convergent validity, and moderate divergent validity. Factor analyses demonstrated that a single factor model had the strongest fit to the data.

As a new scale, the SPTS-12 requires further validation to help establish the consistency and strength of its psychometric properties in other populations. The current study aims to do this in a clinical sample of individuals receiving care from the Transitional Pain Service (TPS) at Toronto General Hospital. The TPS was established in 2014 to help prevent the progression of acute postsurgical pain to chronic pain by providing coordinated multidisciplinary care to proactively target biopsychosocial contributors of pain (Katz, Weinrib, et al., 2015). Given that pain after surgery is common, with an estimated 10 to 50% of patients developing chronic post-surgical pain (Katz & Seltzer, 2009; Kehlet et al., 2006), and that post-traumatic stress symptoms, most notably emotional numbing, have been shown to predict chronic post-surgical pain disability (Katz, Asmundson, McRae, & Halket, 2009), this sample provides a unique opportunity to evaluate the intersection of symptoms measured with the SPTS-12. As initial scale development is complete and initial reports of reliability and validity established, scale evaluation is now appropriate (Boateng, Neilands, Frongillo, Melgar-Quiñonez, & Young, 2018).

This evaluation should include confirmatory factor analysis (CFA) to test the a priori prediction of a one-factor model for the SPTS-12 items.

The aims of the present study are to continue to evaluate the psychometric properties of the SPTS-12. Of particular interest is the factor structure, convergent validity, divergent validity, and reliability in a sample of postsurgical patients admitted to the TPS. The following is predicted: (1) the inter-item associations for the items of the SPTS-12 will suggest that retaining a one-factor model will be ideal, (2) the SPTS-12 will demonstrate good internal consistency, (3) the SPTS-12 will be strongly correlated with measures of the theoretically similar construct of trauma, and (4) the SPTS-12 will be less strongly correlated with measures of the theoretically similar, although distinct construct, of depression.

Methods

Procedure. The current study was approved by the University Health Network (UHN) Research Ethics Board (certificate approval #s: 14-7705-AE and 16-5109) and the York University Research Ethics Board (Human Participants Review Subcommittee, certificate approval #: 14-7705-AE). See Appendix C for relevant approval paperwork.

Participants were considered eligible for the study if they were older than 18 years of age and met one or more of the following TPS inclusion criteria: (1) considered to be a "pain alert" patient upon hospital admission (prior to surgery), whereby they have a history of chronic pain, drug abuse or misuse, present or past opioid use, or current use of methadone or buprenorphine, (2) spent more than three days observed by the Acute Pain Service postoperatively due to significant persistent postoperative pain, (3) postoperative pain management included more than 90 mg morphine equivalents (MME) per day of an opioid class drug, (4) postoperative interventional pain procedures are required, (5) subsequent pain consultations are required when

no longer observed by Acute Pain Service, and (6) patients referred by attending surgeons at UHN.

Eligible participants were approached either preoperatively or postoperatively by the TPS study coordinator who explained the study protocol. Participants provided written informed consent prior to involvement in the study. Participants were asked to complete a paper questionnaire package at various timepoints: preoperatively, after discharge from the hospital, at their first outpatient visit in the TPS, and each subsequent visit to the TPS. Data for the present study were derived from the first outpatient visit to the TPS.

Measures.

Demographic and medical history. Demographic information including age and sex was collected. Preoperative variables were available for pre-existing medical conditions and diagnoses, prescription medication use, pain complaints, pain treatments, and surgery type. Daily mg morphine equivalent dosage was available at each visit to the TPS.

Pain experience and interference. The Brief Pain Inventory (BPI; Cleeland & Ryan, 1994) assesses pain severity and interference. The short form of the scale consists of nine items, with the first question asking if the participant is currently experiencing pain as well as the location of pain. The next four items use an 11-point Likert-type response scale asking participants to rate their current level of pain as well as the average, lowest, and worst level of pain they experienced in the previous 24 hours. For these questions, item anchors were 0 = *no pain* and 10 = *pain as bad as you can imagine*. Subsequent questions ask what medications or treatments participants are currently using for pain and the percentage of relief the medications or treatments are providing, with item anchors 0% = *no relief* and 100% = *complete relief*. The final question measures pain interference across seven domains (general activity, mood, walking

ability, normal work, relations with other people, sleep, and enjoyment of life), with item anchors 0 = *does not interfere* and 10 = *completely interferes*. Pain severity items can be reported in two ways that are both supported by the Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials (IMMPACT; Dworkin et al., 2005; Dworkin et al., 2008): (1) as one mean severity score across all four items or (2) the score obtained on a single item, such as the items "average" or "worst" pain. In both cases, higher scores indicating worse pain severity. Pain interference is reported as the mean across the seven interference domains (Cleeland, 2009).

The BPI has good to excellent internal consistency (α = .80 to .87 for pain severity items and α = .89 to .92 for pain interference items; Cleeland et al., 1994) and good test-retest reliability with consecutive daily administration over the course of a week (*r* = .83 to .88 for pain severity items and *r* = .83 to .93 for pain interference items; Mendoza, Mayne, Rublee, & Cleeland, 2006). In the present sample, the BPI showed very good internal consistency for pain severity (α = .87) and pain interference (α = .89) scores.

Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983)**.** The HADS consists of two subscales, one assessing symptoms of anxiety (HADS-A) and the other measuring symptoms of depression (HADS-D). The HADS was developed for use in outpatient primary or secondary care settings (Zigmond & Snaith, 1983). Each subscale consists of seven items, each using a four-point Likert-type response scale with different anchors depending on the item; for example, the first anxiety item asks participants to rate the degree to which they "feel tense or 'wound up'" with anchors 0 = *not at all*, 1 = *from time to time*, *occasionally*, 2 = *a lot of the time*, and 3 = *most of the time*. The first depression item asks if participants they "still enjoy the things I used to enjoy", with anchors 0 = *definitely as much*, 1 = *not quite so much*, 2 = *only a little*, and 3 = *hardly at all*. The total score range for each subscale is 0 to 21, with higher scores

reflecting greater depressive and anxiety symptoms, respectively. Scores between 0 and 7 are considered to be in the normal range, scores between 8 and 10 are considered to be in the borderline range, and scores between 11 and 21 are considered to be in the abnormal range (Zigmond & Snaith, 1983). According to a review of 14 studies, the authors concluded that internal consistency for both the HADS-A (α =.68 to .93, *M* = .83) and the HADS-D (α = .67 to .90, *M* = .82) ranges from poor to excellent (Bjelland, Dahl, Haug, & Neckelmann, 2002). The HADS has good test-retest reliability over a 0 to 2 week period (HADS-A; *r* = .84, HADS-D; *r* = .85; Herrmann, 1997). Furthermore, the HADS has demonstrated good construct validity (Johnston, Pollard, & Hennessey, 2000). In the present study, the HADS showed fair internal consistency for anxiety (α = .79) and good internal consistency for depression (α = .80) scores.

Injustice Experience Questionnaire (IEQ; Sullivan et al., 2008). The IEQ assesses feelings of perceived injustice associated with injury. It consists of 12 items using a five-point Likert-type response scale, with item anchors 0 = *never* and 4 = *all the time*. The total score range is 0 to 48 with higher scores indicating greater levels of perceived injustice. The IEQ has good internal consistency (α = .92) and strong construct validity, with evidence that test-retest reliability scores remain stable across a four-week period (*r* = .90; Sullivan et al., 2008). The IEQ showed excellent internal consistency in the present study (α = .91).

Pain Catastrophizing Scale (PCS; Sullivan et al., 1995). The PCS assesses ruminative thinking, perception of threat, and feelings of helplessness in relation to painful experiences and sensations. It consists of 13 items using a five-point Likert-type response scale, with item anchors 0 = *not at all* and 4 = *all the time*. The total score range is 0 to 52 with higher scores indicating greater levels of pain catastrophizing. The PCS has good internal consistency (α = .87; Sullivan et al., 1995) and test-retest reliability over a six-week period (*r* = .75; Sullivan et al.,

1995). The PCS has demonstrated strong construct validity (Osman et al., 1997; Sullivan et al., 1995). The PCS showed excellent internal consistency in the present study (α = .94).

Abbreviated PTSD Checklist – Civilian Version (Abbreviated PCL-C; Lang & Stein, 2005; Lang et al., 2012). The Abbreviated PCL-C is a shorter version of the PCL-C that was developed to assess the traumatic response that individuals may have in response to a stressful event (Weathers et al., 1994). The abbreviated version consists of six items drawn from the 17-item scale (items 1, 4, 7, 10, 14, and 15). Items are scored using a five-point Likert-type response scale, with item anchors 1 = *not at all* to 5 = *extremely*. The total score range is 6 to 30, with higher scores reflecting greater traumatic responses. The total PCL-C has excellent internal consistency (α = .92 to .94), the test-retest reliability over a two-week period is good (*r* = .66), and the scale demonstrates good convergent and discriminant validity (Conybeare et al., 2012). The abbreviated PCL-C is highly correlated with the PCL-C total score (*r* = .97) and has demonstrated high sensitivity (.80) and specificity (.76) for detecting PTSD (Lang & Stein, 2005). The proposed total score cut-off to screen for PTSD for the abbreviated PCL-C is 14 (Lang & Stein, 2005; Lang et al., 2012). The abbreviated PCL-C had excellent internal consistency in the present study (α = .89).

Sensitivity to Pain Traumatization Scale (SPTS-12; Katz et al., 2017). The SPTS-12 assesses the propensity to develop a traumatic response to pain. It consists of 12 items using a five-point Likert-type response scale, with item anchors 0 = *not at all true* and 4 = *entirely true*. The total score range is 0 to 48 with higher scores indicating a more traumatic response to pain. When the SPTS-12 was first developed (Katz et al., 2017), the wording of item 1 was “pain keeps me awake at night”. Because data collection for the present study started in 2014, the original wording was used. However, the final version of the scale described by Katz et al.

(2017) adapted the wording of this item to "when I am in pain, it keeps me awake at night" to make it applicable to individuals not experiencing pain at the time the scale is being completed. The SPTS-12 has good internal consistency both with the original wording of item 1 ("pain keeps me awake at night", $\alpha = .88$) and the revised wording ("when I am in pain, it keeps me awake at night", $\alpha = .89$). Both versions have demonstrated that the associations among the SPTS-12 items are well-explained by a one-factor structure and that it has very good reliability and validity in a clinical and community sample (Katz et al., 2017). The internal consistency of the SPTS-12 in the present sample is reported in the results section below.

Statistical analysis. See Appendix D for the syntax used in the final analysis.

***Confirmatory factor analysis*.** Analyses were conducted in R (Version 3.5.1) using the packages "psych" and "lavaan" (Revelle, 2018; Rosseel, 2012). Confirmatory factor analysis (CFA) was used to specify a one-factor model for the SPTS-12 items; this model was fitted to polychoric correlations among the items using unweighted least squares estimation with robust standard error and mean- and variance-adjusted test statistics (ULSMV). Polychoric correlations were used because the SPTS-12 items have a categorical, five-point Likert-type scale (Flora et al., 2012). Confirmatory factor analysis was favored over exploratory factor analysis because previous investigations of the factor structure of the SPTS-12 determined that the one-factor model provided the best explanation of the data in both a clinical and community sample, both for individuals reporting pain and those not reporting pain (Katz et al., 2017).

Model fit was evaluated using the root mean square error of approximation (RMSEA), comparative fit index (CFI), Tucker-Lewis index (TLI), and the standardized root mean square residual (SRMR). The general guidelines suggested by Browne and Cudeck (1993) and MacCallum, Browne, and Sugawara (1996) were used in the interpretation of the RMSEA,

where a score less than .05 was considered close fit, score between .05 and .10 considered an acceptable to mediocre fit, and anything larger considered a poor fit. CFI and TLI scores range between approximately 0 and 1, with higher scores (i.e., higher than .90) considered to reflect a better fit (Hu & Bentler, 1999). For the SRMR, values below .08 are considered to indicate adequate fit (Hu & Bentler, 1999). Although Hu and Bentler's guidelines are widely used, Xia and Yang (2019) cautioned that they may provide overly favourable fit indices when models are fitted to polycohoric correlations.

Reliability. Internal consistency reliability was calculated using McDonald's (McDonald, 1970, 1999) omega using the package "semTools" (Jorgensen, Pornprasertmanit, Schoemann, & Rosseel, 2018). Omega was favoured over coefficient alpha because it provides a more accurate estimate of reliability if multidimensionality is present, factor loadings differ across items, or measurement errors are correlated across items (Raykov & Marcoulides, 2016).

Validity. Spearman's correlations were used for comparisons because they do not require a linear relationship between variables. Convergent validity was evaluated with the correlation between the SPTS-12 and a scale measuring a theoretically similar construct, the PCL-C. Divergent validity was evaluated with the correlation between the SPTS-12 and the HADS-D, a measure of a related but theoretically distinct construct. The magnitude of the difference between correlation coefficients for SPTS-12 and PCL-C and for SPTS-12 and HADS-D was evaluated (Lee & Preacher, September, 2013). A significantly larger correlation for the former than the latter would suggest good convergent and divergent validity, respectively.

Results

Missing data. A total of 219 participants were recruited. Thirty-nine participants failed to respond to all questions of the STPS-12. Item non-response is common in clinical settings, and

has many possible reasons, including fatigue of filling out the questionnaire, uncertainty in how to respond to the item, or poor item validity (Little et al., 2012; McKnight, McKnight, Sidani, & Figueredo, 2007; O'Neill & Temple, 2012).

The pattern of missing data for all items of the SPTS-12 was evaluated (see Table 3.1). Item 5 "Pain seems to bother me more than it does other people" had the highest number of missing entries, whereas item 1 "pain keeps me awake at night" had the lowest. For scales other than the SPTS-12, total scores were prorated by calculating the mean score of the available items and then multiplying this mean by the total number of items in the scale to create an adjusted total score (Enders, 2010). Prorating was conducted for a given scale only for participants who completed 80% or more of the items on that scale.

Participant characteristics. Of the 180 participants included in the final analysis, the mean age was 49.7 (*SD* = 14.41) years with 53.89% of the sample identifying as male. Information regarding the ethnic background of participants was not available. Preoperative diagnoses included cancer (56.8%), chronic pain (57.4%), gastroesophageal reflux disease (48.4%), hypertension (37.7%), arthritis (35.0%), diabetes mellitus (18.0%), thyroid disease (16.8%), chronic obstructive pulmonary disease (13.7%), asthma (13.0%), sleep apnea (12.4%), anemia (12.9%), peptic ulcer disease (9.2%), deep vein thrombosis (9.2%), and peripheral vascular disease (8.2%).

A total of 167 participants underwent surgery. Surgery type included thoracic (26.7%), transplant (15.2%), general (13.9%), cardiac (9.7%), ear, nose, and throat (9.1%), vascular (6.1%), obstetrical/gynecological (5.5%), plastic (4.8%), urologic (4.2%), and neurological (3.0%). Surgery type and date was unavailable for five patients, as they were post-surgery, outpatient referrals from another hospital. The mean hospital stay was 12.0 days (*n* = 62

participants with data available on length of hospital stay). Thirteen participants were non-surgical referrals presenting with chronic pain.

For participants who underwent surgery, the first visit to the TPS was on average 80.10 days (*SD* = 129.49) after surgery, ranging from 0 to 917 days. A total of 43.2% attended their first visit within the first month since surgery, 37.0% between 1-3 months since surgery, 16.7% between 3-12 months since surgery, and 3.1% over one year after surgery. The large range is due to the multiple referral pathways to the TPS (Azam et al., 2017), which include recruitment preoperatively, in-hospital, and after hospital discharge (see Methods for a full list of inclusion criteria and procedures). At their first visit to the TPS as assessed with the BPI, 87.8% of participants responded "yes" to currently experiencing "pain other than everyday kinds of pain", while 7.2% responded "no" and 5% did not respond to this question. For those endorsing experiencing pain currently, the mean reported severity was moderate (*M* = 5.14, *SD* = 2.26). Over the previous 24 hours, participants rated their average level of pain as *M* = 5.65 (*SD* = 2.00), least pain as *M* = 3.87 (*SD* = 2.22), and worst pain as *M* = 7.15 (*SD* = 1.85), reporting a mean of 53.14% (*SD* = 25.09%) of pain relief received from pain treatments and medications. Participants reported a moderate to high degree of pain interference over the previous 24 hours (*M* = 6.37, *SD* = 2.11).

Descriptive statistics. Descriptive statistics for the 12 items of the STPS are reported in Table 3.2 and the polychoric correlations are in Table 3.3. Descriptive statistics for the questionnaire data are in Table 3.4.

Factor analysis. The one-factor model showed a good fit to the data with CFI = .97, TLI = .96, and SRMR = .06 according to guideline criteria (Hu & Bentler, 1999). The one-factor

model had RMSEA = .08 (90% CI: .06 to .10), which indicates fair fit to the data (Browne & Cudeck, 1993; MacCallum et al., 1996).

The completely standardized factor loadings of the STPS-12 items ranged from 0.41 to 0.93, showing that each item had a substantial association with the sensitivity to pain traumatization factor. However, the uniqueness estimates varied greatly (13% to 83%), showing that some items still have a large portion of variance that is not explained by the latent variable. The completely standardized factor loadings and uniqueness estimates are displayed in Table 3.5.

Internal consistency. Internal consistency reliability calculated using McDonald's omega was .90 for the STPS-12 total score using the one-factor model.

Convergent and divergent validity. Table 3.6 displays the Spearman correlations among all measures. The correlation between the SPTS-12 total score and the PCL-C total score was strong, $r(163) = .60$, $p < .001$, providing evidence for convergent validity. The correlation between the SPTS-12 total score and the HADS-D total scores was moderate, $r(176) = .53$, $p < .001$. The correlation between the SPTS-12 and the PCL-C total scores was not significantly greater than the correlation between the SPTS-12 and the HADS-D total scores, $z = -1.12$, $p = .26$.

Discussion

The aim of the present study was to evaluate the factor structure, reliability, and validity of the SPTS-12 in a clinical sample of individuals receiving care from the TPS at Toronto General Hospital. Confirmatory factor analysis supported the one-factor model of the SPTS-12, with evidence of excellent internal consistency reliability. While the SPTS-12 showed good convergent validity with the theoretically related construct of trauma, divergent validity was not supported: the SPTS-12 was not more strongly correlated with the PCL-C than it was with the

HADS-D, which measures the theoretically related although distinct construct of depression. Taken together, results of the present study add to the literature establishing the psychometric properties of the SPTS-12.

The confirmatory factor analysis supported the findings of Katz et al. (2017) showing that the SPTS-12 is well-explained by a one-factor model. Katz et al. (2017) used an exploratory factor analysis in three samples across 1,168 participants: a community sample reporting ongoing pain; a community sample reporting no ongoing pain; and a clinical, postsurgical sample after cardiac bypass graft surgery. The results showed that the one-factor model consistently provided the best explanation for the data. Furthermore, both the present findings and those of Katz et al. (2017) support the findings by Kleiman et al. (2011). The latter examined the factor structure of three pain-related anxiety questionnaires, the PASS-20, the PCS, and the ASI, and found that one higher-order factor accounted for 63.8% of the common variance.

The considerable comorbidity between symptoms of pain, trauma, and anxiety both for individuals diagnosed with PTSD (Sareen et al., 2007) and chronic pain (Siqveland et al., 2017) provide clinical support of one higher-order, underlying construct. This stands in contrast to the ongoing debate regarding the factor structure of PTSD symptoms, with researchers suggesting that the best structural fit may be a two-factor model (Hunt, Chesney, Jorgensen, Schumann, & deRoon-Cassini, 2018), four-factor model (King et al., 1998), six-factor model (Liu et al., 2014; Tsai, Harpaz-Rotem, et al., 2015), or even a seven-factor model (Seligowski & Orcutt, 2016). Each proposed model evaluates whether distinct factor clusters should exist for various traumatic symptoms, including reexperiencing, avoidance, negative alterations in mood/cognitions, hyperarousal, intrusion, emotional numbing, dysphoria, and negative affect. In contrast, although the SPTS-12 was developed to assess six symptom domains (pain and emotional numbing, pain

and hyperarousal, pain avoidance, pain experiencing/intrusive thoughts, sensitivity to pain, and fear of pain), by describing the trauma/anxiety-related symptoms as experienced in response to pain, this appears to sufficiently unite the symptoms to be best explained by a single higher-order latent variable of sensitivity to pain traumatization in the current study.

Although the confirmatory factor analysis provided support for the one-factor model in the present sample, nonetheless, some items on the SPTS-12 were associated with a large proportion of unexplained variance. Most notably, this included item 1 "pain keeps me awake at night" (for which 83% of the variance was not accounted for), item 3 "I try to avoid activities that cause pain" (for which 70% of the variance was not accounted for), and item 9 "as soon as pain comes on, I take medications to reduce it" (for which 64% of the variance was not accounted for). These items are also associated with the highest item means (see Table 3.2). It is possible that Item 1 has the most unexplained source of variance due to the strong relationship between pain and sleep. It is estimated that 67-88% of individuals with chronic pain experience sleep complaints (Morin, LeBlanc, Daley, Gregoire, & Merette, 2006; Smith & Haythornthwaite, 2004), and furthermore that 50% of individuals with insomnia experience chronic pain (Taylor et al., 2007). While rates of anxiety among those with chronic pain are estimated to be high (e.g., 17% for any anxiety disorder for individuals reported chronic low back pain or chronic musculoskeletal pain; Asmundson, Jacobson, Allerdings, & Norton, 1996; Polatin, Kinney, Gatchel, Lillo, & Mayer, 1993), the rate of co-occurrence between pain and sleep is consistently greater. Although a better understanding of the link between pain and sleep after surgery is still needed (Chouchou, Khoury, Chauny, Denis, & Lavigne, 2014), in the present study we can speculate that some of the unaccounted variance for item 1 might be related to sleep difficulties that are present in the absence of other pain anxiety-related symptoms.

The unaccounted variance for items 3 and 9 is likely also related to the increased incidence of avoiding activities that may cause pain or the use of pain medication after surgery when other anxiety- and trauma-related symptoms are not present. Avoidance after surgery can be adaptive: that is, experiencing acute pain can discourage movements that may contribute to further tissue damage and encourage the individual to elicit care (Costigan et al., 2009; Craig, 2009; Loeser & Melzack, 1999). As some participants attended their first visit to the TPS shortly after surgery (of those receiving surgery, 43.2% first visited the TPS within 30 days of surgery), it is expected that some restriction of activities would be recommended to facilitate healing. Therefore, participants without accompanying symptomology related to anxiety may have still endorsed these items. However, avoidance is also considered a central mechanism in the maintenance and exacerbation of chronic pain (Vlaeyen & Linton, 2000), even postoperatively (Archer, Seebach, Mathis, Riley III, & Wegener, 2014; Archer et al., 2011): if an individual experiences pain and it is interpreted as threatening, a fear of pain may develop, increasing hypervigilance to sensations of pain. This may in turn may increase avoidance of activities that cause pain, leading to deconditioning and less engagement with daily activities, contributing to low mood and disability. In consequence, pain may increase, again adding to the perception that pain is threatening (Vlaeyen & Linton, 2000). Therefore, although the unaccounted-for variance for items 1, 3, and 9 may be inflated due to the strong link between pain and sleep as well as the adaptive avoidance of activities and use of pain medication immediately after surgery, the remaining overlap in the variance accounted for by the latent construct demonstrates that the SPTS-12 may help detect the nonadaptive components of avoidance.

The present study found that the SPTS-12 has excellent internal consistency reliability, consistent with the findings of Katz et al. (2017) for both community and clinical samples.

Furthermore, the SPTS-12 demonstrated evidence of convergent validity in the current sample as determined through a strong correlation (r = .60) with the theoretically related construct of trauma using the PCL-C. However, the SPTS-12 failed to demonstrate divergent validity, as the relative strength of the relationship of the SPTS-12 and PCL-C was not statistically stronger than that of the SPTS-12 and a measure of depression, a theoretically related although distinct construct of sensitivity to pain traumatization. This finding might have less to do with a true lack of divergent validity and more to do with our choice of the measure, HADS-D, in a chronic postsurgical pain sample, to assess divergent validity. Rates of depression become increasingly comorbid with anxiety-related constructs when individuals endorse experiencing pain. This is consistent with the findings of Katz et al. (2017), where in a community sample of 823 participants, they only established divergent validity of the SPTS-12 for a sub-group not endorsing pain. That is, for the 268 participants reporting experiencing pain on an ongoing basis, the magnitude of the correlation between the SPTS-12 and a measure of trauma was not significantly greater than the magnitude of the correlation between the SPTS-12 and a measure of depression (Katz et al., 2017). Taken together, this supports the general finding of a positive association between the presence of pain and psychiatric conditions: in a representative sample of 5877 Americans, McWilliams et al. (2003) found that the odds ratio between chronic pain and a 12-month diagnosis of any mood disorder was 2.78 (95% confidence interval [CI]: 2.06–3.75) and for any anxiety disorder was 2.86 (95% CI: 2.06–3.97). This link becomes even more striking in veterans with pain: in a sample, of 250 veterans, Outcalt et al. (2015) found that 17% of the sample met criteria for PTSD and 24% for depression. Furthermore, for those meeting criteria for PTSD, 62.8% also met criteria for major depressive disorder (Outcalt et al., 2015). Due to the increasing levels of comorbidity of psychiatric conditions for individuals experiencing

chronic pain, this helps explain why depression may not be an adequately sensitive construct to evaluate divergent validity for the SPTS-12 in a population with pain. Because the present study is based on a sample of patients receiving treatment in a clinical setting, we were limited to the available measures administered to the patients and therefore a more reasonable choice to assess divergent validity was not available.

The present study adds to the psychometric validation of the SPTS-12 using a cross-section design. Future research would benefit from longitudinal methods that would permit the examination of predictive validity of the SPTS-12, especially regarding important outcome measures such as average pain and pain disability (Dworkin et al., 2008). For surgical populations, it would be of particular interest to examine if presurgical scores impact outcomes after surgery, as the SPTS-12 could serve as a screening tool to identify individuals who are at particularly high risk of chronic postsurgical pain. The present study examined a heterogenous population enrolled in the TPS at a major hospital, and included participants that underwent various surgeries (e.g., thoracic, transplant, and cardiac) or that suffered from non-surgery related pain. While validating the SPTS-12 in a clinically diverse setting addresses concerns regarding feasibility and functionality in a real-world setting (Blonde, Khunti, Harris, Meizinger, & Skolnik, 2018), it is possible that the psychometric properties of the SPTS-12 may vary based on individual or group characteristics such as surgery type, gender, or pain severity. Finally, it would be important to consider the use of another measure than one assessing depression to evaluate divergent validity of the SPTS-12, as the high comorbidity rates of pain-related anxiety and low mood hinder its interpretability.

The present study has several limitations. First, only self-report measures were used to assess symptoms of anxiety, trauma, and mood. While the measures used in the present study

have high sensitivity and specificity of detecting probable diagnosis of anxiety, depression, and PTSD (Bjelland et al., 2002; Lang & Stein, 2005), questionnaires are not an adequate substitute for a clinician-administered semi-structured interview such as the Diagnostic Interview for Anxiety, Mood, and Obsessive Compulsive and Related Neuropsychiatric Disorders (DIAMOND; Tolin et al., 2013). Diagnostic interviews can provide a more in-depth and accurate representation of the clinical symptoms and diagnoses, although at the expense of a lengthy, in-person administration time that is labour-intensive for both participants and researchers (e.g., the DIAMOND takes over an hour to administer; Tolin et al., 2018). However, in developing and validating the SPTS-12, it would be beneficial to determine the concurrent validity by evaluating its relationship with relevant psychiatric diagnoses (e.g., PTSD, major depressive disorder, generalized anxiety disorder, etc.). Second, 17.8% of the sample was excluded due to not completing all of the questions on the SPTS-12. Although no significant differences were detected regarding the demographic information between those that did and did not fill out all of the items, it is possible that differences might exist in the questionnaire data. Given the initial validation of the SPTS-12, future studies should consider using imputation when more than 80% of items have been completed instead of requiring all items to be completed (Enders, 2010).

Conclusion

The present study examined the psychometric properties of the SPTS-12, a scale developed to assess the cognitive, emotional, behavioural, and somatic responses to pain that are similar to a traumatic stress response. The factor structure, reliability, and validity were evaluated in a clinical sample of individuals enrolled in the TPS at Toronto General Hospital. The CFA demonstrated a good fit to the data and the SPTS-12 showed excellent reliability and good convergent validity with the PCL-C. As assessed by the magnitude of the correlation

between the SPTS-12 and a similar construct (PCL-C) compared to the magnitude of the correlation between the SPTS-12 and a measure of depressive symptoms (HADS-D), the SPTS-12 scores had questionable discriminant validity.

Table 3.1. Missing data entries according to each item of the Sensitivity to Pain Traumatization Scale (SPTS-12).

	SPTS-12 item	Valid entry	Missing entry
1.	Pain keeps me awake at night.*	219	0
2.	When I am in pain, everything I see or do reminds me of the pain.	216	3
3.	I try to avoid activities that cause pain.	216	3
4.	When I feel pain, I'm scared that it's the beginning of a terrible problem.	215	4
5.	Pain seems to bother me more than it does other people.	206	13
6.	When I feel pain, I think about it even when I don't mean to.	212	7
7.	I can't stand pain.	214	5
8.	When I'm in pain, I feel distant from people even when I'm talking to them.	215	4
9.	As soon as the pain comes on, I take medications to reduce it.	211	8
10.	Pain sensations terrify me.	210	9
11.	When I'm in pain, things don't feel real.	213	6
12.	I feel sick to my stomach when I am in pain.	217	2

Note. *This item was modified to “when I am in pain, it keeps me awake at night” in the final published version of the SPTS-12 (Katz et al., 2017).

Table 3.2. Descriptive statistics for the 12 items of the Sensitivity to Pain Traumatization Scale (SPTS-12).

	SPTS-12 item	*M*	*SD*	*Skewness*	*Kurtosis*
1.	Pain keeps me awake at night.*	2.48	1.27	-.44	-.90
2.	When I am in pain, everything I see or do reminds me of the pain.	1.59	1.31	.37	-1.01
3.	I try to avoid activities that cause pain.	2.63	1.22	-.69	-.57
4.	When I feel pain, I'm scared that it's the beginning of a terrible problem.	1.23	1.35	.74	-.73
5.	Pain seems to bother me more than it does other people.	1.16	1.41	.78	-.88
6.	When I feel pain, I think about it even when I don't mean to.	1.52	1.40	.35	-1.29
7.	I can't stand pain.	1.92	1.52	.12	-1.46
8.	When I'm in pain, I feel distant from people even when I'm talking to them.	1.77	1.30	.16	-1.12
9.	As soon as the pain comes on, I take medications to reduce it.	1.93	1.43	.07	-1.32
10.	Pain sensations terrify me.	1.16	1.31	.84	-.56
11.	When I'm in pain, things don't feel real.	1.10	1.37	.92	-.56
12.	I feel sick to my stomach when I am in pain.	1.17	1.32	.84	-.54

Note. $N = 180$. SPTS-12 item scores range from 0 to 4. *This item was modified to "when I am in pain, it keeps me awake at night" in the final published version of the SPTS-12 (Katz et al., 2017).

Table 3.3. Polychoric correlation matrix of the 12 items of the Sensitivity to Pain Traumatization Scale (SPTS-12).

	1	2	3	4	5	6	7	8	9	10	11	12
1	-											
2	.36	-										
3	.37	.50	-									
4	.33	.58	.41	-								
5	.15	.47	.24	.55	-							
6	.25	.64	.42	.55	.62	-						
7	.29	.55	.37	.44	.61	.68	-					
8	.30	.51	.31	.57	.52	.54	.61	-				
9	.23	.38	.38	.31	.41	.53	.51	.46	-			
10	.27	.64	.43	.67	.67	.74	.66	.64	.61	-		
11	.27	.61	.33	.67	.52	.52	.55	.63	.36	.74	-	
12	.28	.45	.31	.42	.34	.45	.45	.49	.44	.59	.61	-

Note. $N = 180$.

Table 3.4. Descriptive statistics for questionnaire data.

	M	*SD*	*N*	*Min*	*Max*
BPI-I	6.37	2.11	180	0	10
HADS-A	9.19	4.21	176	0	18
HADS-D	9.19	4.34	176	0	20
IEQ	20.30	11.91	161	0	48
PCL-C	14.49	6.33	163	6	30
PCS	23.30	14.17	179	0	52
SPTS-12	19.66	11.21	180	0	48

Note. BPI-I - Brief Pain Inventory - Pain Interference subscale, HADS-A - Hospital Anxiety and Depression Scale - Anxiety subscale, HADS-D - Hospital Anxiety and Depression Scale - Depression subscale, IEQ - Injustice Experience Questionnaire, PCL-C - PTSD Checklist - Civilian Version, PCS - Pain Catastrophizing Scale, SPTS-12 – 12-item Sensitivity to Pain Traumatization Scale

Table 3.5. Completely standardized factor loadings and uniqueness estimates for the one-factor model.

	SPTS-12 item	Completely Standardized Factor Loading	Uniqueness Estimate
1.	Pain keeps me awake at night.*	0.41	0.83
2.	When I am in pain, everything I see or do reminds me of the pain.	0.75	0.44
3.	I try to avoid activities that cause pain.	0.55	0.70
4.	When I feel pain, I'm scared that it's the beginning of a terrible problem.	0.73	0.47
5.	Pain seems to bother me more than it does other people.	0.69	0.52
6.	When I feel pain, I think about it even when I don't mean to.	0.81	0.35
7.	I can't stand pain.	0.77	0.41
8.	When I'm in pain, I feel distant from people even when I'm talking to them.	0.76	0.43
9.	As soon as the pain comes on, I take medications to reduce it.	0.60	0.64
10.	Pain sensations terrify me.	0.93	0.13
11.	When I'm in pain, things don't feel real.	0.80	0.37
12.	I feel sick to my stomach when I am in pain.	0.63	0.61

Note. $N = 180$. *This item was modified to “when I am in pain, it keeps me awake at night” in the final published version of the SPTS-12 (Katz et al., 2017).

Table 3.6. Spearman correlation matrix.

	SPTS-12	BPI-I	HADS-A	HADS-D	IEQ	PCL-C
BPI-I	.52					
HADS-A	.59	.50				
HADS-D	.53	.59	.55			
IEQ	.63	.41	.55	.49		
PCL-C	.60	.52	.70	.52	.61	
PCS	.72	.61	.66	.50	.61	.53

Note. N ranges from 149 to 180. All correlations $p < .001$. BPI-I - Brief Pain Inventory - Pain Interference subscale, HADS-A - Hospital Anxiety and Depression Scale - Anxiety subscale, HADS-D - Hospital Anxiety and Depression Scale - Depression subscale, IEQ - Injustice Experience Questionnaire, PCL-C - PTSD Checklist - Civilian Version, PCS - Pain Catastrophizing Scale, SPTS-12 – 12-item Sensitivity to Pain Traumatization Scale.

Chapter 4: Study 3

Introduction

Chronic post-surgical pain is a common complication following surgery, with incidence rates ranging from 10% to 50% (Kehlet et al., 2006). The transition from acute to chronic pain following surgery is complex, with diverse biological and psychosocial risk factors that occur across the preoperative, intraoperative, and postoperative periods (Katz & Seltzer, 2009). In the last several decades, there has been an increased recognition of psychological factors in development of chronic postsurgical pain (Katz & Seltzer, 2009; Weinrib et al., 2017). Of particular interest is the presence of post-traumatic stress symptoms (PTSS) following surgery given that surgery can expose the patient to traumatic medical events or serious injury, one of the diagnostic requirements for Posttraumatic Stress Disorder (PTSD) in the newest edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-5; American Psychiatric Association, 2013).

Pain and trauma are frequently comorbid in clinical settings, with rates of PTSD in pain clinics estimated to be between 9.5 and 33% (Macfarlane et al., 1999; Meltzer-Brody et al., 2007). Following surgery, probable PTSD diagnosis as identified through self-report has been estimated to range between 15 and 20% after cardiac surgery (Stoll et al., 2000), breast cancer (O'Connor, Christensen, Jensen, Møller, & Zachariae, 2011), and total knee replacement surgery (Cremeans-Smith, Greene, & Delahanty, 2011). Furthermore, the impact of PTSS on health-related quality of life may become increasingly important as time elapses post-surgery: in a sample of 47 patients who received lateral thoracotomy, Katz et al. (2009) found that the PTSS symptom of emotional numbing accounted for 4% of the variance of concurrent pain disability scores six months following surgery, but that the proportion of variance due to emotional

numbing increased to 20% when assessed again 12 months after surgery. These results suggest that the relative impact of the PTSS of emotional numbing becomes stronger for quality of life indices such as pain disability over time since surgery.

The high degree of overlap between pain and trauma has been theorized to be related to mutually maintaining factors, shared vulnerability factors, and behavioural responses that are present in both disorders (Asmundson et al., 2002; Keane & Barlow, 2002; Liedl & Knaevelsrud, 2008; Rosenbloom et al., 2013; Sharp & Harvey, 2001; Turk, 2002). This strong theoretical relationship recently led Katz et al. (2017) to develop the Sensitivity to Pain Traumatization Scale (SPTS-12) to assess the propensity to develop a traumatic stress response to pain. The psychometric properties of the SPTS-12 have been examined in both a community (N = 823) and a postsurgical clinical sample (N = 345), and taken together demonstrate support for the reliability and validity of the SPTS-12 (Katz et al., 2017). Despite this preliminary support, the SPTS-12 has yet to be examined over time and its predictive validity has not been evaluated.

There is a growing trend in health research to better understand change over time while simultaneously accounting for patient heterogeneity by using latent class growth mixture models (Benyamini, Ein-Dor, Ginzburg, & Solomon, 2009; Chapman, Donaldson, Davis, & Bradshaw, 2011; Henly, Wyman, & Findorff, 2011; Nosyk et al., 2011). By identifying subgroups of patients with discrete baseline levels or rates of change, a more precise account of subgroup(s) at increased risk of chronic postsurgical pain may be possible. For example, Chapman et al. (2011) measured self-reported pain scores in a sample 502 elective surgery patients daily for six days after surgery. For the entire sample, the mean pain rating was 5.59 (SD = 2.20) with a mean slope of -0.31. The authors then subdivided the sample into three groups: patients with decreasing slopes (n = 314), with flat slopes (n = 127), and with increasing slopes (n = 61). They concluded

that identifying pain trajectories after surgery yielded more precise and accurate information regarding the rate and direction of pain resolution, but see Bauer (2007) for a critique of inferential conclusions made using growth mixture modeling. Because poorly managed pain predicts chronic postsurgical pain (Perkins & Kehlet, 2000), providing more specialized pain management to patients whose pain is not improving could help improve pain-related outcomes. Given that the relative impact of psychological variables such as trauma may increase over time since surgery, identifying latent class trajectories of the SPTS-12 may similarly contribute to an improved understanding of how pain and disability change over time.

The aim of the present study is to investigate how SPTS-12 scores change over time in a clinical sample of patients receiving care after surgery from the Toronto General Hospital Transitional Pain Service. The objectives are: (1) to estimate latent class mixed models to represent trajectories of SPTS-12 scores after surgery; (2) to describe the identified trajectory groups according to patient characteristics, pain history, and questionnaire data; and (3) to examine differences between trajectories for pain, pain disability, and morphine equivalent use at regular intervals after surgery (up to two weeks, three months, six months, one year, and two years).

Methods

Procedure. The current study was approved by the University Health Network (UHN) Research Ethics Board (certificate approval #s: 14-7705-AE and 16-5109) and the York University Research Ethics (Human Participants Review Subcommittee, certificate approval #: 14-7705-AE). See Appendix C for relevant approval paperwork.

Participants were eligible for the study if they were older than 18 years of age and met one or more of the following TPS inclusion criteria: (1) considered to be a “pain alert” patient

upon hospital admission (prior to surgery), whereby they have a history of chronic pain, drug abuse or misuse, present or past opioid use, or current use of methadone or buprenorphine, (2) spent more than three days observed by the Acute Pain Service postoperatively due to significant persistent postoperative pain, (3) postoperative pain management included more than 90 mg morphine equivalents (MME) per day of an opioid class drug , (4) postoperative interventional pain procedures are required, (5) subsequent pain consultations are required when no longer observed by Acute Pain Service, and (6) patients referred by attending surgeons at UHN. For the present study, only participants who underwent surgery were included in the final sample.

Eligible participants were subsequently approached either preoperatively or postoperatively by the TPS study coordinator who explained the study protocol. Participants provided written informed consent prior to involvement in the study. Participants were asked to complete a paper questionnaire package at various timepoints: preoperatively, after discharge from the hospital, at their first outpatient visit in the TPS, and each subsequent visit to the TPS. The present study only uses data obtained postoperatively.

Measures. As the TPS was established in 2014 as a clinical service, some of the measures administered as a part of the questionnaire package have evolved over time. Only the questionnaires relevant to the present research are described below, all of which have been included in the questionnaire package since the inception of the TPS.

Demographic and medical history. Demographic information including age and sex was collected. Preoperative variables were available for pre-existing medical conditions and diagnoses, prescription medication use, pain complaints, pain treatments, and surgery type. Daily MME dosage was available at each visit to the TPS.

Pain experience and interference. The Brief Pain Inventory (BPI; Cleeland & Ryan, 1994) assesses pain severity and interference. The BPI short form consists of nine items, with the first question asking if the participant is currently experiencing pain as well as the location of pain. The next four items use an 11-point Likert-type response scale asking participants to rate their current level of pain as well as the average, lowest, and worst level of pain they experienced in the previous 24 hours. For these questions, item anchors were 0 = *no pain* and 10 = *pain as bad as you can imagine*. Subsequent questions ask what medications or treatments participants are currently using for pain and the percentage of relief the medications or treatments are providing, with item anchors 0% = *no relief* and 100% = *complete relief*. The final question measures pain interference across seven domains (general activity, mood, walking ability, normal work, relations with other people, sleep, and enjoyment of life), with item anchors 0 = *does not interfere* and 10 = *completely interferes*. Pain severity items can be reported in two ways that are both supported by the Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials (IMMPACT; Dworkin et al., 2005; Dworkin et al., 2008): (1) as one mean severity score across all four items or (2) the score obtained on a single item, such as the items "average" or "worst" pain. In both cases, higher scores indicating worse pain severity. Pain interference is reported as the mean across the seven interference domains (Cleeland, 2009).

The commonly used BPI short form has good internal consistency (α = .80 to .87 for pain severity items and α = .89 to .92 for pain interference items; Cleeland et al., 1994) and good test-retest reliability with consecutive daily administration over the course of a week (r = .83 to .88 for pain severity items and r = .83 to .93 for pain interference items; Mendoza, Mayne, Rublee, & Cleeland, 2006). In the present sample, the BPI showed excellent internal consistency across all times points for pain severity (α = .92) and pain interference (α = .92) scores.

Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). The HADS consists of two subscales, one assessing symptoms of anxiety (HADS-A) and the other measuring symptoms of depression (HADS-D). The HADS was developed for use in outpatient primary or secondary care settings (Zigmond & Snaith, 1983). Each subscale consists of seven items, each using a four-point Likert-type response scale with different anchors depending on the item; for example, the first anxiety item asks participants to rate the degree to which they "feel tense or 'wound up' " with anchors 0 = *not at all*, 1 = *from time to time*, *occasionally*, 2 = *a lot of the time*, and 3 = *most of the time*. The first depression item asks if participants they "still enjoy the things I used to enjoy", with anchors 0 = *definitely as much*, 1 = *not quite so much*, 2 = *only a little*, and 3 = *hardly at all*. The total score range for each subscale is 0 to 21, with higher scores reflecting greater depressive and anxiety symptoms, respectively. Scores between 0 and 7 are considered in the normal range, scores between 8 and 10 are considered in the borderline range, and scores between 11 and 21 are considered in the abnormal range (Zigmond & Snaith, 1983). According to a review of 14 studies, internal consistency for both the HADS-A (α =.68 to .93, *M* = .83) and the HADS-D (α = .67 to .90, *M* = .82) ranges from poor to excellent (Bjelland et al., 2002). The HADS has good test-retest reliability over a 0- to 2-week period (HADS-A; *r* = .84, HADS-D; *r* = .85; Herrmann, 1997). Furthermore, the HADS has demonstrated good divergent validity by determining that the items on the HADS measure depression and anxiety rather than physical symptoms secondary to injury or illness and by establishing that the HADS can differentiate between depression and anxiety (Johnston et al., 2000). In the present sample, the HADS showed very good internal consistency across all times points for anxiety (α = .86) and depression (α = .85) scores.

Pain Catastrophizing Scale (PCS; Sullivan et al., 1995). The PCS assesses ruminative thinking, perception of threat, and feelings of helplessness in relation to painful experiences and sensations. It consists of 13 items using a five-point Likert-type response scale, with item anchors 0 = *not at all* and 4 = *all the time*. The total score range is 0 to 52 with higher scores indicating greater levels of pain catastrophizing. The PCS has good internal consistency (α = .87; Sullivan et al., 1995) and test-retest reliability over a six-week period (*r* = .75; Sullivan et al., 1995). The PCS has demonstrated strong discriminant validity by finding that individuals classified as "catastrophizers" endorsed more negative statements related to pain than individuals classified as "noncatastrophizers" (Osman et al., 1997; Sullivan et al., 1995). Total scores of 30 and higher are considered to be in the clinically relevant range (Sullivan, 2009). In the present sample, the PCS showed excellent internal consistency across all timepoints (α = .96).

Abbreviated PTSD Checklist – Civilian Version (Abbreviated PCL-C; Lang & Stein, 2005; Lang et al., 2012). The Abbreviated PCL-C is a shorter version of the PTSD Checklist – Civilian Version (PCL-C) that was developed to assess the traumatic response that individuals may have in response to a stressful event (Weathers et al., 1994). The abbreviated version consists of six items drawn from the original 17-item scale. Items are scored using a five-point Likert-type response scale, with item anchors 1 = *not at all* to 5 = *extremely*. The total score range is 6 to 30, with higher scores reflecting greater traumatic responses. The total PCL-C has good convergent and discriminant validity (Conybeare et al., 2012). The abbreviated PCL-C is highly correlated with the PCL-C total score (*r* = .97) and has demonstrated high sensitivity (.80) and specificity (.76) for detecting PTSD (Lang & Stein, 2005). The proposed total score cut-off to screen for PTSD for the abbreviated PCL-C is 14 (Lang & Stein, 2005; Lang et al., 2012). In the present sample, the abbreviated PCL-C showed good internal consistency across all times

points (α = .89). This questionnaire was only administered on the first participant visit to the TPS.

Sensitivity to Pain Traumatization Scale (SPTS-12; Katz et al., 2017). The SPTS-12 assesses the propensity to develop a traumatic response to pain. It consists of 12 items using a five-point Likert-type response scale, with item anchors 0 = *not at all true* and 4 = *entirely true*. The total score range is 0 to 48 with higher scores indicating a more traumatic response to pain. When the SPTS-12 was first developed (Katz et al., 2017), the wording of item 1 was "pain keeps me awake at night". Because data collection for the present study started in 2014, this original wording was used. However, the final version of the scale described by Katz et al. (2017) adapted the wording of this item to "when I am in pain, it keeps me awake at night" to make it applicable to individuals not experiencing pain at the time the scale is being completed. The SPTS-12 has good internal consistency both with the original wording of item 1 ("pain keeps me awake at night", α = .88) and the revised wording ("when I am in pain, it keeps me awake at night", α = .89). Both versions have demonstrated that the associations among the SPTS-12 items are well-explained by a one-factor structure and that it has very good reliability and validity in a clinical and community sample (Katz et al., 2017). In the present sample, the SPTS-12 had excellent internal consistency across all times points (α = .91).

Sample size estimation. Growth curve models have flexible requirements regarding the type and nature of data that can be used (Curran, Obeidat, & Losardo, 2010). Sample size estimates are based on the research goals and data characteristics. Generally speaking, at least 100 participants are recommended for analysis, with greater power associated with a larger sample size and a greater number of measurement occasions (Curran et al., 2010; Hertzog, Lindenberger, Ghisletta, & von Oertzen, 2006). Hertzog et al. (2006) showed that, given a

sample size of 200 with a moderate slope correlation effect size (r = .50) and a growth curve reliability above .9, four longitudinal measurement occasions is associated with a power to detect slope variances of greater than 20%, five occasions is associated with a power to detect slope variances of greater than 60%, and six occasions is associated with a power to detect slope variances of greater than 80%.

Statistical analyses. See Appendix E for the syntax used in the final analysis.

***Trajectory analysis*.** Latent-class mixed-model analysis for Gaussian longitudinal outcomes (i.e., normal distribution for error terms assumed at each timepoint) was used to estimate latent trajectories of SPTS-12 scores across days (0 to 1583) after surgery for participants with two or more TPS visits after surgery. Analyses were conducted in R (Version 3.6.0) using the package "lcmm" with the function "hlme" (Proust-Lima, Philipps, Diakite, & Liquet, 2019; Proust-Lima, Philipps, & Liquet, 2015; Proust-Lima, Philipps, & Liquet, 2017). Models with 1 to 8 latent trajectory classes (or groups) were estimated with a linear term and then again with the addition of a quadratic term to account for potential curvature in trajectories; as a result, a total of 16 models were estimated. Model fit was based on the lowest Bayesian information criterion (BIC) as well as model interpretability (Nylund, Asparouhov, & Muthén, 2007), and further required that each trajectory be composed of at least 5% of the total participant sample. Following selection of the model based on the number of trajectory groups, the significance of the quadratic term was used to determine if its inclusion improved the model.

Predictor analysis. Several predictors were evaluated by entering them individually into the final model as determined by the model fit requirements described above (i.e., a separate model was estimated for each predictor, included one-at-a-time). Predictors included the preoperative participant characteristics of age, sex, presence/absence of preoperative chronic

pain, preoperative daily dose of opioids in MME, the number of preoperative medical conditions, and surgery type. Predictor variables based on self-report scales were available post-surgically when participants filled out the questionnaire packages at their first visit to the TPS. Based on scores obtained from that visit, baseline scores for average pain, pain interference, depression, anxiety, trauma, pain catastrophizing, and MME/day were considered as predictors. Next, significant predictors were concurrently added to the model.

Outcome analysis. As a clinical service based on patient needs, the TPS did not restrict the time after surgery when participants were admitted to the TPS program nor were their requirements to attend the TPS at predetermined visits or for a specific number of visits. As a result, there is a tremendous degree of variability regarding how soon after surgery participants attended their first TPS visit, how frequently they attended the TPS, the time between TPS visits, and the total number of visits they had. This variability creates difficulty in understanding trends and characteristics across participants for a specific visit to the TPS. Therefore, to improve interpretability of the data, five standardized reporting periods were created based on the interval of time when participants were seen at the TPS after surgery: (1) up to two weeks (between 0-14 days), (2) three months (between 60 and 120 days), (3) six months (between 150-210 days), (4) one year (between 275-455 days), and (5) two years (between 550-910 days). For participants who had more than one TPS visit during a given reporting period, an average of the available scores was calculated to create one mean score for each available measure.

In order to evaluate how trajectory classes differed on important outcome measures at each of the five reporting periods, five separate one-way ANOVAs were conducted using trajectory class as the factor. Four dependent variables were used: MME/day, average pain intensity, pain interference, and HADS-D. These variables were chosen from the available array

due to their importance as outcome measure domains recommended for use in clinical trials on chronic pain (Dworkin et al., 2005). Additional variables were not evaluated due to the increased risk of false positives due to multiple comparisons. Significant omnibus tests were followed up with Tukey-Kramer post-hoc tests (Ruxton & Beauchamp, 2008; Toothaker, 1993). Due to the inflated Type I error rate due to multiple comparisons across several ANOVAs, a more conservative cut-off of $p < .001$ was used.

Results

Recruitment & missing data. A total of 766 participants were approached to participate in the study; 222 participants did not provide consent to participate. Of the 544 participants who provided consent, 113 were not surgical referrals to the TPS. Sixty-one participants were excluded from analysis due to having data for only one TPS visit after surgery. For all scales, total scores were prorated by calculating the mean score of the available items and then multiplying this mean by the total number of items in the scale to create an adjusted total score (Enders, 2010). Prorating was conducted for a given scale only for participants who completed 80% or more of the items on that scale. Nine participants were excluded from analysis because they did not have 80% or more of the SPTS-12 completed on at least two TPS visits, although participants with missing data for other variables (e.g., HADS-D, IEQ) were still included. The final sample consisted of 361 participants who provided consent, underwent surgery, had data from at least two TPS visits, and had sufficient data on the SPTS-12 (at least 80% of the SPTS-12 completed on at least two TPS visits). See Figure 4.1 for a diagram of the recruitment process.

Participant characteristics. Of the 361 participants included in the final analysis, the mean age was 50.59 years ($SD = 14.32$). The majority of the sample was male (55.40%; $n = 200$). Information regarding the ethnic background of participants was not available. The number

of participants' visits to the TPS ranged from 2 to 68 (M = 7.39, SD = 8.00; see Figure 4.2), with n = 291 participants with three or more visits, n = 185 with five or more visits, and n = 81 with 10 or more visits. The number of days between surgery and the first visit to the TPS ranged from 0 to 585 (M = 19.10, SD = 45.91). The number of days between surgery and hospital discharge ranged from 0 to 281 (M = 13.03, SD = 21.32).

Preoperative diagnoses included the following (non-mutually-exclusive): chronic pain (56.5%), cancer (50.3%), gastroesophageal reflux disease (50.2%), hypertension (45.6%), arthritis (38.4%), diabetes mellitus (25.6%), thyroid disease (19.2%), peripheral vascular disease (11.4%), chronic obstructive pulmonary disease (14.9%), asthma (15.0%), anemia (15.2%), sleep apnea (12.9%), chronic kidney failure (10.8%), congestive heart failure (10.4%), angina (10.8%), peptic ulcer disease (10.0%), myocardial infarction (10.0%), and deep vein thrombosis (9.1%). Data on the presence or absence of the above preoperative diagnoses varied, with the least data available for the presence of preoperative anemia (n = 230) and the most data available for the presence of chronic pain (n = 354). The number of preoperative diagnoses that participants presented with ranged between 0 to 20 (M = 3.54, SD = 2.51, n = 276). The number of medications that participants reported taking preoperatively ranged from 0 to 22 (M = 6.43, SD = 4.62, n = 275).

Surgery type included thoracic (24.7%), transplant (16.4%), general (11.7%), cardiac (10.8%), ear, nose, and throat (10.0%), vascular (8.6%), obstetrical/gynecological (3.9%), multiple surgeries (3.9%), plastic (3.3%), neurological (3.3%), urologic (2.8%), and orthopedic (0.8%).

Descriptive statistics. Descriptive statistics for the questionnaire data and MME/day for the complete sample are displayed in Table 4.1 according to the reporting period after surgery (i.e., up to two weeks, three months, six months, one year, and two years).

SPTS-12 trajectories. The model fit indices for the 16 models testing SPTS-12 total scores across time are displayed in Table 4.2. The best model consisted of five SPTS-12 trajectory groups for models tested (1) with a linear term and (2) with a linear term and a quadratic term. When five trajectories were included in the model, the quadratic term was no longer significant for any of the trajectory groups (ps > .10). The addition of a quadratic term to the model was only significant for the one-group model (p = .005). Therefore, the final model did not contain a quadratic term, with each of the five trajectory groups comprising more than 5% of the sample. See Table 4.3 for the trajectory characteristics of the final model.

Predictor analysis. Each of the 13 predictors was individually added to the model. Three predictors were significant with p < .05: depression, anxiety, and pain catastrophizing (see Table 4.4). However, when these predictors were added to the final model simultaneously, the complete-case sample size dropped to n = 89. With 75.35% of data missing, data imputation was considered inappropriate and listwise deletion would leave a sample size that is too small for trajectory analysis (Beckham et al., 1997; Curran et al., 2010; Schafer & Graham, 2002; Van De Schoot, Sijbrandij, Winter, Depaoli, & Vermunt, 2017). As the purpose of the present study is to evaluate the psychometric properties of SPTS-12 scores and how they change over time, excluding predictor variables from the trajectory analysis was considered to be an acceptable alternative, with the caveat that the findings should be interpreted with this limitation in mind.

Description of the SPTS-12 trajectory groups. Figure 4.3 shows a plot of the mean trajectories for the five SPTS-12 trajectory groups. Descriptive characteristics of the five

trajectory groups by reporting periods are displayed in Tables 4.5 to 4.9. Trajectory #1 is the largest trajectory group consisting of 154 participants, with a mean age of 50.79 years (*SD* = 14.99) and 56.49% (*n* = 87) are male. The SPTS-12 scores for this group start low (predicted value of SPTS-12 three days after surgery = 9.01) and remain low (predicted value of SPTS-12 two years after surgery = 7.20), significantly decreasing over time (*p* = .039). Trajectory #2 consists of 66 participants, with a mean age of 54.09 years (*SD* = 12.71) and 65.15% (*n* = 43) are male. This group starts with high SPTS-12 scores (predicted value of SPTS-12 three days after surgery = 27.63) that decrease significantly over time (predicted value of SPTS-12 two years after surgery = 25.46; *p* = .018). Trajectory #3 consists of 62 participants, with a mean age of 48.48 years (*SD* = 13.46) and 50.00% (*n* = 31) are male. The SPTS-12 scores for this group start out moderate (predicted value of SPTS-12 three days after surgery = 16.04) and do not change significantly over time (predicted value of SPTS-12 two years after surgery = 19.48; *p* = .095). Trajectory #4 consists of 43 participants with a mean age of 49.51 years (*SD* = 15.59) and 48.84% (*n* = 22) are male. This trajectory has the steepest downward slope, with the predicted SPTS-12 scores dropping from 21.17 three days after surgery to 11.33 two years after surgery (*p* < .001). Trajectory #5 consists of 36 participants with a mean age of 48.22 years (*SD* = 13.45) and 50.00% (*n* = 18) are male. This trajectory starts as the worst off with the highest SPTS-12 scores (predicted value of SPTS-12 three days after surgery = 36.85), which did not show a significant decrease over time (predicted value of SPTS-12 two years after surgery = 37.82; *p* = .415).

Outcome analysis. Figures 4.4 to 4.7 show how MME/day, average pain, pain interference, and depression scores change over time after surgery for the five SPTS-12 trajectory groups.

MME/day. Up to two weeks after surgery, MME/day did not significantly differ across trajectory groups, $F(4, 271) = 0.76$, $p = .56$. Three months after surgery, there were significant differences across trajectory groups, $F(4, 212) = 2.50$, $p = .04$. None of the follow-up post-hoc tests were significant according to the adjusted $p = .001$ cut-off. Six months after surgery, trajectory groups did not significantly differ, $F(4, 114) = 2.32$, $p = .06$. One year after surgery, there were significant differences across trajectory groups, $F(4, 98) = 3.13$, $p = .018$, but none of the post-hoc tests were significant. Two years after surgery, trajectory groups did not significantly differ, $F(4, 43) = 0.53$, $p = .72$.

Average pain intensity. Up to two weeks after surgery, average pain intensity assessed with the BPI did not significantly differ across trajectory groups, $F(4, 90) = 2.09$, $p = .09$. Three months after surgery, there were significant differences across the trajectory groups, $F(4, 182) = 12.78$, $p < .001$. Post-hoc tests showed that pain intensity for SPTS-12 Trajectory #1 ($M = 3.94$, $SD = 1.75$) was significantly lower than that for SPTS-12 Trajectory #2 ($M = 6.06$, $SD = 1.78$, $p < .001$), SPTS-12 Trajectory #3 ($M = 5.62$, $SD = 1.75$, $p = .001$), and SPTS-12 Trajectory #5 ($M = 6.36$, $SD = 2.41$, $p < .001$). Six months after surgery, there were significant differences between groups, $F(4, 89) = 6.04$, $p < .001$, but none of the post-hoc tests were significant. One year after surgery, there were significant differences between groups, $F(4, 95) = 7.92$, $p < .001$. Post-hoc tests showed that pain intensity for SPTS-12 Trajectory #1 ($M = 4.02$, $SD = 1.73$) was significantly lower than that for SPTS-12 Trajectory #2 ($M = 6.15$, $SD = 1.62$, $p < .001$) and SPTS-12 Trajectory #3 ($M = 6.18$, $SD = 1.68$, $p < .001$). Two years after surgery, trajectory groups did not significantly differ, $F(4, 48) = 0.66$, $p = .62$.

Pain interference. Up to two weeks after surgery, average pain interference assessed with the BPI did not significantly differ across trajectory groups, $F(4, 84) = 1.91$, $p = .12$. Three

months after surgery, there were significant differences across the trajectory groups, $F(4, 180) = 13.83$, $p < .001$. Post-hoc tests showed that pain interference scores for SPTS-12 Trajectory #1 ($M = 3.88$, $SD = 2.06$) were significantly lower than those for SPTS-12 Trajectory #2 ($M = 6.36$, $SD = 1.92$, $p < .001$), SPTS-12 Trajectory #3 ($M = 6.03$, $SD = 2.05$, $p < .001$), and SPTS-12 Trajectory #5 ($M = 6.68$, $SD = 2.37$, $p < .001$). Six months after surgery, there were significant differences between groups, $F(4, 88) = 12.35$, $p < .001$. Post-hoc tests showed that pain interference scores for SPTS-12 Trajectory #1 ($M = 3.88$, $SD = 2.29$) were significantly lower than those from SPTS-12 Trajectory #2 ($M = 6.42$, $SD = 1.82$, $p < .001$) and SPTS-12 Trajectory #5 ($M = 8.27$, $SD = .94$, $p < .001$). Furthermore, pain interference scores for SPTS-12 Trajectory #4 ($M = 4.65$, $SD = 2.56$) were significantly lower than those for SPTS-12 Trajectory #5 ($M = 8.27$, $SD = .94$, $p < .001$). One year after surgery, there were significant differences between groups, $F(4, 93) = 8.58$, $p < .001$. Post-hoc tests showed that pain interference scores for SPTS-12 Trajectory #1 ($M = 3.79$, $SD = 2.26$) were significantly lower than those for SPTS-12 Trajectory #2 ($M = 6.30$, $SD = 2.03$, $p = .001$), SPTS-12 Trajectory #3 ($M = 6.25$, $SD = 1.63$, $p < .001$), and SPTS-12 Trajectory #5 ($M = 6.75$, $SD = 2.68$, $p < .001$). Two years after surgery, there were significant differences between groups, $F(4, 48) = 7.73$, $p < .001$. Post-hoc tests showed that pain interference scores for SPTS-12 Trajectory #1 ($M = 4.91$, $SD = 1.90$) were significantly lower than those for SPTS-12 Trajectory #5 ($M = 7.88$, $SD = 1.62$, $p < .001$). Furthermore, pain interference scores for SPTS-12 Trajectory #4 ($M = 4.31$, $SD = 1.50$) were significantly lower than those for SPTS-12 Trajectory #5 ($M = 7.88$, $SD = 1.62$, $p = .001$).

Depression. Up to two weeks after surgery, average depression assessed with the HADS-D differed significantly across trajectory groups, $F(4, 87) = 12.44$, $p < .001$. Post-hoc tests showed that HADS-D scores for Trajectory #1 ($M = 6.41$, $SD = 3.70$) were significantly lower

than those for SPTS-12 Trajectory #2 (M = 11.42, SD = 4.14, p < .001), SPTS-12 Trajectory #4 (M = 11.00, SD = 2.84, p < .001), and SPTS-12 Trajectory #5 (M = 15.75, SD = 2.22, p < .001). Three months after surgery, there were significant differences across trajectory groups, F(4, 176) = 12.05, p < .001. Post-hoc tests showed that HADS-D scores for SPTS-12 Trajectory #1 (M = 6.26, SD = 3.77) were significantly lower than those for SPTS-12 Trajectory #2 (M = 10.85, SD = 3.76, p < .001) and SPTS-12 Trajectory #5 (M = 11.38, SD = 4.69, p < .001). Six months after surgery, there were significant differences between groups, F(4, 79) = 12.44, p < .001. Post-hoc tests showed that HADS-D scores for SPTS-12 Trajectory #1 (M = 6.26, SD = 2.83) were significantly lower than those for SPTS-12 Trajectory #2 (M = 11.07, SD = 3.27, p < .001) and SPTS-12 Trajectory #5 (M = 14.13, SD = 2.61, p < .001). Furthermore, HADS-D scores for SPTS-12 Trajectory #4 (M =8.04, SD = 4.84) were significantly lower than those for SPTS-12 Trajectory #5 (M = 14.13, SD = 2.61, p < .001). One year after surgery, there were significant differences between groups, F(4, 89) = 9.27, p < .001. Post-hoc test showed that HADS-D scores for SPTS-12 Trajectory #1 (M = 5.93, SD = 3.92) were significantly lower than those for SPTS-12 Trajectory #2 (M = 11.00, SD = 3.96, p < .001) and SPTS-12 Trajectory #5 (M = 12.41, SD = 4.31, p < .001). Two years after surgery, there were significant differences between groups, F(4, 47) = 6.01, p = .001, but none of the post-hoc tests were significant.

Discussion

The aim of the present study was to further validate the SPTS-12 by examining how its scores change over time in a postsurgical population of patients receiving care at the Transitional Pain Service. To represent patient heterogeneity, latent class mixed modeling was used. The optimally fitting model consisted of five SPTS-12 trajectories, three of which were characterized by significantly decreasing scores over time. Analysis of pain-related outcomes predicted by

SPTS-12 trajectories provided evidence of criterion validity of the SPTS-12. Although SPTS-12 trajectories did not significantly differ on MME/day at any timepoint, average pain, pain interference, and depression scores differed at two or more postsurgical visits. Taken together, the present study supported the long-term stability of SPTS-12 scores, as well as the ability of the SPTS-12 to predict important pain-related outcomes over time.

SPTS-12 trajectory analysis. The latent class mixed model analysis supported a five-trajectory model of SPTS-12 scores over days since surgery. Trajectory #1 was the largest group (42.7% of the sample) and showed the best overall outcome, with SPTS-12 scores starting low and remaining low. In other words, the individuals in Trajectory #1 did not consistently endorse experiencing symptoms suggesting that they were experiencing a traumatic response to pain after surgery. Furthermore, their mean scores on the PCL-C at each timepoint did not exceed the proposed cut-off score to screen for PTSD (Lang & Stein, 2005; Lang et al., 2012), nor were the means for this group in the clinically relevant range for depression, anxiety, or pain catastrophizing (Sullivan, 2009; Zigmond & Snaith, 1983). This finding is consistent with previous research showing that most individuals who undergo surgery do not develop problematic mental health symptoms (Cremeans-Smith et al., 2011; O'Connor et al., 2011; Oxlad & Wade, 2008; Stoll et al., 2000).

Trajectory #3 started with the next lowest SPTS-12 scores, which remained stable over time. This group met clinical cut-offs for depression six months and two years after surgery, for anxiety two years after surgery, and for trauma at three months and one year after surgery. Individuals in Trajectory #4 showed the steepest decrease in SPTS-12 scores, starting at 21 three days after surgery and decreasing nearly in half two years later. The initially higher SPTS-12 scores were associated with scores meeting the clinical cut-offs for depression and anxiety up to

two weeks after surgery. On average, the proposed abbreviated PCL-C cut-off score to screen for PTSD was exceeded at three months and one year after surgery by individuals in this group.

Trajectories #2 and #5 were the worst off, with both groups starting with endorsing high levels of symptoms that are suggestive of a traumatic response to pain. SPTS-12 scores decreased significantly (though clinically, only slightly) for Trajectory #2. In contrast, scores did not change for Trajectory #5. For both of these trajectory groups, mean scores on the abbreviated PCL-C exceeded the proposed cut-off score to screen for PTSD at all available timepoints. Moreover, both trajectory groups had mean scores above the clinically relevant range for depression and anxiety at all timepoints, with high reported levels of pain catastrophizing at most timepoints. Together, Trajectories #2 and #5 comprised 28.3% of the sample, demonstrating that a sizable proportion of the sample endorsed considerable psychological distress. These statistics are similar to rates of anxiety and depression identified in previous research. For example, in a sample of heart-lung transplant recipients, Stilley et al. (1999) found that depression and anxiety were clinically significant in 26.5% and 34.6% of patients up to a year and a half after surgery. In addition, researchers have estimated that probable PTSD diagnoses are present in between 15 and 20% of patients after surgery (Cremeans-Smith et al., 2011; O'Connor et al., 2011; Stoll et al., 2000). The higher rates of reported pain catastrophizing comprise a unique finding which is important because individuals reporting higher levels of pain catastrophizing experience more pain for a longer duration after surgery (Granot & Ferber, 2005; Pavlin, Sullivan, Freund, & Roesen, 2005; Strulov et al., 2007).

The identification of five distinct SPTS-12 trajectory patterns highlights the heterogeneity of pain-related trauma symptoms among individuals after surgery. This heterogeneity would not have been represented using the overall sample average, as is done in most prospective studies of

patients after surgery. To illustrate this point, the one-trajectory model of SPTS-12 scores over days since surgery (Figure 4.8) demonstrates that SPTS-12 scores start moderately high, decrease gradually, and then start to increase approximately one and a half years after surgery. This model does not adequately reflect the nuanced fluctuations of subgroups with different growth curves, and thus misrepresents the symptom experience of many individuals after surgery. Accordingly, this heterogeneity has implications for treatment recommendations: knowing that an individual has a high SPTS-12 score at the outset of treatment could introduce an opportunity to intervene and address the symptoms related to a traumatic response to pain.

Outcome analysis. Consistent with the IMMPACT guidelines (Dworkin et al., 2005), four outcome measures related to chronic pain were evaluated: MME/day, average pain and pain interference as assessed by the BPI, and depression as assessed by the HADS-D.

MME/day use. For all timepoints after surgery, SPTS-12 trajectories did not significantly differ according to MME/day use. This result suggests that morphine equivalent dosing relies more heavily on patient-specific variables, such as surgery type, than was captured with the SPTS-12 trajectories. For example, Overton et al. (2018) published prescription recommendations proposing MME/day dosage rates based on specific surgical procedures, with suggested ranges for general surgery, breast surgery, thoracic surgery, orthopaedic surgery, gynecologic surgery/obstetric delivery procedures, urologic surgery, otolaryngology, and cardiac surgery. Variability in dosing can also differ depending on past experience with opioids (e.g., opioid-naïve or a history of using opioids) as well as additional sociocultural factors that when taken together can make accurately predicting opioid use challenging (Tan et al., 2018). Overall, this literature suggests that the considerable variability involved in prescribing opioids may have contributed to the lack of detection of differences between SPTS-12 trajectories.

Average pain. Within the first two weeks after undergoing surgery, SPTS-12 trajectory groups did not differ according to average pain. This result suggests that during the acute period following surgery, individuals with differing levels of SPTS-12 have similar pain management and pain experiences. In contrast, three months after surgery, pain ratings from individuals in Trajectory #1 were significantly lower than for Trajectories #2, #3, and #5, and one year after surgery, individuals in Trajectory #1 were significantly lower than Trajectories #2 and #3, with no significant differences two years after surgery. Thus, the relationship between SPTS-12 trajectories and reported pain level varies over time. The lack of difference immediately following surgery may be related to the universal experience and management of pain after surgery, as surgery necessarily involves a certain degree of tissue damage at the surgical site (Reddi & Curran, 2014). Therefore, pain after surgery is expected, which is captured in the commonly used definition of acute pain as "the normal, predicted physiological response to an adverse chemical, thermal or mechanical stimulus . . . associated with surgery, trauma and acute illness" (Federation of State Medical Boards of the United States, 1998, p. 7). Furthermore, the absence of immediate differences may be partly because all patients receive opioids as a part of standard pain management in the days after surgery.

In contrast, in the months after surgery, reported pain levels begin to differ, with individuals in Trajectory #1 consistently reporting the lowest levels of pain. As the SPTS-12 measures a traumatic response to pain, it stands to reason that higher reported levels of pain occur for individuals in SPTS-12 trajectories characterized by a greater number of symptoms. For example, with higher levels of reported pain, it is more likely that individuals will report more avoidance of pain and higher sensitivity to pain. Furthermore, this effect may be bidirectional, wherein psychological factors can, in themselves, contribute to an individual's

reported pain experience (Linton, 2000; Pincus, Burton, Vogel, & Field, 2002; Turk & Okifuji, 2002); Sullivan et al. (2001) estimated that between 7 to 31% of the variance in pain ratings can be accounted for by pain catastrophizing. Therefore, it is also likely that the SPTS-12 may account for some of the variance of reported pain levels in the present study.

Pain interference. Reported pain interference levels varied between SPTS-12 trajectories at all timepoints except for during the first two weeks after surgery. Like average reported pain levels, it is possible that the initial lack of difference is due to expected healing time after surgery when limiting engagement in activities and movements may prevent further tissue damage. However, after three months, individuals in Trajectories #2, #3, and #5, all with higher SPTS-12 scores, report greater pain interference than Trajectory #1, with similar findings again six months, one year, and two years after surgery. Within each reporting period, the trajectory with the highest reported SPTS-12 scores consistently had the worst pain interference scores. This finding is especially compelling when considered alongside the findings regarding average pain intensity, since the differences between pain interference across trajectories remained significant over time. Although pain intensity and pain interference are similar constructs, they are distinct: pain intensity is measured by asking individuals to rate their current level of pain, whereas pain interference refers to how much their pain has disrupted their general activities, their mood, walking ability, normal work, their relations with other people, sleep, and enjoyment of life (Cleeland, 2009; Cleeland & Ryan, 1994). In this manner, the way that pain is perceived as impacting daily living may be more important than the severity of pain itself. Cuff, Fann, Bombardier, Graves, and Kalpakjian (2014) found that in a sample of individuals with acute spinal cord injury, pain interference accounted for 13% to 26% of variance in depression scores, whereas pain severity only accounted for 0.2% to 1.2%. Although disruptions to quality of life

may be more pronounced after spinal cord injury, this finding still addresses how interference of daily activities may have a greater impact than pain ratings on their own. In the current sample, the differences between SPTS-12 trajectories on pain interference scores demonstrates the strong, consistent relationship in individuals with more severe symptoms of pain-related trauma and the perceived impact of pain on various life domains. Importantly, this association is maintained even two years after surgery.

Furthermore, clinicians and researchers have moved away from considering a reduction in pain intensity scores as the only treatment goal in pain management (Ballantyne & Sullivan, 2015; McCracken, Carson, Eccleston, & Keefe, 2004). Ballantyne and Sullivan (2015) argue that focusing solely on pain intensity can contribute to the current misuse of opioids, where increasing doses in an attempt to "titrate to effect" not only can fail to reduce pain, but can also increase addiction and mortality rates. Instead, an empirical and clinical consideration of suffering and pain interference may be a more appropriate way to improve patient quality of life. This aim has been proposed to be accomplished by incorporating psychosocial treatments in conjunction with medical interventions that target symptoms of hopelessness and uncertainty that accompany the experience of chronic pain. By focusing on the willingness to accept pain and engage in valued activities, patients can experience a reduction in pain disability, anxiety, depression, and even pain severity (McCracken & Eccleston, 2003; Reiner, Tibi, & Lipsitz, 2013; Veehof, Trompetter, Bohlmeijer, & Schreurs, 2016). The association between psychological well-being and pain interference is evident in the present study in that two years after surgery, trajectory groups continued to differ in pain interference scores, but not in pain intensity scores. It may be that higher reported levels of pain traumatization become more linked

with pain-related interference and suffering over time and less related to the amount of pain that patients report experiencing.

Depression. Within two weeks of surgery, rates of reported depression significantly differed between trajectories, with scores of individuals in Trajectory #1 being significantly lower than Trajectories #2, #4, and #5. While more universally higher ratings of pain and pain interference may be expected in the acute phase after surgery, mood disruptions also appear to be more common in individuals with higher reported levels of traumatic symptomatology related to pain: in the acute period following surgery, Trajectories #2, #4, and #5 have the highest SPTS-12 scores as well as the highest depression scores (as measured by the HADS-D). This finding may reflect the high level of comorbidity between trauma and mood-related disorders; it has been estimated that approximately half of individuals with a diagnosis of PTSD have a comorbid diagnosis of depression (Breslau, Davis, Peterson, & Schultz, 1997; Breslau et al., 1998; Rytwinski, Scur, Feeny, & Youngstrom, 2013). The considerable overlap between symptoms of trauma and a traumatic response to pain (Katz et al., 2017) likely helps account for the present findings. Furthermore, although depression scores were significantly higher for individuals in Trajectory #4 compared to Trajectory #1 in the first two weeks after surgery, they dropped over time alongside SPTS-12 scores. This finding is important because depression is considered a psychosocial vulnerability in the development of chronic postsurgical pain (Althaus, Arránz Becker, & Neugebauer, 2014; Hinrichs-Rocker et al., 2009). Although this link was also observed in Trajectories #2 and #5, there appears to be a subset of individuals in Trajectory #4 who experienced a subsequent decline in symptoms and overall better recovery.

In the months following surgery, the relationship between reported depression symptoms and STPS scores begins to change across trajectories. The STPS and depression scores begin to

drop for individuals in Trajectory #4 to the point that six months after surgery, depression levels are significantly lower than those for Trajectory #5. In contrast, the two trajectory groups with the highest levels of SPTS-12 scores (Trajectories #2 and #5) continued to report significantly higher symptoms of depression than the trajectory with the lowest SPTS-12 scores (Trajectory #1) at three months, six months, and one year after surgery. Notably, there were no longer any significant differences in depression scores between trajectory groups two years after surgery.

The pattern of depression scores after surgery is important because depression is a risk factor for adverse events following surgery. For example, higher levels of depression symptoms increase the risk of heart attack and hospital readmissions after coronary artery bypass graft surgery (Connerney, Shapiro, McLaughlin, Bagiella, & Sloan, 2001; Tully, Baker, Turnbull, & Winefield, 2008), increase the average length of hospital stay after thoracic surgery (Kitagawa, Yasui-Furukori, Tsushima, Kaneko, & Fukuda, 2011), predict mortality after cardiac valve surgery (Ho et al., 2005), and contribute to greater disability, worse symptom severity, and reduced walking capacity after lumbar spinal stenosis surgery (Sinikallio et al., 2011). Therefore, better understanding the link between depression and sensitivity to pain traumatization over time may help disentangle the complex, relative function that each plays in the development of chronic postsurgical pain.

Future directions and limitations. The present study has several limitations. First, participation in the study did not require patients to attend appointments in the TPS a specified number of times or at pre-determined intervals of time (e.g., attend the clinic at least once every month). Consequently, the number of participant appointments varied greatly ($M = 7.39$, $SD = 8.00$), with the number of days between surgery and the first visit to the TPS taking place as late as a year and a half after surgery. Intervals between appointments also varied greatly. For these

reasons, generalizability is likely affected in that individuals who experienced more pain, disability, and psychological distress may have attended appointments more frequently and for longer periods post-surgery than individuals that did not attend as many appointments. For this reason, the current results must consider that the trajectory in the long term may be artificially flat, as it does not account for those no longer participating in the service. Missing data due to participant drop-out or discontinuation is a common problem in clinical research (Little et al., 2012; Turk, Rudy, & Sorkin, 1993). Having access to follow-up responses for less than 30% of the initial sample size is not unusual (Turk et al., 1993). Follow-up research is needed to ensure the consistency of the present findings both in the TPS and in samples from other hospitals.

Second, there was a considerable amount of missing data from participants not completing all questionnaire items. While a total of 361 participants had complete data available for the SPTS-12, only 89 participants also had completed at least 80% of other questionnaires included in the package. For this reason, the final SPTS-12 trajectory model was unable to include important predictors (i.e., depression, anxiety, and pain catastrophizing); these were instead assessed in post-hoc analyses. Reasons for missing data are likely related to both participant characteristics (e.g., beliefs and attitudes that individuals have about the research topic) and study design characteristics (e.g., length of questionnaire, some questions on the back of the page that participants cannot readily see; McKnight et al., 2007). Another possible explanation for missing data in the present study is that participants were provided with questionnaire packages prior to their appointments, and it is possible that short waiting times might have contributed to a failure to finish filling them out. In the future, this confounding factor could be partly addressed by randomizing the order of scales within the questionnaire packages to help prevent systematic bias of failing to complete a given scale.

Third, the present study examined a complex heterogenous sample of patients. The mean number of preoperative diagnoses that participants had was 3.54 and included cancer, chronic obstructive pulmonary disease, and congestive heart disease. Furthermore, the type of surgery that participants underwent was diverse. It is possible that the pattern of findings would differ according to a specific diagnosis or surgery. For example, thoracic surgery is associated with some of the highest incidence rates of chronic pain after surgery, with a recent meta-analysis estimating that six months after thoracotomy, 47% of patients report pain (Bayman & Brennan, 2014), whereas modified radical mastectomy is associated with chronic pain incidence of 10% to 15% one year after surgery (Tasmuth, Blomqvist, & Kalso, 1999). Thus, it is possible that individuals who undergo a more painful surgery will experience more symptoms of pain-related trauma following the surgery. Although it has been suggested that psychological factors are more important than surgery type (Hinrichs-Rocker et al., 2009), future research should examine differences in SPTS-12 scores and their trajectories based on surgery type. Even while the heterogeneity of the sample introduces potential sources of variance, it is noteworthy that even across the considerable diversity of the sample, distinct SPTS-12 trajectories were identified. This may reflect that symptoms of pain traumatization are generalizable across sample characteristics such as surgery type and medical diagnosis, although future research is needed to further investigate this assertion.

Conclusion

The present study examined how SPTS-12 scores changed over time in a clinical sample of patients receiving care in the TPS at Toronto General Hospital after surgery. Using latent class growth mixture models, five prototypical SPTS-12 trajectory patterns were identified. Trajectory #1 represented the largest proportion of the sample and began with the lowest SPTS-12 scores

that stayed low for up to 2 years after surgery; Trajectory #2 started with high SPTS-12 scores that decreased significantly over time; Trajectory #3 started with moderate SPTS-12 scores that did not significantly change over time; Trajectory #4 started with moderately high SPTS-12 scores that significantly dropped over time; Trajectory #5 started with high SPTS-12 scores that remained high over time. Outcome analyses evaluated whether MME/day, average pain, pain interference, and depression scores differed across trajectory groups up to two weeks after surgery, three months, six months, one year, and two years after surgery. There were significant differences between trajectory groups for average pain, pain interference, and depression, but not for MME/day. Study limitations included variable participant attendance to the TPS, missing data, and clinical heterogeneity.

Table 4.1. Descriptive data for the total sample (N = 361).

	Up to two weeks (0-14 days after surgery)		Three months (60-120 days after surgery)		Six months (150-210 days after surgery)		One year (275-455 days after surgery)		Two years (550-910 days after surgery)	
	n	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)
MME/day	276	108.81 (100.62)	217	70.89 (86.89)	119	59.02 (86.18)	103	46.73 (57.63)	48	50.30 (84.95)
≤ 90 MME/day	169	-	165	-	98	-	86	-	41	-
> 90 MME/day	107	-	52	-	21	-	17	-	7	-
BPI-P	95	5.58 (1.93)	187	4.95 (2.05)	94	5.35 (2.13)	100	5.15 (1.95)	53	5.43 (1.60)
BPI-I	89	6.82 (2.05)	185	5.11 (2.36)	93	5.50 (2.50)	98	5.21 (2.40)	53	5.92 (1.93)
HADS-D	92	8.91 (4.41)	181	8.41 (4.40)	84	9.18 (4.28)	94	8.57 (4.56)	52	9.00 (4.77)
HADS-A	92	8.69 (4.35)	180	8.65 (4.75)	84	8.98 (4.49)	94	8.76 (4.62)	52	9.36 (4.00)
PCL-C	86	12.81 (5.60)	40	12.78 (5.49)	15	14.23 (4.67)	12	14.33 (6.83)	7	14.43 (5.00)
PCS	92	20.10 (12.54)	153	18.98 (13.82)	71	20.60 (15.42)	85	20.73 (13.99)	50	22.89 (14.13)
SPTS-12	92	19.03 (10.24)	116	16.58 (10.76)	51	19.24 (11.47)	71	16.27 (10.24)	49	18.73 (10.95)

Note. MME – mg morphine equivalent, BPI-P - Brief Pain Inventory - Average Pain subscale, BPI-I - Brief Pain Inventory - Pain Interference subscale, HADS-D - Hospital Anxiety and Depression Scale - Depression subscale, HADS-A - Hospital Anxiety and Depression Scale - Anxiety subscale, PCL-C - PTSD Checklist - Civilian Version, PCS - Pain Catastrophizing Scale, SPTS-12 – 12-item Sensitivity to Pain Traumatization Scale

Table 4.2. Model fit indices for Sensitivity to Pain Traumatization Scale (SPTS-12) scores.

Number of trajectory groups	Linear			Linear + quadratic		
	AIC	BIC	All *n*'s > 5%?	AIC	BIC	All *n*'s > 5%?
1	8063.04	8074.70	Yes	8057.156	8072.71	Yes
2	7525.47	7548.81	Yes	7529.137	7560.25	Yes
3	7350.43	7385.43	Yes	7308.417	7355.08	Yes
4	7251.48	7298.15	Yes	7239.742	7301.96	Yes
5	7237.26	7295.60	Yes	7223.688	7301.47	Yes
6	7228.73	7298.73	No	7228.914	7322.25	No
7	7220.65	7302.32	No	7222.862	7331.75	Yes
8	7223.40	7316.73	No	7210.384	7334.83	No

Note. AIC – Akaike information criterion, BIC – Bayesian information criterion.

Table 4.3. Characteristics of the Sensitivity to Pain Traumatization Scale (SPTS-12) trajectory groups for the final five-trajectory model.

Trajectory group	*n*	Intercept	Slope	*p*	Predicted values				
					3 days	90 days	180 days	365 days	730 days
#1	154	9.02157	-0.0025	.039	9.01	8.80	8.57	8.11	7.20
#2	66	27.93279	-0.0034	.018	27.92	27.63	27.32	26.70	25.46
#3	62	16.02601	0.0047	.095	16.04	16.45	16.88	17.75	19.48
#4	43	21.20954	-0.0135	<.001	21.17	19.99	18.77	16.27	11.33
#5	36	36.84238	0.0014	.415	36.85	36.96	37.08	37.33	37.82
Full sample*	361	19.33	0.0080	.017	-	-	-	-	-

Note. *Since the quadratic term was significant for the one-trajectory model ($p = .005$), the characteristics for the full sample are based on the model including the quadratic term.

Table 4.4. Fixed effects of the three significant predictors, depression, anxiety, and pain catastrophizing, with trajectory 5 of the Sensitivity to Pain Traumatization Scale (SPTS-12) being the trajectory of reference.

	Coefficient	SE	Wald	*p*
HADS-D (*n* = 89)				
HADS_D class1	-0.490	0.157	-3.133	.002
HADS_D class2	-0.612	0.162	-3.788	< .001
HADS_D class3	-0.165	0.179	0.920	.358
HADS_D class4	-0.209	0.138	-1.512	.130
HADS-A (*n* = 91)				
HADS_A class1	-0.791	0.212	-3.727	< .001
HADS_A class2	-0.596	0.243	-2.453	.014
HADS_A class3	-0.332	0.172	-1.927	.054
HADS_A class4	-0.284	0.168	-1.688	.092
PCS (*n* = 91)				
PCS class1	-0.271	0.072	-3.753	< .001
PCS class2	-0.230	0.093	-2.483	.013
PCS class3	-0.166	0.061	-2.730	.006
PCS class4	-0.074	0.086	-0.864	.388

Note. Class – trajectory, HADS-D - Hospital Anxiety and Depression Scale - Depression subscale, HADS-A - Hospital Anxiety and Depression Scale - Anxiety subscale, PCS - Pain Catastrophizing Scale.

Due to the small sample size and the fact that 75.35% of data is missing for these analyses, the decision was made to report these findings but not to interpret them. The final model did not include any predictors.

Table 4.5. Descriptive data for the Sensitivity to Pain Traumatization Scale (SPTS-12) trajectory groups for the final model up to two weeks (0-14 days) after surgery.

	Trajectory #1		Trajectory #2		Trajectory #3		Trajectory #4		Trajectory #5	
	n	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)
MME/day[a]	120	114.27 (106.79)	50	89.95 (66.57)	45	104.45 (92.76)	37	109.73 (99.28)	24	127.55 (139.22)
≤ 90 MME/day	68	-	34	-	29	-	23	-	15	-
> 90 MME/day	52	-	16	-	16	-	14	-	9	-
BPI-P[a]	37	4.91 (2.13)	18	6.03 (1.59)	17	5.74 (2.02)	19	6.24 (1.53)	4	6.00 (1.41)
BPI-I[a]	33	6.34 (2.36)	17	7.18 (2.21)	16	6.82 (1.57)	19	6.84 (1.54)	4	9.11 (0.67)
HADS-D[a]	34	6.41 (3.70)	19	11.42 (4.14)	17	7.26 (3.67)	18	11.00 (2.84)	4	15.75 (2.22)
HADS-A	34	5.74 (2.85)	19	10.84 (4.22)	17	8.61 (3.28)	18	10.92 (3.81)	4	14.00 (6.63)
PCL-C	33	9.97 (2.85)	17	16.59 (7.58)	15	12.53 (2.26)	17	13.24 (5.26)	4	19.50 (8.35)
PCS	36	12.25 (8.47)	17	30.20 (13.66)	17	19.14 (9.59)	18	23.99 (11.20)	4	34.46 (7.29)
SPTS-12	35	9.22 (3.79)	18	31.70 (3.85)	17	16.63 (3.35)	18	23.05 (2.74)	4	40.00 (5.60)

Note. MME – mg morphine equivalent, BPI-P - Brief Pain Inventory - Average Pain subscale, BPI-I - Brief Pain Inventory - Pain Interference subscale, HADS-D - Hospital Anxiety and Depression Scale - Depression subscale, HADS-A - Hospital Anxiety and Depression Scale - Anxiety subscale, PCL-C - PTSD Checklist - Civilian Version, PCS - Pain Catastrophizing Scale, SPTS-12 – 12-item Sensitivity to Pain Traumatization Scale.

[a] See text for a description of statistical tests conducted on these variables.

Table 4.6. Descriptive data for the Sensitivity to Pain Traumatization Scale (SPTS-12) trajectory groups for the final model three months (60-120 days) after surgery.

	Trajectory #1		Trajectory #2		Trajectory #3		Trajectory #4		Trajectory #5	
	n	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)
MME[a]	91	55.26 (73.76)	42	92.20 (90.22)	35	74.08 (109.05)	26	56.79 (48.52)	23	104.86 (110.96)
≤ 90 MME/day	78	-	25	-	27	-	22	-	13	-
> 90 MME/day	13	-	17	-	8	-	4	-	10	-
BPI-P[a]	82	3.94 (1.75)	36	6.06 (1.78)	26	5.62 (1.75)	27	5.08 (1.84)	16	6.36 (2.41)
BPI-I[a]	81	3.88 (2.06)	36	6.36 (1.92)	26	6.03 (2.05)	27	5.41 (2.26)	15	6.68 (2.37)
HADS-D[a]	80	6.26 (3.77)	36	10.85 (3.76)	24	8.83 (4.05)	26	9.50 (4.17)	15	11.38 (4.69)
HADS-A	79	6.22 (3.84)	36	10.95 (4.00)	24	8.35 (4.50)	26	10.31 (4.70)	15	13.53 (3.92)
PCL-C	24	9.58 (2.73)	5	17.00 (3.81)	2	14.00 (2.83)	5	14.60 (3.36)	4	23.75 (4.43)
PCS	65	10.79 (10.25)	30	30.33 (10.98)	22	16.52 (8.59)	23	20.33 (11.66)	13	35.51 (13.94)
SPTS-12	56	7.85 (4.19)	19	27.80 (4.24)	15	17.59 (2.55)	16	19.93 (4.88)	10	37.23 (7.04)

Note. MME – mg morphine equivalent, BPI-P - Brief Pain Inventory - Average Pain subscale, BPI-I - Brief Pain Inventory - Pain Interference subscale, HADS-D - Hospital Anxiety and Depression Scale - Depression subscale, HADS-A - Hospital Anxiety and Depression Scale - Anxiety subscale, PCL-C - PTSD Checklist - Civilian Version, PCS - Pain Catastrophizing Scale, SPTS-12 – 12-item Sensitivity to Pain Traumatization Scale.

[a]See text for a description of statistical tests conducted on these variables.

Table 4.7. Descriptive data for the Sensitivity to Pain Traumatization Scale (SPTS-12) trajectory groups of the final model six months (150-210 days) after surgery.

	Trajectory #1		Trajectory #2		Trajectory #3		Trajectory #4		Trajectory #5	
	n	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)
MME[a]	38	38.90 (44.13)	31	94.94 (136.70)	14	45.14 (71.31)	19	41.34 (43.77)	17	69.67 (69.47)
≤ 90 MME/day	35	-	21	-	11	-	18	-	13	-
> 90 MME/day	3	-	10	-	3	-	1	-	4	-
BPI-P[a]	30	4.16 (1.59)	26	6.01 (1.99)	9	6.53 (1.12)	19	5.02 (2.61)	10	6.82 (1.71)
BPI-I[a]	30	3.88 (2.29)	26	6.42 (1.82)	9	6.81 (1.29)	18	4.65 (2.56)	10	8.27 (0.94)
HADS-D[a]	28	6.26 (2.83)	23	11.07 (3.27)	7	11.02 (2.95)	17	8.04 (4.84)	9	14.13 (2.61)
HADS-A	28	5.21 (3.06)	23	11.85 (3.11)	7	7.19 (2.93)	17	9.02 (3.83)	9	14.71 (1.55)
PCL-C	5	10.80 (4.27)	3	18.00 (4.00)	3	12.33 (4.51)	4	17.10 (2.20)	0	-
PCS	24	6.66 (5.56)	18	28.53 (11.58)	6	27.72 (11.22)	15	19.24 (13.92)	8	41.78 (9.17)
SPTS-12	16	7.42 (3.21)	11	25.96 (4.73)	6	17.57 (6.94)	11	17.79 (5.28)	7	39.43 (3.11)

Note. MME – mg morphine equivalent, BPI-P - Brief Pain Inventory - Average Pain subscale, BPI-I - Brief Pain Inventory - Pain Interference subscale, HADS-D - Hospital Anxiety and Depression Scale - Depression subscale, HADS-A - Hospital Anxiety and Depression Scale - Anxiety subscale, PCL-C - PTSD Checklist - Civilian Version, PCS - Pain Catastrophizing Scale, SPTS-12 – 12-item Sensitivity to Pain Traumatization Scale.

[a] See text for a description of statistical tests conducted on these variables.

Table 4.8. Descriptive data for the Sensitivity to Pain Traumatization Scale (SPTS-12) trajectory groups for the final model one year (275-455 days) after surgery.

	Trajectory #1		Trajectory #2		Trajectory #3		Trajectory #4		Trajectory #5	
	n	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)
MME[a]	41	27.26 (31.95)	17	82.43 (77.48)	17	49.50 (41.72)	12	48.02 (53.17)	16	54.74 (83.51)
≤ 90 MME/day	41	-	9	-	14	-	9	-	13	-
> 90 MME/day	0	-	8	-	3	-	3	-	3	-
BPI-P[a]	39	4.02 (1.73)	17	6.15 (1.62)	21	6.18 (1.68)	10	4.94 (1.07)	13	5.77 (2.24)
BPI-I[a]	38	3.79 (2.26)	17	6.30 (2.03)	21	6.25 (1.63)	10	4.71 (1.58)	12	6.75 (2.68)
HADS-D[a]	38	5.93 (3.92)	16	11.00 (3.96)	19	10.02 (3.78)	10	7.67 (3.63)	11	12.41 (4.31)
HADS-A	38	5.57 (3.26)	16	11.91 (3.81)	19	9.24 (4.30)	10	9.86 (3.96)	11	13.42 (3.12)
PCL-C	5	9.60 (4.16)	2	13.50 (2.12)	4	16.75 (4.35)	0	-	1	30 (-)
PCS	34	8.75 (8.19)	14	33.95 (8.25)	18	26.07 (8.81)	9	17.79 (9.45)	10	36.00 (10.88)
SPTS-12	31	7.67 (4.47)	9	27.90 (4.47)	15	18.95 (3.86)	8	14.82 (4.42)	8	35.95 (7.30)

Note. MME – mg morphine equivalent, BPI-P - Brief Pain Inventory - Average Pain subscale, BPI-I - Brief Pain Inventory - Pain Interference subscale, HADS-D - Hospital Anxiety and Depression Scale - Depression subscale, HADS-A - Hospital Anxiety and Depression Scale - Anxiety subscale, PCL-C - PTSD Checklist - Civilian Version, PCS - Pain Catastrophizing Scale, SPTS-12 – 12-item Sensitivity to Pain Traumatization Scale.

[a] See text for a description of statistical tests conducted on these variables.

Table 4.9. Descriptive data for the Sensitivity to Pain Traumatization Scale (SPTS-12) trajectory groups for the final model two years (550-910 days) after surgery.

	Trajectory #1		Trajectory #2		Trajectory #3		Trajectory #4		Trajectory #5	
	n	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)
MME[a]	15	75.06 (137.33)	11	29.65 (52.42)	10	45.21 (36.18)	5	31.65 (37.02)	7	50.30 (49.19)
≤ 90 MME/day	12	-	10	-	9	-	5	-	5	-
> 90 MME/day	3	-	1	-	1	-	0	-	2	-
BPI-P[a]	17	5.15 (1.76)	10	5.36 (1.29)	12	5.86 (1.70)	6	4.89 (1.50)	8	5.87 (1.62)
BPI-I[a]	17	4.91 (1.90)	10	5.87 (1.26)	12	6.91 (1.21)	6	4.31 (1.50)	8	7.88 (1.62)
HADS-D[a]	17	6.15 (3.21)	9	10.33 (3.64)	12	12.26 (4.29)	6	5.57 (3.84)	8	11.23 (5.60)
HADS-A	17	6.51 (3.15)	9	10.50 (2.54)	12	10.81 (3.89)	6	7.78 (4.02)	8	13.17 (2.72)
PCL-C	5	12.80 (4.97)	1	17.00 (-)	0	-	0	-	1	20.00 (-)
PCS	16	13.62 (12.77)	9	28.22 (10.29)	11	25.58 (6.63)	6	11.29 (8.35)	8	40.42 (10.17)
SPTS-12	16	8.95 (3.22)	8	23.88 (5.87)	11	19.05 (4.89)	6	12.37 (4.26)	8	37.45 (5.83)

Note. MME – mg morphine equivalent, BPI-P - Brief Pain Inventory - Average Pain subscale, BPI-I - Brief Pain Inventory - Pain Interference subscale, HADS-D - Hospital Anxiety and Depression Scale - Depression subscale, HADS-A - Hospital Anxiety and Depression Scale - Anxiety subscale, PCL-C - PTSD Checklist - Civilian Version, PCS - Pain Catastrophizing Scale, SPTS-12 – 12-item Sensitivity to Pain Traumatization Scale.

[a] See text for a description of statistical tests conducted on these variables.

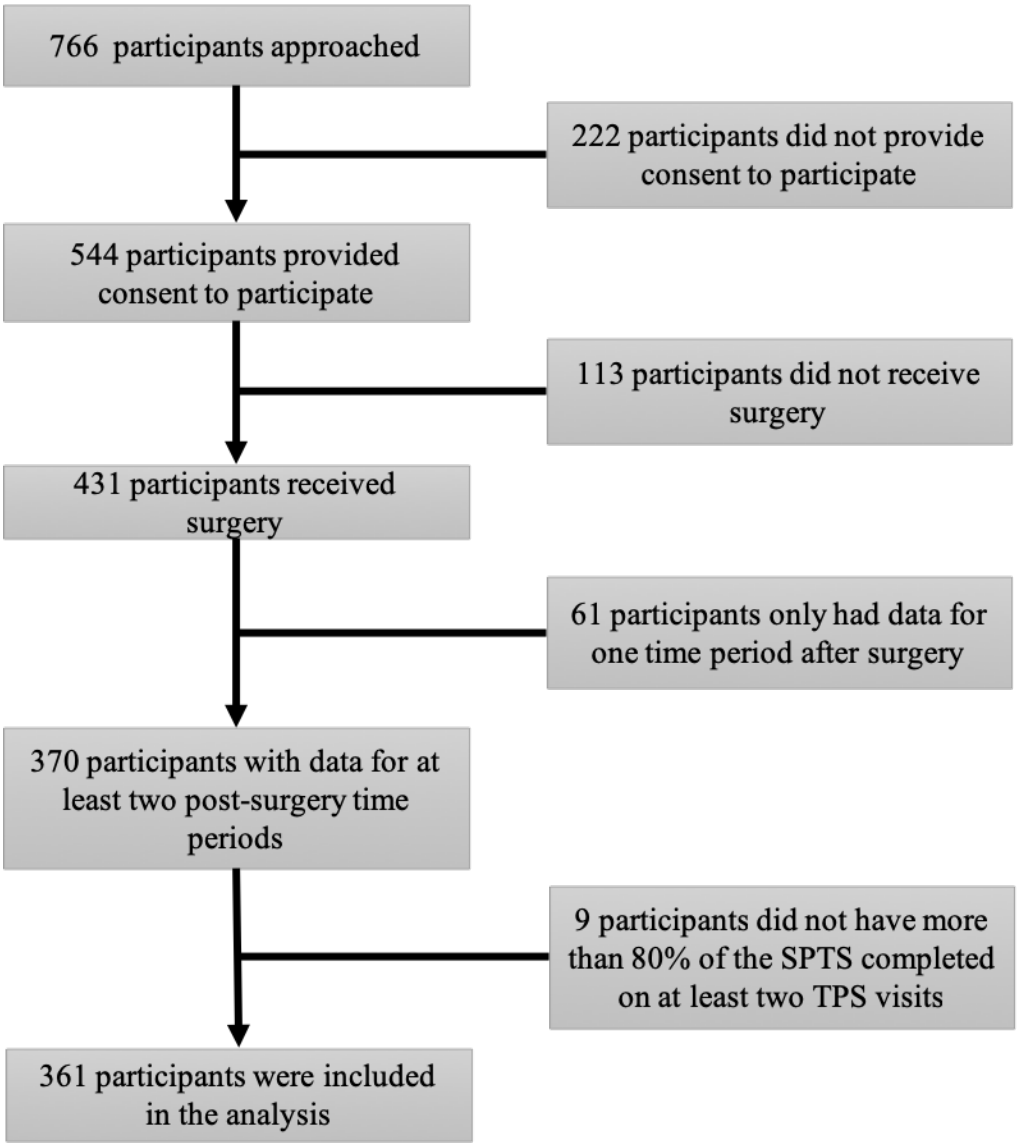

Figure 4.1. The recruitment process displaying the exclusion criteria for inclusion in the final sample.

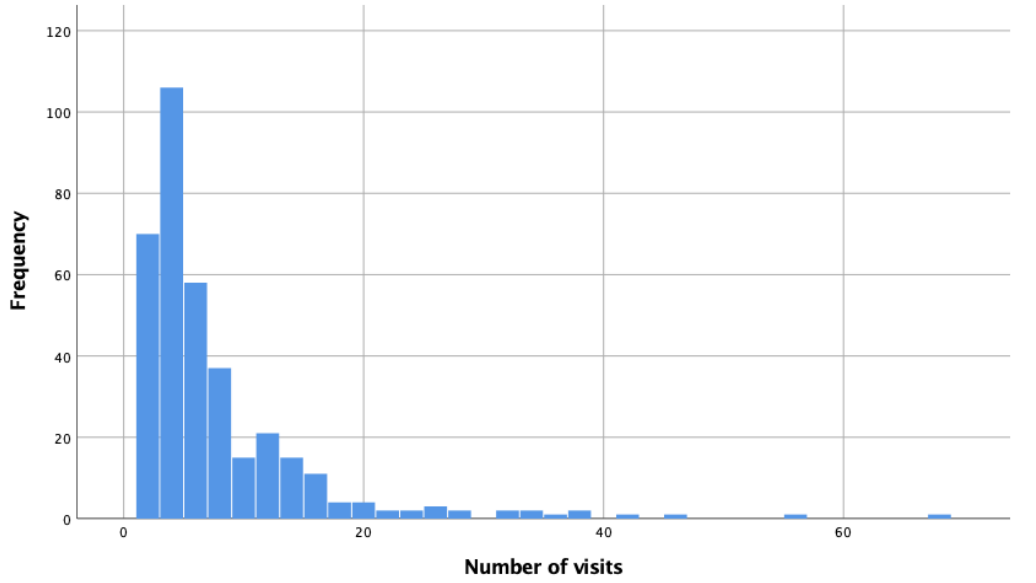

Figure 4.2. Histogram displaying the frequency of participant visits to the Transitional Pain Service (TPS) after surgery.

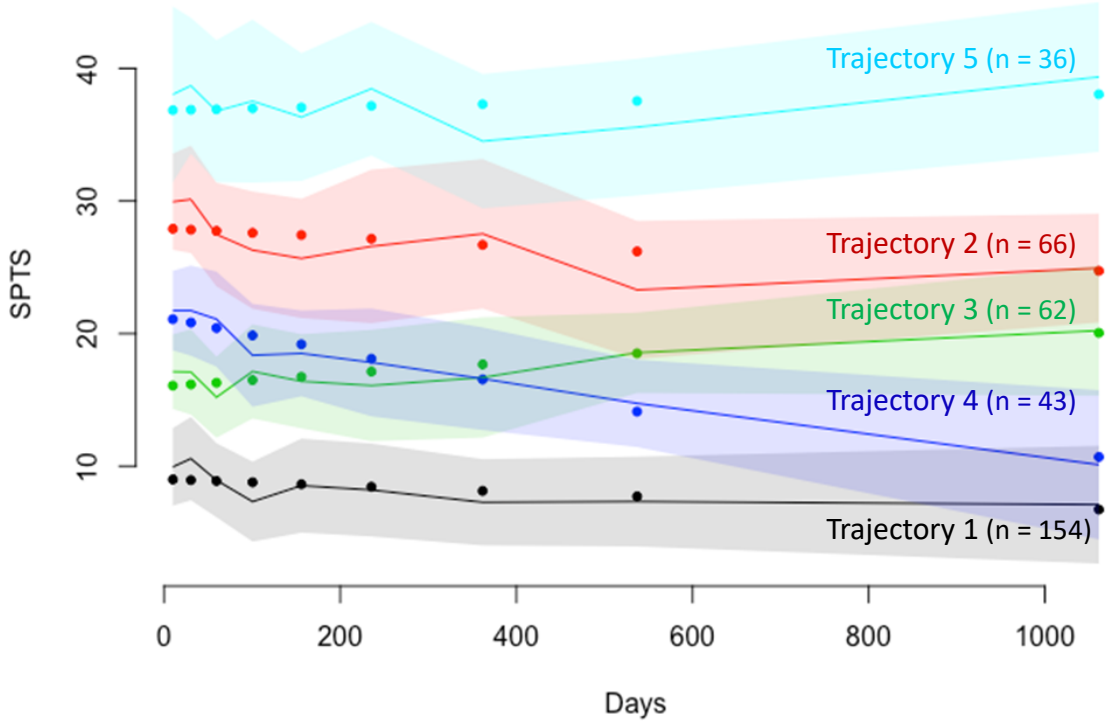

Figure 4.3. Trajectory groups of Sensitivity to Pain Traumatization Scale (SPTS-12) scores over days since surgery for the five-trajectory model. Predicted values are shown by the dots; observed values by the solid lines; and colored shading above and below each line shows the 95% confidence interval.

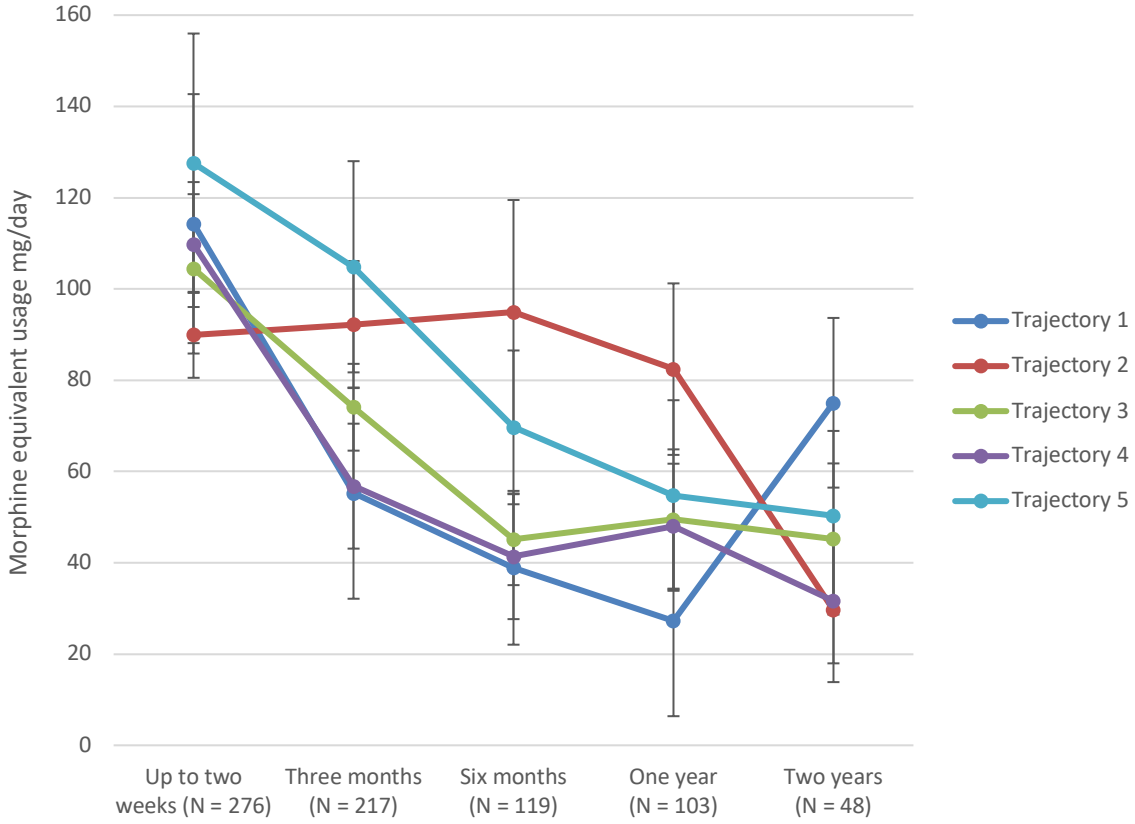

Figure 4.4. Reported mean rates of mg morphine equivalent usage per day over time after surgery for the five Sensitivity to Pain Traumatization Scale (SPTS-12) trajectory groups. Bars represent standard error of the mean.

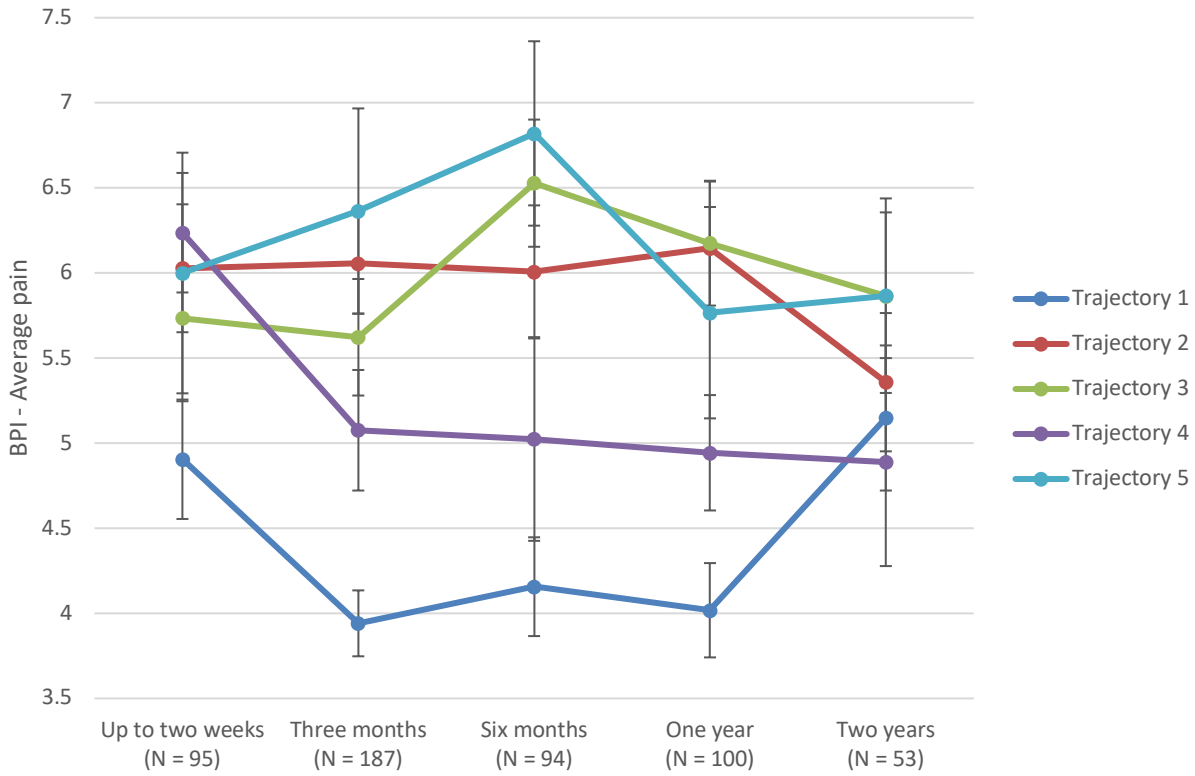

Figure 4.5. Reported mean pain ratings assessed with the Brief Pain Inventory (BPI) over time after surgery for the five Sensitivity to Pain Traumatization Scale (SPTS-12) trajectory groups. Bars represent standard error of the mean.

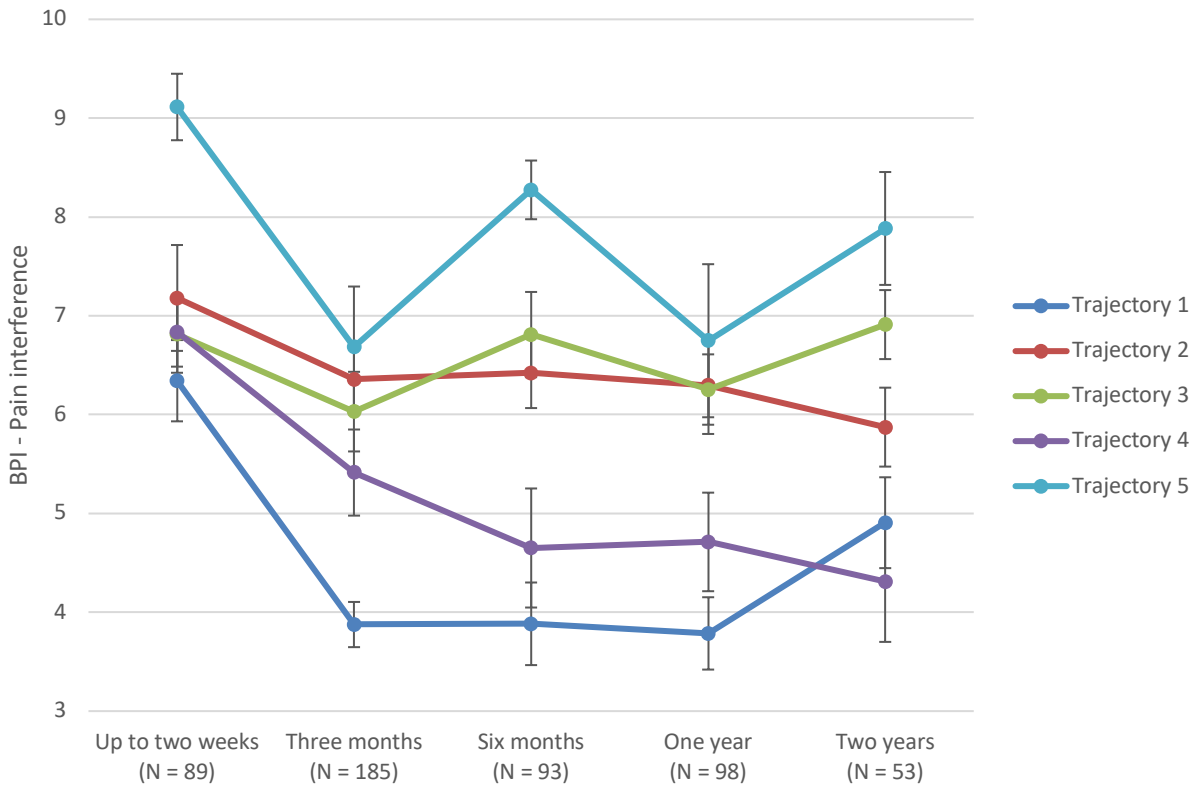

Figure 4.6. Reported mean pain interference ratings assessed with the Brief Pain Inventory (BPI) over time after surgery for the five Sensitivity to Pain Traumatization Scale (SPTS-12) trajectory groups. Bars represent standard error of the mean.

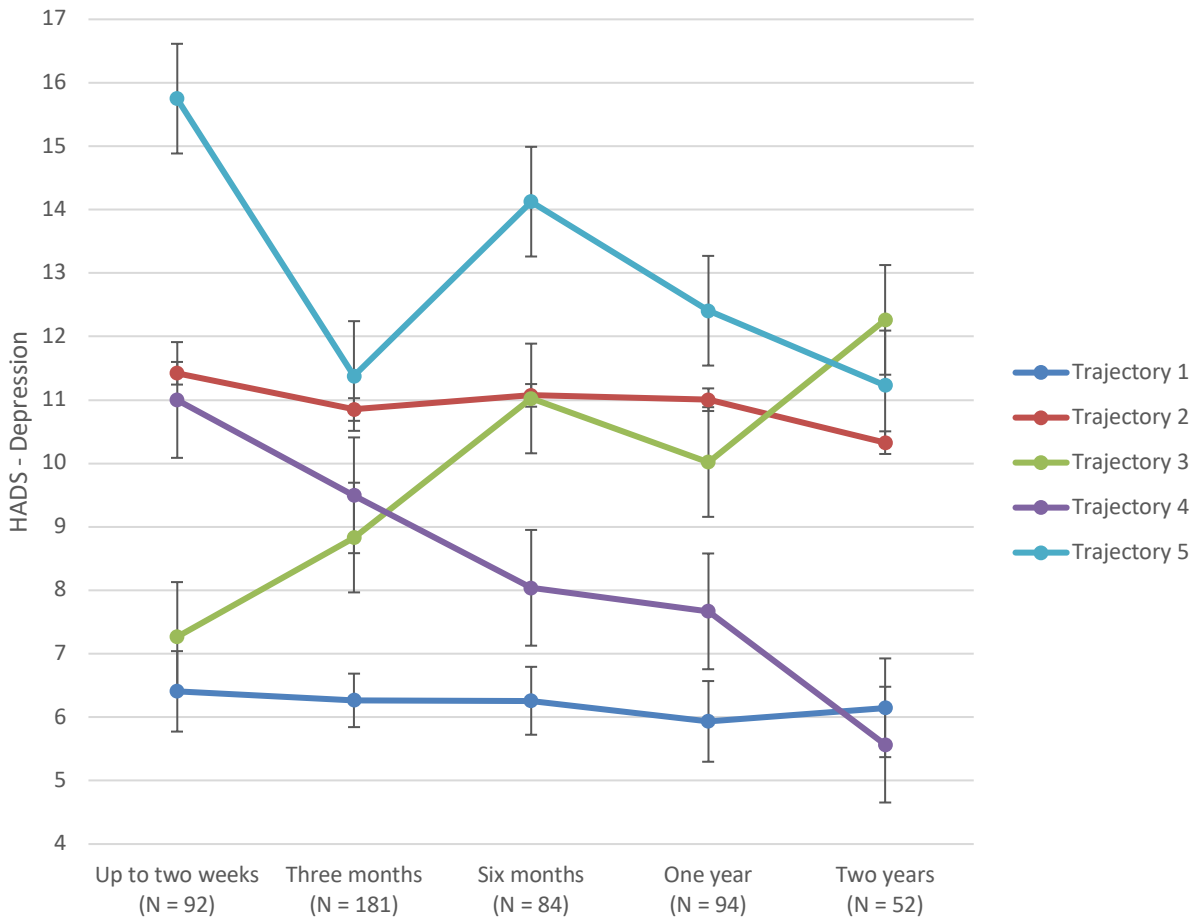

Figure 4.7. Reported mean depression ratings assessed with the Hospital Anxiety and Depression Scale (HADS) over time after surgery for the five Sensitivity to Pain Traumatization Scale (SPTS-12) trajectory groups. Bars represent standard error of the mean.

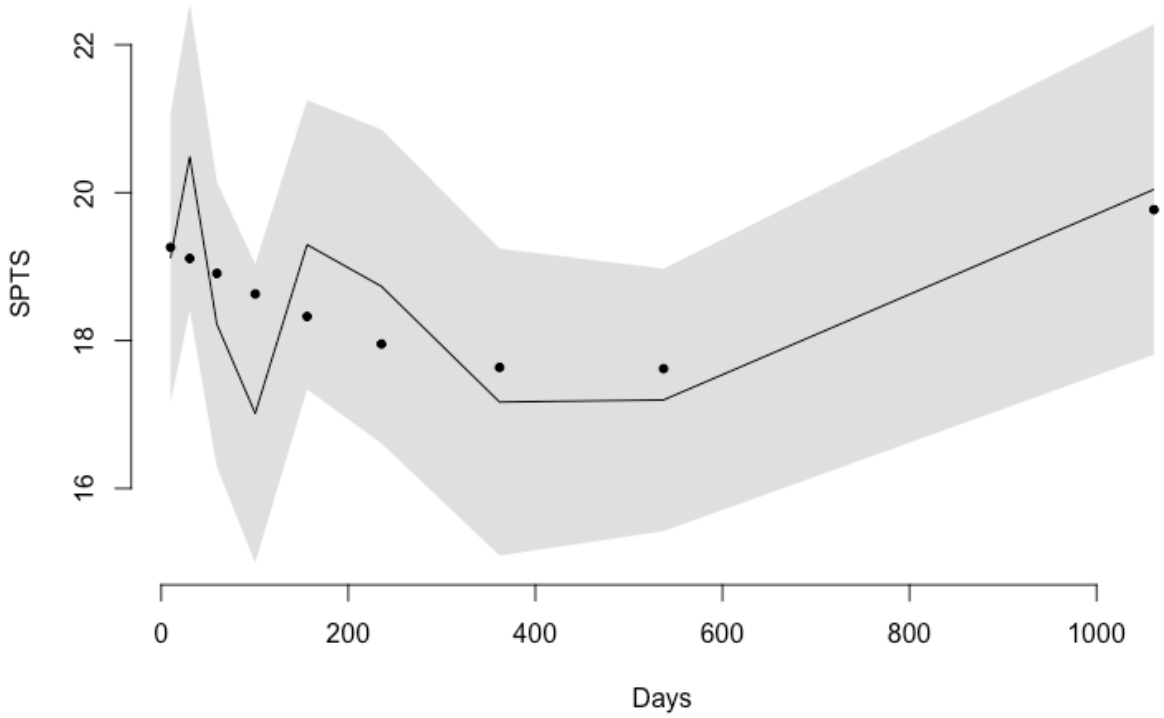

Figure 4.8. Trajectory of Sensitivity to Pain Traumatization Scale (SPTS-12) scores over days since surgery for the one-trajectory model, including the quadratic term. Predicted values are shown by the dots; observed values by the solid line; and the shading above and below the line shows the 95% confidence interval.

Chapter 5: General Discussion

Summary of Findings

The aim of the present dissertation was to examine the validity of scores on the 12-item Sensitivity to Pain Traumatization Scale (SPTS-12), a scale developed to assess the propensity of individuals to develop a traumatic stress response to pain. Chapter 1 started with a literature review providing an overview of pain and trauma. A definition of chronic pain was provided in consideration of its physiology, time course, and specific syndromes. The burden of pain, models of pain, and methods of measuring pain were then described. Next, trauma was defined and the burden of trauma was described. The substantial comorbidity of Posttraumatic Stress Disorder (PTSD) and chronic pain was highlighted, including a description of models that have been proposed to better understand this relationship. Current tools for assessing trauma and pain-related anxiety were described, including outlining the development of the SPTS-12.

Chapter 2 evaluated the SPTS-12 in a sample of 823 undergraduate students, some who reported ongoing pain (n = 268) and some who did not report pain (n = 555). A one-factor model adequately represented the internal dimensional structure of the SPTS-12 for both groups, with standardized factor loadings ranging from .46 to .78. The item with the poorest association with the common factor for both groups was item 9, “as soon as the pain comes on, I take medications to reduce it”. Even though the one-factor model demonstrated the best fit compared to two-factor, three-factor, and four-factor models, there may be other minor dimensions among the 12 items of the scale. Convergent validity was evaluated by examining the relationship between the SPTS-12 total score and the PTSD Checklist – Civilian Version (PCL-C). Divergent validity was evaluated by examining whether the relationship between SPTS-12 total score and the PCL-C is stronger than the relationship between the SPTS-12 total score and the Beck

Depression Inventory-II (BDI-II), a measure of a depression. Evidence of convergent and divergent validity was demonstrated among participants without pain. For participants with pain, the correlation between the SPTS-12 and the PCL-C did not significantly differ from the correlation between the SPTS-12 and the BDI-II. This result may be attributable to the high comorbidity rates between pain and depression, in that individuals who experience pain are more likely to report depressive symptoms (Fishbain et al., 1997). This comorbidity was supported by significantly higher rates of BDI-II scores among participants reporting pain than among those without pain. For this reason, it is possible that depression becomes more related to pain over time. Study 1 limitations were discussed, including the possibility that the breadth of symptoms assessed with the SPTS-12 may have contributed to a poorer overall fit for the factor analysis and the sample of participants reporting ongoing pain were variable according to the severity, duration, frequency, and functional impact of their physical symptoms. It was proposed that future research should consider evaluating the SPTS-12 in a variety of clinical and non-clinical populations and should consider modifying or using an alternative statement for item 9. It was suggested that it would be particularly important to evaluate the predictive validity of the SPTS-12. In conclusion, Study 1 provided support for the factor structure, reliability, and validity of the SPTS-12 in a community sample.

Chapter 3 examined the SPTS-12 in a clinical sample of 180 individuals receiving care from the Transitional Pain Service (TPS) at Toronto General Hospital. Confirmatory factor analyses were used to evaluate the factor structure of the SPTS-12, finding support for the one-factor model. This finding adds to the growing literature that pain traumatization is well-explained by a single factor, even though it was designed to assess six symptoms domains (i.e., pain and emotional numbing, pain and hyperarousal, pain avoidance, pain experiencing/intrusive

thoughts, sensitivity to pain, and fear of pain). The SPTS-12 demonstrated good convergent validity with a theoretically related measure of trauma as assessed with the PCL-C. However, divergent validity was not supported, as the SPTS-12 total score was not more strongly correlated with the PCL-C than it was with the depression subscale of the Hospital Anxiety and Depression Scale (HADS-D). Similar to Study 1, this finding may be due to the increasing comorbidity of depression and anxiety-related constructs among people with clinical pain conditions. Study 2 limitations included missing data and the use of self-report measures, which are less reliable than clinician-administered clinical interviews for assessing anxiety, depression, and PTSD. Furthermore, because Study 2 used a cross-sectional design, future research should use longitudinal designs to better understand how SPTS-12 scores and their associations with important outcomes (e.g., average pain and pain interference) change over time. Given that the sample was composed of a wide variety of clinical pain diagnoses, it would also be useful to consider whether the psychometric properties of the STPS-12 vary based on characteristics such as post-surgical pain, gender, or pain severity. It was concluded that Study 2 provided support for the factor structure, reliability, and convergent validity of SPTS-12 scores in a clinical sample.

Chapter 4 investigated the SPTS-12 in a clinical sample of individuals receiving care after surgery from the TPS at Toronto General Hospital. Latent class mixed modeling was used to represent how SPTS-12 scores change over time. The model with the best fit consisted of five SPTS-12 trajectory classes. Trajectory #1 consisted of the largest proportion of the sample and started with low SPTS-12 scores that significantly decreased over time. Trajectory #3 started with low SPTS-12 scores that remained stable over time, whereas Trajectory #4 showed the steepest decrease in SPTS-12 scores after surgery. Trajectories #2 and #5 started with high SPTS-12 scores that decreased significantly over time for Trajectory #2, but not for Trajectory

#5. Overall, the higher the SPTS-12 scores, the greater the endorsement of other clinical symptoms, such as depression, anxiety, trauma, and pain catastrophizing. The identification of five distinct SPTS-12 trajectory classes demonstrates that there is a high level of heterogeneity of post-surgery changes in pain-related trauma symptoms. Understanding these distinct patterns of scores across time post-surgery could potentially inform treatment recommendations to address the considerable psychological distress that accompanies a traumatic response to pain after surgery. Four outcome measures related to chronic pain were evaluated. Whereas SPTS-12 trajectory classes did not significantly differ regarding the daily dose of opioids used at any timepoint, average pain, pain interference, and depression scores did significantly differ across the trajectory classes. Study 3 limitations included missing data from participants not completing all questionnaire items and missing data through variable attendance to the TPS. Future research should evaluate the sources of missing data and determine how trajectories may differ based on sample characteristics (e.g., multiple complex diagnoses, different surgeries). Study 3 supported the ability to distinguish between important pain-related outcomes over time.

Taken together, these three studies add to the psychometric validation of the STPS-12 in both community and clinical samples. Exploratory and confirmatory factor analysis demonstrated that a one-factor model is a good representation of the SPTS-12 items. Furthermore, SPTS-12 scores have excellent internal reliability and good convergent validity with the PCL-C. Support for discriminant validity was questionable, given that it was determined with the relative relationship strength with a measure of depression that may not be a sufficiently disparate measure of reference. Finally, heterogeneity of the long-term trajectories of SPTS-12 scores over time post-surgery was demonstrated using latent class mixed models.

Clinical Implications

The present research helped validate the SPTS-12, a new questionnaire assessing traumatic stress-related symptoms that may be experienced in response to pain. Until the development of the SPTS-12, only questionnaires assessing related constructs, such as pain-related anxiety or symptoms of trauma, were available. This development has important implications for theory and understanding of pain-related trauma and the SPTS-12's potential utility in detecting individuals at a higher risk of developing chronic postsurgical pain.

The SPTS-12 provides a way to more directly measure traumatization that individuals may experience in response to pain, which may contribute to our understanding of why trauma and pain co-occur so frequently: individuals with a diagnosis of PTSD are more likely to experience pain and pain-related disability than individuals without a diagnosis of PTSD. Furthermore, individuals with pain are more likely to experience PTSD than individuals without pain. It has even been posited that the number of traumatic exposures an individual experiences has a cumulative effect to the point where a greater number of lifetime exposures predicts the presence of chronic medical conditions (Sledjeski, Speisman, & Dierker, 2008). Several models have been proposed to explain this relationship, including that the symptoms of both pain and anxiety maintain each other and that pre-existing dispositions make certain individuals more vulnerable to both pain and anxiety (Asmundson et al., 2002; Katz et al., 2014; Keane & Barlow, 2002; Liedl & Knaevelsrud, 2008; Rosenbloom et al., 2013; Sharp & Harvey, 2001; Turk, 2002). Through validating the SPTS-12, empirical support is provided for the idea that pain acts as a higher-order factor across trauma symptoms when it is present (Pagé et al., 2009). This effect was supported by the finding that in both a community sample of undergraduate students and a

clinical sample of individuals receiving treatment in the TPS, a one-factor SPTS-12 model was supported.

Given that 10 to 50% of surgery patients go on to develop chronic postsurgical pain (Kehlet et al., 2006), developing pain interventions to reduce the high incidence is warranted. Using a more precise measurement tool to identify individuals experiencing a greater traumatic response to pain after surgery permits the opportunity to tailor postsurgical treatment in a way that more broadly captures biopsychosocial contributors of pain. To best address symptoms of pain and trauma, an integrated care model is recommended that considers the unique and complex needs of each individual (Friedman, Lowry, & Ruzek, 2010). It has been suggested that effective treatment begin with psychoeducation, cognitive behavioural therapies, and relaxation techniques, which can be followed with more trauma-specific treatment given a positive response (Wald, Taylor, & Fedoroff, 2004). More broadly, there is evidence for the efficacy of psychotherapy postoperatively (Nicholls et al., 2018; Weinrib et al., 2017). Wang et al. (2018) conducted a meta-analysis of randomized controlled trials that evaluated the efficacy of perioperative psychotherapy for postsurgical pain and impairment. Across 15 studies, they found that cognitive behaviour therapy or relaxation therapy reduced both pain and physical impairment. Furthermore, in previous research in the TPS designed to assess the efficacy of the program, patients benefitted from psychotherapy interventions: Azam et al. (2017) found that individuals who received psychotherapy after surgery demonstrated greater reductions in opioid use, pain interference, and depression than those who did not receive psychotherapy. Given that the SPTS-12 can predict important pain-related outcomes such as average pain, pain interference, and depression, it is possible that tailoring psychotherapeutic interventions to target pain-related trauma symptoms may increase the efficacy of pain management following surgery.

By establishing the psychometric characteristics of the SPTS-12, we are better able to understand the theories explaining the high comorbidity of pain and trauma. Furthermore, given the high incidence rates of pain and trauma as well as the established efficacy of psychotherapy in treating pain after surgery, tailoring treatment to address trauma-specific symptoms may help improve pain management treatment strategies.

Limitations

The present research has several limitations, including the following: (1) The exploratory factor analysis of the SPTS-12 in Study 1 identified several large residual correlations among some items; (2) in each of the three studies, the samples were heterogenous and there may have been potentially important differences in participants according to diagnosis, pain, or functional disability; and (3) missing data may have contributed to a systematic bias in the results, especially for Studies 2 and 3. Each of these limitations is described in more detail below.

First, the exploratory factor analysis in Study 1 demonstrated that the one-factor model had a strong fit for the SPTS-12, but there were still several large residual correlations among items both for individuals who reported pain and for those who did not. This finding may indicate that other, minor dimensions may be present in the SPTS-12, which would benefit from exploration in future studies. It is possible that these residual correlations are due to the wide breadth of symptoms assessed with the SPTS-12. The SPTS-12 was designed to include two items in six different categories that capture various cognitive, emotional, behavioural, and somatic domains of a traumatic response to pain: pain and emotional numbing, pain and hyperarousal, pain avoidance, pain experiencing/intrusive thoughts, sensitivity to pain, and fear of pain. It is possible that the diverse symptoms assessed led to stronger correlations among items from the same domain than could be explained by the single factor.

Second, in all three studies, the participants were highly heterogenous. For example, in Study 1, there were considerable differences reported in the severity, duration, frequency, and functional impact of pain symptoms. Among participants who reported experiencing pain on an ongoing basis, the majority (66%) indicated that they experienced no or only slight pain-related interference whereas the rest of the sample who indicated moderate or severe pain-related interference. Additionally, 16.4% of participants reported that they experienced pain monthly while 38.1% endorsed experiencing pain daily. It is possible that a subset of participants whose symptoms are more severe might exhibit different psychometric properties for the SPTS-12. This difference was apparent in the method used to assess divergent validity in Study 1, whereby the correlation between the SPTS-12 and PCL-C was significantly greater than the correlation between the SPTS-12 and the BDI-II only among individuals reporting no pain. In Studies 2 and 3, the samples were similarly diverse. Preoperative diagnoses included cancer, chronic pain, gastroesophageal reflux disease, hypertension, arthritis, and diabetes mellitus. For those undergoing surgery, surgery type included thoracic, transplant, general, cardiac, and ear, nose, and throat. Given that some conditions and procedures are associated with more pain and pain interference (e.g., an estimated 47% of thoracic surgery patients report chronic postsurgical pain compared to an estimated 10% to 15% of modified radical mastectomy patients; Bayman & Brennan, 2014; Tasmuth et al., 1999), it is similarly possible that this heterogeneity could impact the factor structure, reliability, and validity of the SPTS-12. Conversely, however, it is important to note that this same heterogeneity may also help demonstrate the universality of pain traumatization symptoms across diverse patients, given that the psychometric properties were still strong given the substantial heterogeneity.

Third, across all three studies, not all questionnaires were filled out by all participants at every timepoint. In Study 1, 4.3% of the sample was excluded due to non-response on the SPTS-12 or failure to indicate if they are currently experiencing ongoing pain. In Study 2, 17.8% of the sample was excluded for non-response on the SPTS-12. In Study 3, although only 2.4% of the sample was excluded from analysis because they did not complete at least 80% of the SPTS-12, most of the sample (75.3%) had not completed at least 80% of the other questionnaires included in the package. In this case, only 89 participants had complete data for all questionnaires, meaning that the final SPTS-12 trajectory model was unable to include predictors of trajectory class membership, including depression, anxiety, and pain catastrophizing. It is possible that individuals who completed the questionnaire packages differed from those who did not in important ways. The greater prevalence of missing data in Study 2 and Study 3 is not surprising, given that missing data in clinical research is a common challenge, especially in longitudinal research (Little et al., 2012; Turk et al., 1993). Across all three studies, data may have been missing due to both participant characteristics (including beliefs about the research topic) and study design characteristics (e.g., the format of the questionnaire, the length of questionnaire; McKnight et al., 2007). For example, the lower rate of missing data in Study 1 may be due to the sample consisting of undergraduate students who completed the study to earn course credits. As a result, they might have felt more obligated to complete all items than participants in Studies 2 and 3 who did not receive any type of compensation for participating. Furthermore, the questionnaire package for Study 1 was completed online, making it more difficult to accidentally miss questions. For Study 2 and Study 3, the questionnaires were provided on paper to participants prior to their appointments. In this case, it is possible that short waiting times might have contributed to a failure to finish filling them out. Overall, even though missing data is a

common challenge in clinical research, it may have introduced a source of bias between individuals with and without missing data.

Future Directions

The present research provides further validation of the SPTS-12 as a measure of the cognitive, emotional, behavioural, and somatic responses to pain that are similar to a traumatic stress response. Future research would benefit from addressing the current findings in several ways, including the following: (1) using a different statement to replace item 9, "as soon as the pain comes on, I take medications to reduce it", (2) evaluating the concurrent validity of the SPTS-12 by examining its relationship with clinically relevant mental health diagnoses, and (3) validating the SPTS-12 in different patient populations. Each of these future directions is described in more detail below.

First, in Study 1, item 9 ("as soon as the pain comes on, I take medications to reduce it") had the poorest fit in the one-factor model of all twelve items of the SPTS-12. This item was included to represent avoidance, which is an important theoretical and behavioural characteristic of a traumatic response. In the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013), one of the four symptom clusters required for a diagnosis of PTSD is avoidance, and avoidance is similarly important as a maintenance factor in models of persistent pain (e.g., Vlaeyen & Linton, 2000). However, taking medications when pain starts may not always be an avoidant response: proper medical management of pain could also be considered as a form of active coping (Van Damme, Crombez, & Eccleston, 2008). Considering the relatively poor fit of item 9 and the possibility that it may also capture adaptive coping, future research should consider developing a new item that better captures the manner in which avoidance is relevant to the construct of sensitivity to

pain traumatization. A new item could be developed from an established, validated source of pain behaviours such as the Patient-Reported Outcome Measurement Information System (PROMIS; Revicki et al., 2009). Possible items may include "when I am in pain I try to stay very still" (p. 164) or "when I am in pain I avoid physical contact with others" (p. 165). Another option might be to modify the wording of the DSM-5 diagnostic criteria for PTSD to reflect the experience of traumatic avoidance in response to pain. For example, to target avoidance of memories, thoughts, or feelings regarding the traumatic event, potential wording may be similar to the following: “When I am in pain, I try to avoid memories, thoughts, or feelings related to my pain”. Establishing a new item that demonstrates a stronger association with the theoretical concept of sensitivity to pain traumatization may improve the overall validity and reliability of the SPTS-12.

Second, all three studies used self-report measures to assess psychological symptoms of anxiety, mood, and trauma. While self-report measures are useful for screening for mental health concerns, they are typically not used as a diagnostic tool. Instead, as with the Hospital Anxiety and Depression Scale (HADS), they are designed to provide an estimate of the severity of symptoms rather than to predict a diagnosis of a given condition (Axford et al., 2010; Goldberg, 1985). In developing the SPTS-12, it would be useful to consider how SPTS-12 scores are related to the clinical diagnosis of relevant conditions such as major depressive disorder, generalized anxiety disorder, and PTSD. This could provide an important source of criterion validity as well as speaking to the clinical utility of the SPTS-12.

Third, the present studies have only evaluated the psychometric properties of the SPTS-12 in two samples, with previous research also examining a sample of 345 participants at least six months after coronary artery bypass graft surgery (Katz et al., 2017). The sample used in

Study 1 consisted of 823 participants enrolled in a psychology course at York University, with 32.56% of the sample endorsing that they experienced pain on an ongoing basis. The samples used in Studies 2 and 3 were receiving care from the TPS at Toronto General Hospital. As a clinical service, inclusion criteria to be admitted to the TPS is broad, and therefore participants included in the studies varied greatly with respect to medical diagnoses. For example, in Study 2, preoperative diagnoses included cancer, chronic pain, gastroesophageal reflux disease, hypertension, arthritis, and diabetes mellitus. Most participants also received surgery, which was similarly diverse. For example, in Study 3, the most common surgery of those included in the final analysis included thoracic, transplant, general, cardiac, ear, nose, and throat, vascular, and obstetrical/gynecological. Given that these samples are highly heterogenous, future research would benefit from examining if there are any differences in the psychometric properties of the SPTS-12 according to pain severity, medical diagnosis, or surgery type.

Overall, as the SPTS-12 is a new questionnaire, further research is required to continue to validate its psychometric properties and clinical utility. Future studies should consider the following: using alternative wording for item #9 to see if this might improve the overall model fit, examining the concurrent validity of the SPTS-12 and findings from semi-structured clinical interviews on related psychological disorders, and validating the SPTS-12 in more homogenous community and patient populations.

Conclusion

The aim of the present dissertation was to examine the validity of the SPTS-12, a scale developed to assess the propensity of individuals to develop a traumatic stress response to pain. First, a literature review provided an overview of pain and trauma, including a discussion of current assessment tools. Next, three studies were described. Study 1 evaluated the SPTS-12 in a

community sample of 823 undergraduate students, some of whom reported experiencing ongoing pain (n = 268) and some who did not endorse experiencing pain (n = 555). A single factor model adequately represented the internal dimensional structure of the SPTS-12 for both groups. Evidence of convergent and divergent validity was demonstrated. Study 2 examined the SPTS-12 in a clinical sample of 180 individuals receiving care in the TPS. Confirmatory factor analysis was used to evaluate the factor structure of the SPTS-12, finding support for the one-factor model. The SPTS-12 demonstrated good convergent validity with a theoretically related measure of trauma. However, divergent validity with a measure of depression was not supported. Study 3 also investigated the SPTS-12 in a clinical sample of individuals receiving care from the TPS. Latent class mixed modeling was used to examine how SPTS-12 scores change over time. The model with the best fit consisted of five SPTS-12 trajectory classes. Each of the five classes were described, with the overall observation that the higher the SPTS-12 scores, the greater the endorsement of other clinical symptoms, such as depression, anxiety, trauma, and pain catastrophizing. Four outcome measures related to chronic pain were evaluated. While SPTS-12 trajectory classes did not significantly differ on morphine equivalent use per day at any timepoint, average pain, pain interference, and depression scores did differ significantly at two or more postsurgical visits. Overall, the literature review and three studies add to the psychometric validation of the SPTS-12 in both community and clinical samples.

Appendix A. Research Ethics Board Approval Letters for Study 1

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OFFICE OF RESEARCH ETHICS (ORE)
Fifth Floor, YRT

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Toronto ON
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Tel 416 736 5914
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www.research.yorku.ca

AMENDMENT

Certificate #: 2009 - 008

2nd Renewal Approved: 01/12/12
Renewal Approved: 01/14/11
5th Amendment Approved: 11/06/12
4th Amendment Approved: 11/03/09
3rd Amendment Approved: 10/21/09
2nd Amendment Approved: 10/09/09
Amendment Approved: 01/26/09

Approval Period: 01/12/12-01/12/13

Memo

To: Professor Joel Katz, Faculty of Health,
Ms. Kaley Roosen, Faculty of Health

From: Alison M. Collins-Mrakas, Sr. Manager and Policy Advisor, Research Ethics

Date: **Tuesday 6th November, 2012**

Re: Ethics Approval

Development of a Sensitivity to Pain Traumatization Scale in Sample of Undergraduate Students

---

With respect to your research project entitled, "Development of a Sensitivity to Pain Traumatization Scale in Sample of Undergraduate Students", the committee notes that, as there are no substantive changes to either the methodology employed or the risks to participants in the research project or any other aspect of the project, a renewal of approval re the amendment to the above project is granted.

Yours sincerely,

Alison M. Collins-Mrakas M.Sc., LLM
Sr.Manager & Policy Advisor, Research Ethics

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Certificate #:	**2013 - 018**
Approval Period:	**01/31/13-01/31/14**

Memo

To: Professor Joel Katz, Faculty of Health

From: Alison M. Collins-Mrakas, Sr. Manager and Policy Advisor, Research Ethics
(on behalf of Duff Waring, Chair, Human Participants Review Committee)

Date: **Thursday 31st January, 2013**

Re: Ethics Approval

Development of a Sensitivity to Pain Traumatization Scale in a Sample of Undergraduate Students

---

I am writing to inform you that the Human Participants Review Sub-Committee has reviewed and approved the above project.

Yours sincerely,

Alison M. Collins-Mrakas M.Sc., LLM
Sr. Manager and Policy Advisor,
Office of Research Ethics

Appendix B. Syntax for the Statistical Analysis in Study 1

Syntax for the statistical analysis in R.

```
#Examining the factor structure for for participants with no pain
View(nopain)
library(psych)
library(car)

#Review the data for normality, distribution, and correlations
describe(nopain)
scatterplotMatrix(nopain, nopain, smooth=F, reg.line=F, diag="hist")
cor(nopain, use="pairwise.complete.obs")
polychoric(nopain)

# Examine the factor models to determine how many factors to retain using OLS and polychoric correlations.
oneFmod <- fa(nopain, nfactors=1, residuals=T, cor="poly")
oneFmod
oneFmod$values
plot(oneFmod$values, type="b", ylab='eigenvalues of reduced correlation matrix', xlab='Factor Number')
fa.parallel(nopain, fa='fa', show.legend=F)
oneFmod$rms
oneFmod$residual
omega(nopain,nfactors=1)

#Examining the factor structure for for participants with pain
View(pain)
library(psych)
library(car)

#Review the data for normality, distribution, and correlations
describe(pain)
scatterplotMatrix(pain, pain, smooth=F, reg.line=F, diag="hist")
cor(pain, use="pairwise.complete.obs")
polychoric(pain)

# Examine the factor models to determine how many factors to retain using OLS and polychoric correlations.
oneFmodpain <- fa(pain, nfactors=1, residuals=T, cor=“poly”)
oneFmodpain
oneFmodpain$values
plot(oneFmodpain$values, type="b", ylab='eigenvalues of reduced correlation matrix', xlab='Factor Number')
fa.parallel(pain, fa='fa', show.legend=F)
```

```
oneFmodpain$rms
oneFmodpain$residual
omega(pain,nfactors=1)
```

Syntax for the statistical analysis in SPSS

```
USE ALL.
COMPUTE filter_$=(Missing_Data = 0).
VARIABLE LABELS filter_$ 'Missing_Data = 0 (FILTER)'.
VALUE LABELS filter_$ 0 'Not Selected' 1 'Selected'.
FORMATS filter_$ (f1.0).
FILTER BY filter_$.
EXECUTE.

T-TEST GROUPS=PAIN(0 1)
  /MISSING=ANALYSIS
  /VARIABLES=ASI_TOTAL_N BDI_TOTAL_N FPQ_TOTAL_N ISI_TOTAL_N
PASS_TOTAL_N PCLC_TOTAL_N PCS_TOTAL_N
    SPTS_12_TOTAL_N STAI_TOTAL_N
  /CRITERIA=CI(.95).
```

Appendix C: Research Ethics Board Approval Letters for Studies 2 & 3

YORK
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Fax 416 736-5512
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ETHICS APPROVAL
Hospital/ Clinical/ Medical Facilities

To: **Dr. Hance Clarke – Principal Investigator**
Toronto General Hospital

Professor Joel Katz
Department of Psychology, Faculty of Health

From: Alison M. Collins-Mrakas, Sr. Manager and Policy Advisor, Research Ethics
(on behalf of Denise Henriques, Chair, Human Participants Review Committee)

Date: Friday, February 24, 2017

Title: **Database Project: Transitional Pain Service**

Risk Level: ☒ Minimal Risk ☐ More than Minimal Risk

Level of Review: ☒ Delegated Review ☐ Full Committee Review

I am writing to inform you that the Human Participants Review Sub-Committee has received a copy of your hospital/ medical facility ethics <renewal/ amendment> approval certificate.

Further ethics review is not required by the York University Research Ethics Board as the research is minimal-risk and had undergone a delegated review by the respective hospital/ medical REB. If there are any changes to the risk or review level, you are required to notify the Office of Research Ethics as a full review will be required by the York University Research Ethics Board.

Name of Institution:	University Health Network
Principal Investigator:	Hance Clarke Joel Katz – Co-Investigator
Research Title:	Database Project: Transitional Pain Service
REB approval cert. #:	UHN approval: 14-7705-AE
Approval Period:	Originally approved: 02/23/14
Renewals:	06/23/16-06/23/17
Amendments:	

Please note that you are required for forward renewal and amendment certificates for our records.

Yours sincerely,

Alison M. Collins-Mrakas M.Sc., LLM
Sr. Manager and Policy Advisor,
Office of Research Ethics

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ETHICS APPROVAL
Hospital/ Clinical/ Medical Facilities

To: **Professor Hance Clarke & Joel Katz (co: PI)**
Department of Psychology, Faculty of Health

From: Alison M. Collins-Mrakas, Sr. Manager and Policy Advisor, Research Ethics
(on behalf of Denise Henriques, Chair, Human Participants Review Committee)

Date: October 15th, 2018

Title: **Database Project: Transitional Pain Service**

Risk Level: ☒ Minimal Risk ☐ More than Minimal Risk

Level of Review: ☒ Delegated Review ☐ Full Committee Review

I am writing to inform you that the Human Participants Review Sub-Committee has received a copy of your hospital/ medical facility ethics <renewal/ amendment> approval certificate.

Further ethics review is not required by the York University Research Ethics Board as the research is minimal-risk and had undergone a delegated review by the respective hospital/ medical REB. If there are any changes to the risk or review level, you are required to notify the Office of Research Ethics as a full review will be required by the York University Research Ethics Board.

Name of Institution:	University Health Network
Principal Investigator:	Hance Clarke Joel Katz (co: PI)
Research Title:	Database Project: Transitional Pain Service
REB approval cert. #:	UHN approval: 14-7705-AE
Approval Period:	08/04/18-08/04/19
Renewals:	06/23/17-06/23/18; 08/04/18
Amendments:	06/23/17-06/23/18

Please note that you are required for forward renewal and amendment certificates for our records.

Yours sincerely,

Alison M. Collins-Mrakas M.Sc., LLM
Sr. Manager and Policy Advisor,
Office of Research Ethics

UHN Toronto General Toronto Western Princess Margaret Toronto Rehab

University Health Network
Research Ethics Board
10th Floor, Room 1056
700 University Ave.
Toronto, Ontario, M5G 1Z5
Phone: (416) 581-7849

NOTIFICATION OF REB RENEWAL APPROVAL

Date: July 26, 2018

To: Hance Clarke

Re: 16-5109
Transitional pain service: Reducing heath care costs and improving opioid safety for complex chronic pain patients following hospital discharge

REB Review Type:	Delegated
REB Initial Approval Date:	August 4, 2016
REB Renewal Approval Effective Date:	August 4, 2018
Lapse In REB Approval:	N/A
REB Expiry Date:	August 4, 2019

The University Health Network Research Ethics Board has reviewed and approved the Renewal (16-5109.6) for the above mentioned study.

Best wishes on the successful completion of your project.

Sincerely,
Meenal Mistry
Ethics Coordinator, University Health Network Research Ethics Board

For: Morris Sherman
Co-Chair, University Health Network Research Ethics Board

The UHN Research Ethics Board operates in compliance with the Tri-Council Policy Statement; ICH Guideline for Good Clinical Practice E6(R1); Ontario Personal Health Information Protection Act (2004); Part C Division 5 of the Food and Drug Regulations; Part 4 of the Natural Health Products Regulations and the Medical Devices Regulations of Health Canada.

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Notification of REB Continued Approval

Date: June 13th, 2016
To: Dr. Hance Clarke

Re: 14-7705-AE
Database Project: Transitional Pain Service

REB Review Type:	Expedited
REB Initial Approval Date:	June 23rd, 2014
REB Annual Approval Date:	June 23rd, 2016
REB Expiry Date:	June 23rd, 2017

The UHN Research Ethics Board operates in compliance with the Tri-Council Policy Statement; ICH Guideline for Good Clinical Practice E6(R1); Ontario Personal Health Information Protection Act (2004); Part C Division 5 of the Food and Drug Regulations; Part 4 of the Natural Health Products Regulations and the Medical Devices Regulations of Health Canada. The approval and the views of the REB have been documented in writing. The REB has reviewed and approved the clinical trial protocol and informed consent form for the trial which is to be conducted by the qualified investigator named in the letter.

Furthermore, members of the Research Ethics Board who are named as Investigators in research studies do not participate in discussions related to, nor vote on such studies when they are presented to the REB.

Best wishes on the successful completion of your project.

Sincerely,

Meénal Mistry, BSc
Research Ethics Coordinator

For: Alan Barolet, MD PhD FRCPC
Co-Chair, University Health Network Research Ethics Board

Appendix D. Syntax for the Statistical Analysis in Study 2

Syntax for the statistical analysis in R.

```
View(tps)
library("lavaan")
library("psych")
library("semTools")

# Review the data for normality, distribution, and correlations.
describe(tps)
summary(tps) #provides the item stats on the 12 variables identified
summary(as.factor(is.na(tps))) #This provides information if there is any missing data for
("FALSE" means no)

#Create a 9 x 9 matrix of all bivariate scatter plots to examine. The diagonal is a histogram of
each observed variable.
scatterplotMatrix(tps, tps, smooth=F, reg.line=F, diag="hist")

#Calculate the correlations among the variables.
cor(tps, use="pairwise.complete.obs")
polychoric(tps)

#Specify the model for the one-factor solution and look at the results
spts1f <- 'spts =~
SPTS_1+SPTS_2+SPTS_3+SPTS_4+SPTS_5+SPTS_6+SPTS_7+SPTS_8+SPTS_9+SPTS_10+
SPTS_11+SPTS_12'
oneFresults <- cfa(model=spts1f, data=tps, estimator="ULSMV", std.lv=T,
ordered=c('SPTS_1','SPTS_2','SPTS_3','SPTS_4','SPTS_5','SPTS_6','SPTS_7','SPTS_8','SPTS_9
','SPTS_10','SPTS_11','SPTS_12'))
summary(oneFresults, standardized=T, fit.measures=T)

#Specify the three-factor solution and look at the results
spts3f <- 'F1 =~ SPTS_6+SPTS_9+SPTS_10+SPTS_5+SPTS_7+SPTS_8
F2 =~ SPTS_12+SPTS_4+SPTS_11
F3 =~ SPTS_1+SPTS_2+SPTS_3'
threeFresults <- cfa(model=spts3f, data=tps, estimator="ULSMV", std.lv=T,
ordered=c('SPTS_1','SPTS_2','SPTS_3','SPTS_4','SPTS_5','SPTS_6','SPTS_7','SPTS_8','SPTS_9
','SPTS_10','SPTS_11','SPTS_12'))
summary(threeFresults, standardized=T, fit.measures=T)

#Calculate the reliability estimates
library(semTools)
reliability(oneFresults)
alpha(tps) #Can evaluate the internal consistency by calculating coefficient alpha
```

Syntax for the statistical analysis in SPSS

```
CORRELATIONS
  /VARIABLES=SPTS_Total BPI_Score HADS_Anx HADS_Dep HADS_Total IEQ_Score
PCL_Score PCS_Total
    SFMPQ2_Total SOAPP_Score
  /PRINT=TWOTAIL NOSIG
  /MISSING=PAIRWISE.
NONPAR CORR
  /VARIABLES=SPTS_Total BPI_Score HADS_Anx HADS_Dep HADS_Total IEQ_Score
PCL_Score PCS_Total
    SFMPQ2_Total SOAPP_Score
  /PRINT=SPEARMAN TWOTAIL NOSIG
  /MISSING=PAIRWISE.
```

Appendix E. Syntax for the Statistical Analysis in Study 3

Syntax for the statistical analysis in R.

```
library(lcmm)
library(foreign)
library(haven)
View(merged)

#Running the growth mixture models for STPS over time (days)
m1SPTS <- hlme(SPTS~days, subject ='id', ng=1,idiag=T, data=merged)
summary(m1SPTS)
m2SPTS <- hlme(SPTS~days, subject ='id', ng=2, mixture=~days, idiag=T, data=merged)
summary(m2SPTS)
m3SPTS <- hlme(SPTS~days, subject ='id', ng=3, mixture=~days, idiag=T, data=merged)
summary(m3SPTS)
m4SPTS <- hlme(SPTS~days, subject ='id', ng=4, mixture=~days, idiag=T, data=merged)
summary(m4SPTS)
m5SPTS <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T, data=merged)
summary(m5SPTS)
m6SPTS <- hlme(SPTS~days, subject ='id', ng=6, mixture=~days, idiag=T, data=merged)
summary(m6SPTS)
m7SPTS <- hlme(SPTS~days, subject ='id', ng=7, mixture=~days, idiag=T, data=merged)
summary(m7SPTS)
m8SPTS <- hlme(SPTS~days, subject ='id', ng=8, mixture=~days, idiag=T, data=merged)
summary(m8SPTS)

#Determine if using a quadratic slope is better than a linear one.
merged$daysquad <- merged$days^2
s1SPTS <- hlme(SPTS~days+daysquad, subject ='id', ng=1,idiag=T, data=merged)
summary(s1SPTS)
s2SPTS <- hlme(SPTS~days+daysquad, subject ='id', ng=2, mixture=~days+daysquad, idiag=T,
data=merged)
summary(s2SPTS)
s3SPTS <- hlme(SPTS~days+daysquad, subject ='id', ng=3, mixture=~days+daysquad, idiag=T,
data=merged)
summary(s3SPTS)
s4SPTS <- hlme(SPTS~days+daysquad, subject ='id', ng=4, mixture=~days+daysquad, idiag=T,
data=merged)
summary(s4SPTS)
s5SPTS <- hlme(SPTS~days+daysquad, subject ='id', ng=5, mixture=~days+daysquad, idiag=T,
data=merged)
summary(s5SPTS)
s6SPTS <- hlme(SPTS~days+daysquad, subject ='id', ng=6, mixture=~days+daysquad, idiag=T,
data=merged)
summary(s6SPTS)
```

```
s7SPTS <- hlme(SPTS~days+daysquad, subject ='id', ng=7, mixture=~days+daysquad, idiag=T,
data=merged)
summary(s7SPTS)
s8SPTS <- hlme(SPTS~days+daysquad, subject ='id', ng=8, mixture=~days+daysquad, idiag=T,
data=merged)
summary(s8SPTS)

#Determine if the quadractic term is significant for the final five-trajectory model.
WaldMult(s5SPTS, pos=15) #daysquad class1, shows if quad term is sig
WaldMult(s5SPTS, pos=16) #daysquad class2, shows if quad term is sig
WaldMult(s5SPTS, pos=17) #daysquad class3, shows if quad term is sig
WaldMult(s5SPTS, pos=18) #daysquad class4, shows if quad term is sig
WaldMult(s5SPTS, pos=19) #daysquad class5, shows if quad term is sig
WaldMult(s5SPTS, pos=c(15:19)) #summary of the sig of the above

#Determine information on class membership
m5SPTS$ns
FMm5SPTS <-m5SPTS$pprob
table(FMm5SPTS$class)
round(table(FMm5SPTS$class)/361,2)

#Create a plot of the final model and the one trajectory model.
merged <- data.frame(merged)
plot(m1SPTS, which="fit",marg=FALSE,var.time="days",bty="n",
shades=TRUE,legend=NULL,
    ylab="SPTS", xlab="Days", main="Trajectories of SPTS scores over days since surgery")
plot(m5SPTS, which="fit",var.time="days",bty="n", shades=TRUE,legend=NULL,
    ylab="SPTS", xlab="Days", main="Trajectories of SPTS scores over days since surgery")

#Testing predictors
m5SPTS.age <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T, data=merged,
classmb=~Age)
summary(m5SPTS.age)
m5SPTS.sex <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T, data=merged,
classmb=~Sex)
summary(m5SPTS.sex)
m5SPTS.preopChronPain <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T,
data=merged, classmb=~preopChronPain)
summary(m5SPTS.preopChronPain)
m5SPTS.MEQ <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T, data=merged,
classmb=~MEQ)
summary(m5SPTS.MEQ)
m5SPTS.preopNumConditions <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days,
idiag=T, data=merged, classmb=~preopNumConditions)
summary(m5SPTS.preopNumConditions)
```

```
m5SPTS.SurgeryType <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T,
data=merged, classmb=~Surgery_Type)
summary(m5SPTS.SurgeryType)
m5SPTS.AvgPain.base <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T,
data=merged, classmb=~AvgPain.base)
summary(m5SPTS.AvgPain.base)
m5SPTS.BPI_Interfere.base <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T,
data=merged, classmb=~BPI_Interfere.base)
summary(m5SPTS.BPI_Interfere.base)
m5SPTS.HADS_D.base <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T,
data=merged, classmb=~HADS_D.base)
summary(m5SPTS.HADS_D.base)
m5SPTS.HADS_A.base <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T,
data=merged, classmb=~HADS_A.base)
summary(m5SPTS.HADS_A.base)
m5SPTS.PCL_Total.base <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T,
data=merged, classmb=~PCL_Total.base)
summary(m5SPTS.PCL_Total.base)
m5SPTS.PCS_Total.base <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T,
data=merged, classmb=~PCS_Total.base)
summary(m5SPTS.PCS_Total.base)
m5SPTS.MEQ.base <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T,
data=merged, classmb=~MEQ.base)
summary(m5SPTS.MEQ.base)

#Calculate the final model including significant predictors
m5SPTS.covar.FV <- hlme(SPTS~days, subject ='id', ng=5, mixture=~days, idiag=T,
data=merged, classmb=~HADS_D.base+HADS_A.base)
summary(m5SPTS.covar.FV)

#Download pprob and pred to an excel file
PPROBm5SPTS <- m5SPTS$pprob
write.csv(PPROBm5SPTS,file="PPROBm5SPTS.csv",row.names=F)
summary(PPROBm5SPTS)
summary(PPROBm5SPTS$pprob)
summary(PPROBm5SPTS$class)
table(PPROBm5SPTS$class)

PREDm5SPTS <-m5SPTS$pred
write.csv(PREDm5SPTS,file="PREDm5SPTS.csv",row.names=F)
summary(PREDm5SPTS)

#Calculate the predicted values
newdata<-
data.frame(days=c(3,90,180,365,730),X1=rep(0,5),X2=rep(0,5),X3=rep(0,5),X4=rep(0,5),X5=re
p(0,5))
```

```
pred0 <- predictY(m5SPTS,newdata,var.time="days")
head(pred0)

new1data<-data.frame(days=c(3,90,180,365,730))
pred1 <- predictY(m1SPTS,newdata,var.time="days")
pred1
```

Syntax for the statistical analysis in SPSS

```
IF  (days >= 0 and days <= 7) RP=1.
VARIABLE LABELS  RP 'Reporting period'.
EXECUTE.
IF  (days >= 60 and days <= 120) RP=2.
EXECUTE.
IF  (days >= 150 and days <= 210) RP=3.
EXECUTE.
IF  (days >= 305 and days <= 425) RP=4.
EXECUTE.
IF  (days >= 550 and days <= 910) RP=5.
EXECUTE.

DATASET DECLARE by_RP.
AGGREGATE
  /OUTFILE='by_RP'
  /BREAK=id RP
  /MorphineEQ_mean=MEAN(MorphineEQ)
  /AvgPain_mean=MEAN(AvgPain)
  /BPI_Interfere_mean=MEAN(BPI_Interfere)
  /HADS_D_mean=MEAN(HADS_D)
  /HADS_A_mean=MEAN(HADS_A)
  /IEQ_Total_mean=MEAN(IEQ_Total)
  /PCL_Total_mean=MEAN(PCL_Total)
  /PCS_Total_mean=MEAN(PCS_Total)
  /SOAPP_Total_mean=MEAN(SOAPP_Total)
  /SPTS_mean=MEAN(SPTS).

DATASET ACTIVATE by_RP.
FILTER OFF.
USE ALL.
SELECT IF (RP >= 1).
EXECUTE.

SORT CASES BY id RP.
CASESTOVARS
  /ID=id
```

```
  /INDEX=RP
  /GROUPBY=VARIABLE.

DATASET ACTIVATE DataSet2.
SORT CASES BY id.
DATASET ACTIVATE by_RP.
SORT CASES BY id.
DATASET ACTIVATE DataSet2.
MATCH FILES /FILE=*
  /FILE='by_RP'
  /BY id.
EXECUTE.

FILTER OFF.
USE ALL.
SELECT IF (class >= 1).
EXECUTE.

ONEWAY MorphineEQ_mean.1 MorphineEQ_mean.2 MorphineEQ_mean.3
MorphineEQ_mean.4 MorphineEQ_mean.5
    AvgPain_mean.1 AvgPain_mean.2 AvgPain_mean.3 AvgPain_mean.4 AvgPain_mean.5
BPI_Interfere_mean.1
    BPI_Interfere_mean.2 BPI_Interfere_mean.3 BPI_Interfere_mean.4 BPI_Interfere_mean.5
HADS_D_mean.1
    HADS_D_mean.2 HADS_D_mean.3 HADS_D_mean.4 HADS_D_mean.5 BY class
  /MISSING ANALYSIS.

FREQUENCIES VARIABLES=preopChronPain preopCancer preopGERD preopHT
preopArthritis preopDM
    preopThyroidDisease preopPVD preopCOPD preopAsthma preopAnemia preopSleepApnea
preopCKF preopCHF
    preopAngina preopPUD preopMI preopDVT
  /STATISTICS=STDDEV MINIMUM MAXIMUM MEAN
  /ORDER=ANALYSIS.

IF  (MorphineEQ_mean.1 <= 90) MEQ_1.cutoff=0.
IF  (MorphineEQ_mean.1 > 90) MEQ_1.cutoff=1.
IF  (MorphineEQ_mean.2 <= 90) MEQ_2.cutoff=0.
IF  (MorphineEQ_mean.2 > 90) MEQ_2.cutoff=1.
IF  (MorphineEQ_mean.3 <= 90) MEQ_3.cutoff=0.
IF  (MorphineEQ_mean.3 > 90) MEQ_3.cutoff=1.
IF  (MorphineEQ_mean.4 <= 90) MEQ_4.cutoff=0.
IF  (MorphineEQ_mean.4 > 90) MEQ_4.cutoff=1.
IF  (MorphineEQ_mean.5 <= 90) MEQ_5.cutoff=0.
IF  (MorphineEQ_mean.5 > 90) MEQ_5.cutoff=1.
EXECUTE.
```

```
VALUE LABELS
MEQ_1.cutoff MEQ_2.cutoff MEQ_3.cutoff MEQ_4.cutoff MEQ_5.cutoff
0 'Below or equal 90 MEQ'
1 'Above 90 MEQ'
EXECUTE.

CROSSTABS
  /TABLES=class BY MEQ_1.cutoff
  /FORMAT=AVALUE TABLES
  /STATISTICS=CHISQ PHI
  /CELLS=COUNT EXPECTED ROW COLUMN TOTAL RESID SRESID
  /COUNT ROUND CELL
  /BARCHART.

CROSSTABS
  /TABLES=class BY MEQ_2.cutoff
  /FORMAT=AVALUE TABLES
  /STATISTICS=CHISQ PHI
  /CELLS=COUNT EXPECTED ROW COLUMN TOTAL RESID SRESID
  /COUNT ROUND CELL
  /BARCHART.

CROSSTABS
  /TABLES=class BY MEQ_3.cutoff
  /FORMAT=AVALUE TABLES
  /STATISTICS=CHISQ PHI
  /CELLS=COUNT EXPECTED ROW COLUMN TOTAL RESID SRESID
  /COUNT ROUND CELL
  /BARCHART.

CROSSTABS
  /TABLES=class BY MEQ_4.cutoff
  /FORMAT=AVALUE TABLES
  /STATISTICS=CHISQ PHI
  /CELLS=COUNT EXPECTED ROW COLUMN TOTAL RESID SRESID
  /COUNT ROUND CELL
  /BARCHART.

CROSSTABS
  /TABLES=class BY MEQ_5.cutoff
  /FORMAT=AVALUE TABLES
  /STATISTICS=CHISQ PHI
  /CELLS=COUNT EXPECTED ROW COLUMN TOTAL RESID SRESID
  /COUNT ROUND CELL
  /BARCHART.
```

```
RELIABILITY
  /VARIABLES=WorstPain LeastPain AvgPain CurrPain
  /SCALE('BPI_Pain_Severity') ALL
  /MODEL=ALPHA.

RELIABILITY
  /VARIABLES=BPI_9a to BPI_9g
  /SCALE('BPI_Interference') ALL
  /MODEL=ALPHA.

RELIABILITY
  /VARIABLES=HADS_1 HADS_3 HADS_5 HADS_7 HADS_9 HADS_11 HADS_13
  /SCALE('HADS_Anxiety') ALL
  /MODEL=ALPHA.

RELIABILITY
  /VARIABLES=HADS_2 HADS_4 HADS_6 HADS_8 HADS_10 HADS_12 HADS_14
  /SCALE('HADS_Depression') ALL
  /MODEL=ALPHA.

RELIABILITY
  /VARIABLES=IEQ_1 to IEQ_12
  /SCALE('IEQ') ALL
  /MODEL=ALPHA.

RELIABILITY
  /VARIABLES=PCS_1 to PCS_13
  /SCALE('PCS') ALL
  /MODEL=ALPHA.

RELIABILITY
  /VARIABLES=PCL_1 PCL_4 PCL_7 PCL_10 PCL_14 PCL_15
  /SCALE('PCL_C_6') ALL
  /MODEL=ALPHA.

RELIABILITY
  /VARIABLES=SPTS_1 to SPTS_12
  /SCALE('SPTS-12') ALL
  /MODEL=ALPHA.
```

References

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