Validation of the Sensitivity to Pain Traumatization Scale (SPTS) in a Clinical Sample of Post-Cardiac Surgery Patients With Persistent Pain

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## ABSTRACT

The present study aimed to validate the psychometric properties of the Sensitivity to Pain Traumatization Scale (SPTS), which measures the propensity to develop anxiety-related somatic, cognitive, emotional, and behavioral responses to pain the resemble features of a traumatic reaction. The factor structure, reliability, and construct (convergent and discriminant) validity of the scale were evaluated using a sample of 108 patients that reported chronic pain post-cardiac surgery. The 12-item SPTS was found to have a one-factor structure, which was consistent with results of the prior EFA analyses of Kleiman et al. (2011) and Roosen (2009). Results of the reliability analysis demonstrate that the 12-item SPTS has excellent overall internal consistency for the overall sample as well as for both men and women. The validity analysis suggested that the SPTS shares significantly more variance with a measure of PTSD symptoms, than with a measure of depressive symptoms, supporting construct validity. Additionally, 12-item SPTS scores were correlated with current pain intensity ratings, and were higher in women compared with men. Aligning with previous research, the SPTS demonstrated good psychometric properties, providing preliminary evidence that the scale is reliable for use in various settings.


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# Introduction

Once considered a one-dimensional construct, pain is now commonly conceptualized as multidimensional and includes physiological, biological, genetic, psychological, emotional and social etiologies, expressions, and maintaining factors. As researchers have gained an appreciation of the complexity of pain, pain modeling has necessarily evolved to reflect this growing understanding. Research has linked an individual's subjective experience of pain, perpetuation of pain, resulting disability, and treatment response to cognitive, affective, and behavioral factors (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). Contemporary pain models highlight the contribution of psychological constructs in pain. Alongside the developing understanding of the multidimensional phenomenon of pain and pain modeling, the measurement of pain has necessarily developed as well. Yet, improvements to the psychometric evaluation of pain are ongoing (Craig & Hadjistavropoulos, 2004). The present thesis examines the relationships among a number of key psychological variables that have been proposed to contribute to chronic pain and prolonged disability, as well as evaluate the reliability and validity of a psychometric measure of a higher-order pain-related anxiety construct proposed to contribute to the vulnerability and maintenance of chronic pain and Post-traumatic Stress Disorder (PTSD).

The introduction is organized into seven sections. In Section 1, the prevalence, burden, and mechanisms of pain are reviewed. In Section 2, important psychological variables identified in pain research are defined. In Section 3, the comorbidity between chronic pain and PTSD is highlighted. In Section 4, models of the etiology and

maintenance of chronic pain and PTSD are reviewed. These models utilize the psychological variables introduced in Section 2 and propose explanations for the high comorbidity between chronic pain and PTSD described in Section 3. In Section 5, the hierarchical organization of the pain-related anxiety constructs identified in Section 2 is explored. Section 6 presents the history of a higher-order factor hypothesized to underlie the pain-related anxiety constructs, Sensitivity to Pain Traumatization (SPT). In the final section, the present study is introduced which endeavors to validate a 12-item self-report measure of this proposed construct, the Sensitivity to Pain Traumatization Scale (SPTS).

1. *Prevalence, Burden, and Mechanisms of Pain*

Modern theories conceptualize pain as a complex interaction of biological, psychological, and social variables. Various physiological and psychological processes interact to affect pain perception (Melzack & Wall, 1965). Thus pain processes are inherently challenging to objectively quantify. While some theorists strive to create an objective definition of chronic pain others, acknowledging its subjective nature, have questioned the utility of such measures (Macrae & Davies, 1999). However, for research purposes, chronic pain is commonly characterized as pain without functional value, which endures for greater than a set timeframe, often 3 months (Merskey, & Bogduk, 1994).

Huge variability exists in prevalence estimates of chronic pain, largely due to variance in survey methods and definitions (Schopflocher, Taenzer, & Jovey, 2011). International prevalence rates range from 2-55% (Schopflocher et al., 2011; Tsang, Von Korff, Lee, Alonso, Karam, Angermeyer, Borges, Bromet, de Girlamo, de Graaf, Gureje, Lepine, Haro, Levinson, Oakley Browne, Posada-Villa, Seedat, &Watanabe, 2008). In

kind, variability among Canadian estimates range from 16% to 41% (Schopflocher et al., 2011). Using the more conservative criteria of pain for six months or longer and of moderate to severe intensity, the 12-month prevalence of chronic pain is observed in alarmingly high rates internationally in developed countries (37.3%, Tsang et al., 2008) and in Canadian adults (18.9%, Schopflocher et al., 2011). Additionally, the development of chronic pain is high within post-surgical settings. About 10% of patients report chronic pain one year after surgery (Katz & Seltzer, 2009).

Chronic pain is the cause of tremendous personal, social, and economic burden. It is associated with decreased quality of life (Douglas, Graham, Anderson, & Rogers, 2004), wellbeing, independence, social functioning, productivity, (Elliott, Smith, Hannafold, Smith, & Chambers, 2002; Breivik, Collett, Ventafridda, Cohen, & Gallacher, 2006; Tsang et al., 2008) and increased disability (Schopflocher et al., 2011). A resent meta-analysis of 110 studies found that compared with the general population, those who experience chronic pain experience significant and substantial problems in all aspects of psychological functioning (Burke, Mathias, & Denson, 2015). Chronic pain places an enormous strain on the healthcare system as well. The national cost of persistence pain conditions in the United States was recently estimated between $560 and $635 billion annually in direct (e.g. treatment) and indirect costs (e.g. absenteeism), well above the nations priority health conditions (Gaskin & Richard, 2010).

In contrast to early conceptualizations of pain that favored biological mechanisms, current theories of pain recognize that the process of pain perception depends on a combination of sensory and emotional aspects (Katz et al., 2009; Merskey et al., 1994). Further, modern models acknowledge both a biological disease process as

well as an illness process which describes the individual's subjective experience of the disease resulting in a range of physical, behavioral, and psychosocial stressors (Turk & Monarch, 2002). These nuanced features of contemporary ideas of pain emphasize the subjective nature of pain, and acknowledge that the same disease or injury can yield different pain processes in different individuals (Gatchel et al., 2007).

The Neuromatrix Theory of pain (Melzack, 2001) is the foundation of our current perspective on pain processes. This theory describes the impact of the brain's unique neural network, or neurosignature, which integrates cognitive, sensory, and motivational functions, on determining the individual's experience of pain (Melzack & Casey, 1968). Neural network output activates both perceptual and behavioral patterns in response to injury or stress (Gatchel et al., 2007). Importantly, this model emphasizes that neural network activation precedes the experience of pain, highlighting that the perception of pain is a process created by a complex internal system (Melzack, 2005).

The organic and dynamic nature of the neuromatrix, implies that our pain processing systems are impacted by our experiences (Gatchel et al., 2007). The hypothalamic-pituitary-adrenal axis (HPA) is the body's stress regulation system that works to restore homeostasis after injury or stress. In the case of chronic pain, the HPA axis is compromised by ongoing stress that impacts immune and limbic response (Gatchel, Haggard, Thomas & Howard, 2012). The physiological impact of this stress can facilitate the biological degradation of muscle, bone, and neural tissue; and fear, anxiety, and cognitions amplify this distress and perpetuate a pain-stress feedback loop (Gatchel et al., 2007).

Informed by the Neuromatrix Theory, biopsychosocial models of pain represent the contemporary framework for pain processes that recognize the influence of biological, physiological, and neural factors, as well as psychosocial and socioeconomic factors that interact to effect the clinical presentation of pain (Gatchel, 2004). This comprehensive framework has been a particular asset to modeling chronic pain, facilitating an understanding of how the various internal and external factors interact to produce the inception and maintenance of pain. For example, diathesis-stress models describe how one's internal framework (e.g. one's predisposition) when activated by acute stressors can set into motion cycles in which emotional, cognitive, and behavioral factors contribute to perpetuate pain experience (e.g. Turk, 2002). The biopsychosocial understanding of pain has supported several lines of research including neuroimaging studies highlighting the role of cortisol in stress system activation and pain perception (e.g. deCharms, 2005), the impact of cognitions on pain perception (e.g. Turk, 2002), and the contribution of behavior patterns to maintaining pain (e.g. fear-avoidance models, Vlaeyen & Linton, 2000).

2. *Key Psychological Variables in Chronic Pain*

A substantive body of research has been dedicated to delineating the psychological constructs that are most relevant to biopsychosocial models of pain. Three key psychological constructs have been identified in the pain literature due to their predisposing and maintaining roles in pain conditions (Martin, Halket, Asmundson, Flora & Katz, 2010): Anxiety Sensitivity, Fear of Pain/Pain Anxiety, and Pain Catastrophizing.

Anxiety Sensitivity (AS) describes an individual's predisposition towards heightened attention to bodily sensations, accompanying negative evaluation of these

sensations, and resulting fear of bodily experiences such as the physical correlates of anxiety (e.g. rapid hard beat) (Reiss, 1991). Individuals high in AS also experience a heightened awareness of and pain and tend respond more fearfully to these sensations (Keogh & Birkby, 1999; Asmundson & Taylor, 1996). Research has also associated AS and PTSD (for review see; Asmundson, Coons, Taylor, & Katz, 2002). AS has been shown to be a predictor for developing PTSD symptoms (Fedoroff, Taylor, Asmundson & Koch, 2000), a maintenance factor (Taylor, Koch, & McNally, 1992), and is higher in individuals with anxiety disorder such as PTSD compared with non-clinical populations (Olatunji & Wolitzky-Taylor, 2009).

Pain Anxiety (PA) describes the tendency to respond to pain with cognitive, affective, physiological and behavioral aspects of fear or anxiety (McCracken, Zayfert & Gross, 1992; McCracken & Dhingra, 2002). In parallel to the negative attributions ascribed to physical sensations in individuals high in AS, individuals high in PA exhibit unrealistic concern about engaging in activities for fear of injury and pain (McCracken et al., 1992). A related construct, Fear of Pain (FOP) (McNeil & Rainwater, 1998) has been linked with physical impairment and disability in different chronic pain conditions (Vlaeyen, Kole-Snijders, Rotteveel, Ruesink & Heuts, 1995; Martin, McGrath, Brown & Katz, 2007). It has been shown that AS and FOP significantly predict disability over several constructs including pain severity (McCracken et al., 1992; Zvolensky, Goodie, McNeil, Sperry & Sorrell, 2001).

Pain Catastrophizing (PC) describes a psychological response to pain that includes rumination, magnification, and thoughts of helplessness (Sullivan, Bishop & Pivik, 1995; Burns, Ritvo, Ferguson, Clarke, Seltzer & Katz, 2015). In PC, negative

interpretations about the experience of pain and future proliferate which in turn intensify the physiological experience of pain (Sullivan, Thorn, Haythornwaite, Keefe, Martin, Bradley, Lefebvre, 2001). Similar to AS and PA, PC supports a fear-based avoidance response. PC has been shown to predict pre- and post- operative pain ratings (Burns et al., 2015; Roth, Tripp, Harrison, Sullivan & Carson, 2007; Sullivan, Tanzer, Stanish, Fallaha, Keefe, Simmonds & Dunbar, 2009) and disability (Sullivan, Stanish, Waite, Sullivan & Tripp, 1998).

*3. Comorbidity of Chronic Pain and PTSD*

In addition to examining the role of individual psychological factors in chronic pain populations, psychological comorbidity is a growing focus of chronic pain research. In particular, Post-Traumatic Stress Disorder (PTSD) and Post-Traumatic Stress Disorder symptoms (PTSS) are highly comorbid with chronic pain. In large community samples, the odds ratios for the anxiety disorders in people with chronic pain are between 1.5 and 2.6 (Von Korff , Crane, Lane, Miglioretti, Simon, Saunders, Stang, Brandenburg & Kessler, 2005) and for pain in people with PTSD between 2.0 and 3.5 (Sareen, Cox, Stein, Afifi, Fleet, & Asmundson, 2007). The comorbidity of pain and PTSS is approximately 50% in chronic pain patients (Asmundson et al., 2002).

PTSD is considered a risk factor for chronic pain, extending the chronicity of pain (Shaw, Means-Christensen, Slater, Webster, Patterson, Grant, Garfin, Wahlgren, Patel & Atkinson, 2010), and pain-related disability (Katz, Pagé, Fashler, Rosenbloom & Asmundson, 2014). In chronic pain patients, comorbid PTSD has been shown to worsen the subjective experience of pain and is associated with greater emotional distress (Geisser, Roth, Bachman & Eckert, 1996) and intensity of pain (Asmundson et al., 2002).

PTSD has also been shown to complicate pain prognosis and is associated with greater functional impairment (Duckworth & Iezzi, 2005; Palyo & Beck, 2005) and utilization of healthcare services (Asmundson et al., 2002).

Chronic pain and PTSD/PTSS significantly overlap in their symptomatology. In particular, the role of avoidance is highlighted in the literature on both conditions. One of the foundational symptom clusters in PTSD is avoidance of stimuli and situations associated with the traumatic event(s) (American Psychiatric Association, 2013). Similarly, fear-avoidance models of pain describe the tendency of individuals to avoid feared painful situations and the accompanying reduction in anxiety reinforces the avoidant behavior (Vlaeyen et al., 2000). In pain conditions, avoiding possible pain by restricting movement can exacerbate the condition by increasing muscle stiffness, impeding functioning, and contributing to physical deconditioning (Vlaeyen, Seelen, Peters, de Jong, Artz & Beisiegel, 1999). This cycle has also been associated with an increase in disability (Asmundson, Norton, & Norton, 1999; Crombez, Vlaeyen, Heuts, & Lysens, 1999).

4. *Models of Chronic Pain and PTSD Etiology and Maintenance*

Researchers have begun to model the ways that these distinct variables interact to explain the predisposition to and maintenance of chronic pain and disability, as well as the comorbidity between chronic pain and PTSD. These models can be grouped in to: mutual maintenance, shared vulnerability, and combined models.

The original mutual maintenance model forwarded by Sharp & Harvey (2001) proposes that physiological, cognitive, affective, and behavioral aspects of PTSD perpetuate symptoms of pain and vice versa. In alignment with the biopsychosocial

description of pain, this model identifies seven interacting mechanisms and possible pathways that perpetuate these disorders: perception (e.g. attention and reasoning), the impact of individual difference factors (e.g. anxiety sensitivity), memory/learning (e.g. past trauma), behavior (e.g. avoidance), comorbid psychiatric factors (e.g. depression and under-activity; and anxiety and pain perception), and stress (e.g. cognitive burden resulting from pain).

For example, chronic pain that results from trauma can become a trigger for memories of the initial traumatic event, and thus precipitate the symptoms of PTSD such as avoidance and hyper-arousal (Sharp et al., 2001). This sequence of events becomes a motivator to avoid potential pain exacerbating situations that may perpetuate the psychological and physiological symptoms of chronic pain and PTSD, inadvertently reinforcing withdrawal and disability. Several of these mechanisms have received empirical support. For example, as previously discussed, AS has been linked independently to PTSD and to pain.

Liedel and Knaevelsrud (2008) forwarded the Perpetual Avoidance Model, which was informed by Cognitive Behaviour Therapy (CBT) for PTSD (Ehlers & Clark, 2000) and the Fear-avoidance Model of musculoskeletal pain (Vlaeyen et al., 2000). In this model, avoidance is the reinforcing link between PTSD and pain symptoms. When trauma/painful events occur and are perceived as threats, maladaptive coping and fear-avoidance follows resulting in distress and disability. Symptoms of PTSD can exacerbate pain, which in turn promotes fear and avoidance and reinforces the belief that movement is harmful, which then leads to inactivity. However, this model does not explain the specific contribution of chronic pain symptoms to maintaining PTSD (Katz et al., 2014).

Shared vulnerability models conceptualize mutual maintenance factors as predisposing risk factors for the comorbidity between PTSD and chronic pain. These frameworks propose that psychological factors which influence individual dispositions, such as AS, when met with certain environmental conditions can provide a diathesis for chronic pain and anxiety (Asmundson et al., 2002; Martin et al., 2010).

Asmundson et al. (2002) developed the initial Shared Vulnerability Model for comorbid musculoskeletal pain and PTSD, which highlights three individual difference factors predisposing toward this comorbidity when subject to environmental precipitants. Genetic psychological vulnerability (e.g. AS), low physiological alarm threshold, and stressful life events (e.g. trauma, pain) are thought to facilitate negative emotional responses (e.g. negative thoughts), which in turn interact with physiology, behavior, and cognitions. This model explains the development of PTSD and chronic pain independently as well as the increased risk for comorbidity. Different aspects of the model may explain different anxiety disorders, for example the hyperarousal related to panic disorder, and the apprehensive thinking to social phobia (Katz et al., 2014).

The Triple Vulnerability Model (Otis, Keane, & Kerns, 2003) also has a tripartite predispositional structure: generalized biological vulnerability, impoverished sense of control, and psychological predisposition to focus on anxiety. In this model, various psychological tendencies such as individual response styles to pain, past experience, impoverished coping, and a sense of helplessness, facilitate lowered self-efficacy and increased negative affect, thus supporting avoidance and disability. These two vulnerability models highlight the role of biological and psychological dispositions in the increased risk for this comorbidity. Whereas mutual maintenance models are especially

relevant to cases where chronic pain and PTSD are the result of a shared traumatic of the two conditions.

Combined mutual maintenance and vulnerability models describe how individual difference factors predispose and maintain PTSD and chronic pain. Turk (2002) proposed a diathesis-stress model of chronic pain and disability. This model was an elaboration of a model by Asmundson & Taylor's (1996) in which pain-related escape/avoidance behaviors were increased by high AS through its influencing on FOP. In a study of adults with musculoskeletal pain, 30% of the variance in FOP was accounted for by AS, and 68% of the variance in escape/avoidance was accounted for by fear of pain. This model was also replicated with a sample of headache pain patients (Norton & Asmundson, 2004). Turk (2002) synthesized mutual maintenance, shared vulnerability, and fear-avoidance models. He proposed that individuals who are high in AS (e.g. a predisposing individual difference factor) who experience trauma, are more likely to develop FOP, PC, and diminished self-efficacy, which support escape and avoidance behaviors that lead to disability. Disability then reinforces FOP and PC, and lowered self-efficacy. Like the mutual maintenance models, this is lends well to the context in which a single stressor precipitates both chronic pain and PTSD (Katz et al., 2014).

Martin and colleagues (2010) set out to examine the relationships proposed in Turk's model (2002; omitting self-efficacy) using Structural Equation Modeling (SEM) with a sample of patients scheduled for general surgery. Additionally, they proposed a second model that incorporated the potential contribution of PTSS. The first SEM analysis supported a model that included several of relationships proposed by Turk. This included a path in which AS predicted FOR and PC, and FOP predicted escape and

avoidance behaviors, which in turn predicted disability, and a feedback loop by which disability affected FOP. The second analysis provided preliminary support for role of PTSS in the model, as it accounted for a significant proportion of the variance in disability.

Recently, Rosenbloom and colleagues (2013) proposed a comprehensive combined model in which biological and psychological vulnerabilities when exposed to traumatic injury increase risk for chronic pain and PTSD. This model includes several of the factors from previous models (e.g. AS, low threshold for alarm) and adds others (e.g. HPA sensitivity, previous trauma, comorbid diseases), which interact to facilitate mutual maintenance through fear-avoidance behavior. The symptom overlap between each of the disorders was proposed to trigger and maintain the other. This model was tested in a prospective observational study examining the development of PTSD symptoms subsequent to traumatic musculoskeletal injury (Rosenbloom, 2014). Results suggested that neuropathic pain, general anxiety, pain anxiety, and PC were associated with symptoms of PTSD.

5. *Hierarchal Organization of Pain-related Anxiety Constructs*

The mutual maintenance, shared vulnerability, fear/anxiety avoidance, and diathesis stress paradigms have advanced an understanding of the etiology and maintenance of comorbid pain and PTSD. The contribution of the pain-related anxiety constructs to these models cannot be understated. Substantial evidence points to the differential presentation of the pain-related anxiety constructs in chronic pain compared with nonclinical samples, and several pain-related anxiety constructs have been separately connected to pain and PTSD. However, research examining how these

constructs are conceptually related has lagged behind theoretical models (Kleiman et al., 2011). Some researchers have posed the question whether the comorbidity between chronic pain and PTSD may be due to an underlying construct to which these individual variables can be attributed (e.g. Keogh & Asmundson, 2004). Within the few studies that have examined the relationship between these constructs, there is disagreement on how they are connected.

Several studies have highlighted the partial overlap of these constructs (e.g. Hadjistavropoulos, Asmudnson & Kowalyk, 2004; Keogh et al., 2004; McCracken, Gross, Aikens, & Carnike, 1996). Of particular debate is the relationship between AS and PA. It has been shown that reducing AS also reduces PA in undergraduate women (Watt, Stewart, Lefaivre, Uman, 2006), and that AS targeted CBT reduces PA (Olthuis, Watt, Mackinnon, Potter, Stewart, 2015). Some researchers have proposed that AS is a higher-order construct that subsumes PA (Asmundson et al., 1999a; Asmundson, Norton, & Veloso, 1999), and that PA is a manifestation of AS (Greenberg & Burns, 2003). AS was found to be highly associated with similar psychological constructs such as FOP and negative affect in clinical samples (Ocañez, McHugh & Otto, 2010).

In contrast, the independent and unique predictive value of these constructs has also been emphasized (Carleton & Asmundson, 2009; Hadjistavropoulos et al., 2004). The contribution of both AS and PA for the development and maintenance of chronic pain has been highlighted (e.g. Asmundson, Vlaeyen, & Crombez, 2004; Norton et al., 2004), and researchers have attempted to elucidate this distinction. The findings from a study using Confirmatory Factor Analysis (CFA) and canonical correlations (Carleton et al., 2009a) supported the construct independence and interdependence of FOP, AS, and

PA. The closer correlation between the AS and PA suggest a difference between anxiety and fear states (represented by FOP), substantiating literature suggesting this distinction (Barlow, 2000). AS and PA have been independently related to fear of bodily sensations, (Gonzalez, Zvolensky, Hogan, McLeish, Weibust, 2011) and while highly correlated, their respective presentations in anxiety disorder compared with non-clinical samples differs (Carleton, Abrams, Asmundson, Antony, McCabe, 2009). The independence of the PC construct has also received attention. For example, PC has been shown to increase attentional interference whereas AS and illness/injury sensitivity do not (Vancleef & Peters, 2006). Drahovzal and colleagues (2006) concluded that while AS and PC are empirically separate constructs, they overlap through a fear of physical catastrophe.

Contrary findings and interpretations point to the need to clarify the organizational structure framing these constructs and several authors have hypothesized different systems. At present, efforts to uncover a hierarchy of the pain-related anxiety constructs have taken a broad scope by examining how these constructs fit into a general framework of negative affect and anxiety. Highlighting the relationship between AS and negative affect more broadly (including other emotional intolerance and sensitivity constructs), investigations concerning their structural relationship have been recommended (Bernstein, Zvolensky, Vujanovic, & Moos, 2009).

One investigation of the hierarchy of the anxiety and fear constructs that relate to pain, found four underlying factors (Vancleef, Vlaeyen & Peters, 2009). This study involved a card sort task of nine pain-related anxiety measures in a group of undergraduate students. Cluster analyses yielded four clusters: negative emotion and anxiety (highest general level), cognitive performance concerns and physical health

concerns (fundamental fears), and pain-specific concerns (specific manifest expression of the higher levels). Multidimensional scaling analyses suggested these constructs are best characterized dimensionally from general (e.g. negative affect and anxiety) to specific (e.g. pain-specific concerns). This investigation included a wide range of measure, including depression (e.g. Hospital Anxiety and Depression Subscale) and general affective subscales (e.g. Negative Emotionality Subscale). It found that pain specific concerns were at the lowest level of a three-tiered hierarchy.

Bernstein and colleagues (2009) examined the latent dimensional and hierarchal structure of AS, distress tolerance, discomfort intolerance. The Distress Tolerance Scale (Simons & Gaher, 2005) measures the extent to which affective intensity is overwhelming and intolerable, while the Discomfort Intolerance Scale (Schmidt, Richey, & Fitzpatrick, 2006) measures the extent to which physical discomfort is unbearable. Questionnaires were completed by a control sample and a two-stage Exploratory Factor Analysis (EFA) yielded one higher-order factor (affect tolerance and sensitivity) accounting for 40.3% of the variance in two lower-order facets (AS and distress tolerance). Discomfort intolerance was not related to the higher-order factor. It was concluded that distress intolerance and AS are lower-order constructs of a higher-order factor that accounts for individual differences in sensitivity and intolerance of emotional states. This added to the literature associating distress tolerance, which is typically connected with emotional dysregulation disorders, with PTSD symptoms (e.g. avoidance, hyperarousal, re-experiencing) and coping (e.g. negative appraisal, maladaptive coping). Thus suggesting that distress tolerance is relevant to PTSD (Fetzner, Peluso, & Asmundson, 2014).

Others have referred the pain-related anxiety constructs back to the "three fundamental fears" underlying the anxiety disorders proposed by Reiss (1991): AS, Illness/Injury Sensitivity (IIS), and Fear of Negative Evaluation (FNE). Previous studies have independently linked these three fear categories to negative affect (Thibodeau, Carleton, Collimore, & Asmundson, 2012) although the relationship of positive affect to this group is unclear. Some have suggested that PA should be added to the three fear categories (Carleton et al., 2009a) and along with Intolerance of Uncertainty (IU), that the five may together be organized according to a hierarchy (Carleton, Thibodeau, Osborne, Taylor, & Asmundson, 2014).

Using correlation and regression analyses, Thibodeau and colleagues (2012) found the 3 fundamental fears, including AS, shared significant shared variance (42%) with measures of transdiagnostic constructs representing distress [e.g. Negative and Positive Affect: Clark & Watson (1991)]. Another study tested the construct independence of the three fundamental fears (and added IU and PA) using CFA and EFAs, which supported their independence as well as highlighted their overlap (Carleton et al., 2014). This study replicated the early findings of Taylor (1993), providing evidence for their distinction, as well as raising questions on their interdependence.

Relatedly, in an study examining the relationship between the cold pressor test (CPT) pain threshold with pain-anxiety measures (e.g. AS, FOP, PC, hypochondriasis, and somatic amplification) and personality constructs, it was found that negative affect, neuroticism, and all pain anxiety measures loaded on a single underlying latent factor ("pain/body sensitivity") substantiating their meaningful intercorrelations (Lee, Watson, & Frey Law, 2010). Interestingly, this factor was more strongly predictive of pain quality

than were the higher-order traits. They proposed AS is related to the tendency to catastrophize somatic sensations, which has previously been suggested (Drahovzal et al., 2006).

It is clear from these investigations that the dual overlap and distinction between the pain-related anxiety constructs such as AS, PA, and PC challenges attempts to definitively categorize them with respect to one another. However, given their independent contributions to forwarding our understanding of chronic pain processes and comorbidity with PTSD, mapping their inter-relationships is of central importance. Several studies that have examined these constructs with reference to transdiagnostic factors and begun to relate them to a more general disposition toward negative affect. Nevertheless, it has not yet been clarified how the organization of these constructs relate specifically to the predisposition toward to comorbid pain and PTSD. Several authors have encouraged the investigation of the pain-related anxiety constructs, suggesting that one factor involving "fear of pain or somatic sensations" underlies these lower-order categories (e.g. Carleton, Park & Asmundson, 2006; Keogh et al., 2004; Kleiman et al., 2011).

6. *Sensitivity to Pain Traumatization*

Kleiman et al. (2011), set out to investigate the relationship between pain and trauma by exploring the hierarchical organization of the major pain-related anxiety constructs, and how this order links pain and PTSD. EFA and a subsequent higher-order analysis were utilized to assess the organization of these constructs represented by the Anxiety Sensitivity Index (ASI), Pain Anxiety Symptom Scale (PASS-20) and Pain Catastrophizing Scale (PCS) items, in 444 patients scheduled for major surgery. The

results of the study suggested a bi-factor model in which a first-order analysis revealed a 3-factor structure confirming the uniqueness of the ASI, PCS and PASS-20. A second-order analysis showed that 47 of the 49 items analyzed loaded on a single higher-order factor more strongly than the first 3-factors and that explained 68.3% of the common variance.

The authors also found that 20 items from the ASI, PCS and PASS-20 loaded exclusively on this higher-order factor but did not significantly load on the 3-first order factors. The commonality between these 20-items suggested the higher-order factor described "… the propensity to develop anxiety-related somatic, cognitive, emotional and behavioral responses to pain the resemble features of a traumatic reaction" (Kleiman et al., 2011 p. 175). This factor was termed Sensitivity to Pain Traumatization (SPT). The authors further categorized the items into three main symptom-clusters including: Re-experiencing (e.g. intrusive thoughts, intense psychological distress); Avoidance; and Hyperarousal; which are the behavioral, cognitive, and emotional reactions to pain that resemble the symptoms of PTSD.

The authors then tested the construct validity by correlating the 20-item SPT total with a measure of PTSD, the PTSD Checklist-Civilian Version (PCL-C). They found strong, significant positive correlations ($p < 0.001$) in both those that reported pain ($r = .49$) and who did not ($r = .48$), linking the pain-related anxiety constructs to PTSD and supporting convergent validity. However SPT scores were also distinguished from PCL-C scores. It was found that pre-surgery SPT-scores were significantly higher for those reporting pain at 1-year post-surgery compared with those reporting no pain ($t[288]=2.27$, $p=0.02$) but not PCL-C scores ($t[288]=1.44$, $p=0.15$). Furthermore, the total SPT score

was higher for those who reported pain compared with no pain prior to surgery ($t[419]=2.28$, $p=0.02$). The authors suggest that while construct overlap exists between the SPT and a traumatic stress reaction such as PTSD, there are distinguishing features. While PTSD describes somatic, cognitive, emotional and behavioral responses to trauma broadly defined, the SPT describes a specific propensity to develop a traumatic reaction to pain.

This study offers preliminary evidence of an underlying vulnerability factor that represents a risk to experience a traumatic reaction when exposed to a painful event. These findings also suggest that SPT may also be a maintenance factor, extending pain chronicity by triggering reactions to pain such as catastrophizing and avoidance, which are cognitive and behavioral patterns that facilitate chronic pain. As research has shown, individual pain-related anxiety constructs predispose individuals to develop chronic pain. However, the SPT factor as a higher-order construct subsuming these individual constructs and would represent the individual's cumulative disposition toward developing chronic pain. The authors recommend that future studies design a measure aimed at capturing traumatic-stress reactions to pain. The questionnaires use in the study represented this content only partially and the authors emphasize the need to include the full spectrum of PTSD symptom clusters such as emotional numbing when designing an SPT measure.

Building on the findings of Kleiman et al. (2011), Roosen (2009) developed a 12-item self-report Sensitivity to Pain Traumatization Scale (SPTS) and evaluated its preliminary psychometric properties. Roosen (2009) created an initial sample of items based on the categories that Kleiman et al. (2011) defined for the SPT construct and

added items representing the full spectrum of PTSD symptoms from the DSM-IV-TR (APA, 2000; hyperarousal, avoidance, emotional numbing, re-experiencing), as well as the main pain-related anxiety symptoms inventories (e.g. ASI, PCS, PASS-20, PCL-C, Fear of Pain Questionnaire-III [FPQ-III], Impact of Events Scale [IES]).

The item pool was categorized into 6 subscales: 1) Pain and Avoidance, 2) Pain and Emotional Numbing, 3) Pain and Hyperarousal, 4) Pain Experiencing, 5) Fear of Pain, and 6) Pain Sensitivity. Initially 203 items were generated, and after removing redundant questions through collaborative consultation, 79 items were retained for the study. The questionnaire asked individuals to rate the degree to which they agree with items representing thoughts, feelings, and beliefs individuals might have when experiencing physical pain. Possible responses were on a 5-point likert scale: 0 = "Not at all True"; 1 = "Slightly True"; 2 = "Somewhat True"; 3 = "Very True"; 4 = "Entirely True"). The preliminary 79-item pool of was given to a sample of undergraduate students.

Data from the 79-items of 105 participants were reduced using non-parametric Item Response Theory (IRT), and analyzed using a regression model which yielded 12-items. These 12-items formed the proposed SPTS. These items were analyzed with a parametric IRT graded response model. EFA with principle axis factoring and a varimax rotation was used to examine the underlying factor structure of the items. Using parallel analysis (Horn, 1965), rule of eigenvalues (>1) and Cattell's scree test (Costello et al., 2005) a 1-factor solution was determined. All items demonstrated sufficient factor loadings for the 1-factor solution to be considered reliable (> 0.60; Guadagnoli & Velicer, 1988) and explained 53.67% of the total variance. The scales internal

consistency was assessed using Cronbach's alpha and was found to be high (α = 0.9167) and deletion of any one-item did not affect the internal consistency of the overall scale (α = 0.9028 – 0.9207). These results suggest good preliminary psychometric properties of the scale.

The results of this study suggest some relationship between pain and SPTS score. Participant's who rated questions asking about the intensity of common day-to-day pains (e.g. paper cut, stubbed toe) as highly painful ($M$ = 2.68, $SD$ = 0.95) compared with those who rated them as not very painful ($M$ = 1.84, $SD$ = 0.67) had significantly higher mean total SPTS scores ($F_w$ (1, 69.82) = 19.59, $p < 0.001$). Taken together, the results suggest the 12-item SPTS demonstrates good psychometrics and may differentiate individuals based on pain sensitivity.

Roosen (2009) proposed several areas for the future development of the SPTS. The promising findings from the study suggested the potential clinical utility of the SPTS as a screening tool to identify individuals with greater risk for reacting to pain as a traumatic stressor thereby promoting the transition from acute to chronic pain. Thus, an imminent step is to validate the SPTS within a clinical population. In addition, in order to clarify the relationship between the SPTS and PTSD symptoms, it should be tested alongside a measure of such as the PCL-C. Due to the challenge of studying pain processes empirically, Roosen (2009) suggested studying the SPTS in a post-surgical sample.

7. *Present Study*

SPT may offer explanation for the high comorbidity rate between chronic pain and PTSD, through its predisposing and maintaining mechanisms (Kleiman et al., 2011).

As evidence suggests comorbid chronic pain and an anxiety disorder complicates treatment of both disorders (Asmundson, Abrams & Collimore, 2008; Teh, Morone, Karp, Belnap, Zhu, Weiner & Rollman, 2009), developing clinical self-report measures to identify a predisposition toward both conditions is highly relevant to clinical care. The present study aims to evaluate the reliability, validity, and factor structure of the 12-item SPTS in a clinical sample of patients with persistent pain following cardiac surgery. This project will assist in building an empirical understanding of the relationship between chronic pain and PTSD, with the long-term goal of supporting knowledge translation and informing assessment and treatment protocols for this population.

## Methods

### *Hypotheses*

The goal of the present study is to evaluate the psychometric properties of the 12-item SPTS, including its factor structure, reliability, and construct (convergent and discriminant) validity in a clinical sample of cardiac patients who reported persistent pain post-surgery. It is hypothesized that the:

1) SPTS will have a 1-factor structure;
2) SPTS total scores will correlate with the PTSD Checklist-Civilian Version (PCL-C). More specifically, correlating the SPTS with the PCL-C, a theoretically similar psychological construct but not one specifically related to pain, will assess convergent validity. High correlations ($r > 0.60$) will indicate adequate convergent validity. In addition, higher intensity pain ratings will correlate with higher SPTS total scores.

3) SPTS will have low to moderate correlations ($r < 0.60$) with the Hospital Anxiety and Depression Scale Depression Subscale (HADS-D), indicating adequate discriminant validity.

*Participants*

The study sample consisted of 108 patients who had undergone Coronary Artery Bypass Graft Surgery (CABGS) at the Toronto General Hospital (TGH), Toronto, Ontario and experienced chronic pain at one or both surgical sites (chest and/or leg) at least 6 months after surgery. This sample was selected from a pre-existing anonymized dataset examining risk and protective factors involved in the transition from acute to chronic pain post-surgery.

*Procedures*

The present study was granted ethics approval from the Human Participants Review Committee (HPRC) at the Office of Research Ethics at York University, Toronto, Ontario on May 25th, 2015. The initial research study received ethics approval from the University Health Network Research Ethics Board, Toronto, Ontario on December 13th, 2010 and was conducted at the TGH. Participants were patients who had undergone CABGS and had consented to be contacted regarding their recovery process and upcoming research studies. Participants were invited to participant at a minimum of 6 months post-surgery. At this time a member of the research team explained the study.

Patients were given the three options to participate: 1) complete a short questionnaire over the phone, 2) complete an additional long questionnaire(s) (one if they indicated that they did not have any pain after surgery, two if they indicated they had pain post-surgery) sent by mail, and 3) complete the questionnaires from option 2 in additional

to undergoing psychophysical testing at the TGH lab. Patients interested in participating in the phone portion only (option 1) completed verbal consent that was documented and a research team member offered to mail a copy of the consent form for the patients' records. Patients who participated in option 2 or 3 completed written consent either returned by mail (option 2) or completed at the lab (option 3).

All patients who consented participated in a phone questionnaire during which they were asked to describe and rate the intensity, duration, and frequency of their pain post-surgery. All patients who reported experiencing chronic pain after surgery (pain of three months or longer) beginning at least three months after surgery were considered part of the chronic pain groups, regardless of whether it was current. Participants included in the present analysis were those who reported chronic post-surgical chest or leg pain at some point following surgery (not necessarily current) and completed option 1 and 2 or 3 (data from only the questionnaires from option 3, not the psychophysical testing).

After the telephone questionnaire, participants in the present sample were given a longer questionnaire package either mailed or in person (option 2 or 3) that included the: Sensitivity to Pain Traumatization Scale (SPTS), Anxiety Sensitivity Index 3 (ASI-3), Pain Anxiety Symptom Scale (PASS-20), Pain Catastrophizing Scale (PCS), PTSD Checklist-Civilian Version (PCL-C), Hospital Anxiety and Depression Scale (HADS), Chest Pain Intensity Numeric Rating Scale (CI-NRS), the Self-Report Leeds Assessment of Neuropathic Symptoms and Signs (S-LANSS), and Chronic Post-Cardiac Surgery Pain Questionnaire: Long Survey.

*Measures*

Sensitivity to Pain Traumatization Scale (SPTS)

The SPTS (Roosen, 2009) is a 12-item scale that measures sensitivity to experience pain traumatization and indicates a higher risk to experience a traumatic reaction if exposed to a painful event (see Appendix A). Items on the scale were generated: to represent the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) criteria for PTSD to reflect a traumatic stress reaction to pain; by using the items from the major pain-anxiety scales, and through a comprehensive literature review. SPTS items are rated on a 5-point scale ranging from 0 (not at all true) to 4 (entirely true) with a total possible score of 48. The measure has a total score as well as 6 subscales: Pain and Avoidance, Pain and Emotional Numbing, Pain and Hyperarousal, Pain Experiencing, Fear of Pain, and Pain Sensitivity. Items consist of beliefs, thoughts, feelings, and actions that individuals may experience when they are in physical pain. There is preliminary evidence to suggest it demonstrates good psychometric properties (Roosen, 2009).

Anxiety Sensitivity Index-3 (ASI-3)

The ASI-3 is a revision of the 16-item scale ASI that is widely used (Reiss, Peterson, Gursky, & McNally, 1986). The scale measures concerns that anxiety and anxiety-related symptoms may lead to harmful negative consequences. Items are rated on a 5-point scale ranging from 0 (very little) to 4 (very much), with a total possible score of 80. The ASI-3 yields a total score and three factor analytic subscale scores. The subscale scores measure fear of: Somatic, Cognitive, and Social manifestations of anxiety. The ASI has strong psychometric properties including good test-retest reliability ($r = 0.72$)

and discriminant validity of the three subscales within anxiety outpatient settings (Rodriguez, Bruce, Pagano, Spencer & Keller, 2004).

Pain Anxiety Symptom Scale-20 (PASS-20)

The PASS-20 is an abbreviated 20-item revision of the original scale (McCracken & Dhingra, 2002). This scale is designed to measure fear and anxiety responses specific to pain. Items are rated on a 5-point scale ranging from 0 (never) to 4 (always), with a total possible score of 100. The PASS-20 has a total score as well as four subscales. The subscale scores measure: Cognitive Anxiety, Escape and Avoidance, Fearful Thinking, and Physiological Anxiety. The PASS-20 has been validated in clinical and nonclinical samples (Abrams, Carleton, & Asmundson, 2007; Coons, Hadjistavropoulos & Asmundson, 2004).

Pain Catastrophizing Scale (PCS)

The PCS describes thoughts and feelings that individuals may experience when they are in pain (Sullivan, et al., 1995). The scale consists of 13 items rated on a 5-point scale ranging from 0 (not at all) to 4 (all the time), with a possible score of 65. The PCS yields a total score and three subscale scores. The subscale scores measure: Rumination, Magnification, and Helplessness. The PCS has adequate to excellent internal consistency in community ($\alpha$ = 0.88-0.95) and outpatient pain samples ($\alpha$ = 0.75 – 0.92) (Osman, Barrios, Gutierrez, Kopper, Merrifield & Grittmann, 2000). It also shows good test-retest reliability ($r$ = 0.70 – 0.75) (Sullivan et al., 1995).

PTSD Checklist-Civilian Version (PCL-C)

The PCL-C is a self-report measure that assesses the DSM-IV symptoms of PTSD (Weathers, Litz, Herman, Huska & Keane, 1993). It is a 17-item measure that is rated on a 5-point scale ranging from 1 (not at all) to 5 (all the time) with a possible score of 85. The PCL-C yields a total score and four subscale scores. The subscale scores measure: re-experiencing, avoidance, numbing, and hyperarousal. The PCL-C has been shown to be a reliable and valid measure of clinically significant symptoms (Weathers et al, 1993; Blanchard, Jones-Alexander, Buckley & Forneris, 1996).

Hospital Anxiety and Depression Scale (HADS)

The HADS is a self-report measure that captures clinically significant anxiety and depression (Zigmond & Snaith, 1983). It has 14-items which are rated on a 5-point scale with a possible score of 70. The wording of the scale varies from question-to-question. It yields a total score as well as 2 subscale scores for anxiety and depressive symptoms, respectively (HADS-A; HADS-D). The HADS has been shown to have good screening properties for the separate dimensions of anxiety and depression in general and clinical populations (Bjelland, Dahl, Haug & Neckelmann, 2002).

Chest Pain Intensity Numeric Rating Scale (CI-NRS)

As a part of the Chronic Post-Cardiac Surgery Pain Questionnaire: Short Survey collected by telephone, questions on demographic and clinical information, experience of pain before surgery and the qualitative and quantitative descriptors of pain after surgery (intensity, duration, and frequency) were collected (see Appendix B). Participants were asked if they had experienced chronic chest pain from their surgery (pain for 3 months or longer beginning at least three months after surgery) and if so, asked to rate the intensity

of a typical pain episode rated on an 11-point Numerical Rating Scale (CI-NRS) ranging from 0-10 with zero indicating "no pain" and 10 indicating "most intense pain imaginable". CI-NRS score was taken to represent their average level of chronic chest pain intensity experienced after the surgery. In addition, participants were asked if they had experienced chronic post-surgical leg pain as a result of their surgery ("Yes" or "No").

Self-Report Leeds Assessment of Neuropathic Symptoms and Signs (S-LANSS)

The S-LANSS (Bennett, Smith, Torrance, & Potter, 2005) is a self-report measure that records the location, intensity, and quality of pain to identify pain that is neuropathic in origin. The S-LANSS contains a map of the body to identify pain location, and 11-point NRS scale ranging from 0 (no pain) to 10 (pain as severe as it could be) to rate pain intensity, and 7 "yes"/"no" questions about pain quality. In the present study, only the NRS scale portion of the scale is used, and "S-LANSS" refers to this NRS scale. The S-LANSS has been validated in community (Weingarten, Watson, Hooten, Wollan, Metlon, Locketz, Wong, & Yawn, 2007) and clinical (Bennett et al., 2005) settings. In the self-report format, it has a high internal consistency ($\alpha$ = .76). The S-LANSS score was taken to represent the current pain intensity (i.e., at the time of completing the S-LANSS) of their chronic post-surgical pain.

Chronic Post-Cardiac Surgery Pain Questionnaire: Long Survey

As a part of the long survey completed by patients, questions on pain at surgery cites, pain rating of everyday pains, chronic post-surgical pain duration, other (non-surgical) chronic pain, and current pain related treatments (medication, supplements, physical/psychological/surgical interventions) were collected (see Appendix C).

## Data Analysis

Exploratory Factor Analysis (EFA) was used to test the underlying organizational hierarchy of the SPTS using the statistical software R (Version 3.1.2) Paramap Package, Version 1.0 (O'Connor, 2015). All other analyses were conducted using IBM SPSS Statistics, Version 22.0 (IBM Corporation, 2013). Reliability was tested using Cronbach's alpha, corrected item-total and Cronbach's alpha-if-item-deleted correlations. Construct validity (convergent and discriminant) was evaluated using Spearman's correlations, and by testing the equality of the convergent and discriminant validity correlation coefficients using a computer software program, *Calculation for the Test of the Difference between two Dependent Correlations with one Variable in Common* (Lee & Preacher, 2013).

### *EFA*

Exploratory Factor Analysis (EFA) was selected rather than Confirmatory Factor Analysis (CFA) to evaluate the underlying factor structure of the 12-SPTS items. CFA seeks to verify a theoretical model (Floyd & Widaman, 1995) whereas EFA aims to reveal an underlying structure (Fabrigar, Wegener, MacCallum, Strahan, 1999). As the SPTS has not been previously used in a clinical population, EFA was deemed the most appropriate procedure. The analysis followed current recommendations for EFA of ordinal data (Costello et al., 2005).

EFA was conducted on the matrix of polychoric correlations of the 12-SPTS items using Maximum Likelihood Factor Analysis (ML) for factor extraction. Parallel Analysis (PA) and Velicer's Minimum Average Partial Test (MAP) were conducted on the

correlation matrix to verify the number of factors to retain. As well, Cattell's Scree Test and eigenvalues greater than 1 were examined to inform factor retention.

ML is the recommended method of factor extraction, which is appropriate unless the assumptions of normality are severely violated (Fabrigar et al., 1999). Polychoric correlations are recommended over Pearson correlations for ordinal data (O'Connor, 2015). There is a lack of consensus in the field on the best method to determine the number of factors to retain in EFA; examining eigenvalues associated with the dataset is generally considered the rule of thumb (Costello et al., 2005; Fields, 2009). Typically, selecting factors that have eigenvalues above 1 (Kaiser, 1960) is standard, but the cut-off can vary (e.g. Jolliffe, 2002). This method has been critiqued as different criteria yield different results and eigenvalues are influenced by the number of variables (Fields, 2009). Additionally, some authors have pointed out that eigenvalues of 1 or lower account for the same amount of variance as a variable itself, which diminishes the goal of factor analysis: to reduce the several variables to higher-order factors (Nunally & Berstein, 1994). Examining eigenvalues is now considered one of the least accurate methods (Costello et al., 2005; Velicer & Jackson, 1990).

Among the recommended methods to determine the number of factors to retain are Scree Plots (Cattell, 1966), Parallel Analysis (PA; Longman, Cota, Holden, & Fekken, 1989; Ruscio & Roche, 2012) and Velicer's Minimum Average Partial test (MAP; Velicer, 1976; Gorsuch, 1997) (Costello et al., 2005). In a Scree Plot, eigenvalues are potted against the factors and the inflection point on the curve is considered the cut-off point; factors before are retained, and those after it are discarded (Cattell, 1966).

PA and MAP tests are considered ideal methods of identifying the number of factors to retain (Costello et al, 2005; Schmitt, 2011), thus they were used in the present analysis. The PA procedure compares the eigenvalues extracted using a Principal Component Analysis (PCA) to the mean and percentile of eigenvalues extracted from a bootstrapping procedure of randomly generated datasets from the available data that have the same characteristics as the dataset. When the eigenvalue of a factor obtained from the PCA exceeds the eigenvalue of the sample mean and percentiles randomly generated through the bootstrapping procedure, the factor is retained.

In the MAP test a PCA is also performed. First the principal component is partialled out from the correlation between the variables. The average squared correlation coefficient is computed from the remaining partial correlation matrix. This process is repeated for each successive component identified (up to *k*-1 steps, where k is the number of variables). The average squared partial correlation from the steps is compared. The step number with the lowest average squared partial correlation determines the number of factors.

Maximum Likelihood Factor Analysis (ML) using polychoric correlations was performed on the 12-items of the SPTS. PA (using PCA, 100 datasets from bootstrapping, and the polychoric correlation matrix) and MAP (using PCA and the polychoric correlation matrix) were used to determine the number of factors to retain using the R Paramap Package syntax (O'Connor, 2015). In addition, the Scree Test and eigenvalues were examined.

*Reliability*

The internal consistency of the SPTS was evaluated using unstandardized Cronbach's alpha and item-total correlations. The use of the unstandardized Cronbach's alpha is appropriate when items of a scale are summed to produce a single score (Fields, 2009). A Cronbach's alpha level of 0.7 is typically selected as the significance level for psychological constructs (Kline, 1999), and will be used in the present analysis. Corrected item-total correlations represent the correlation between a specific item and the total scale score. Item-total correlations less than 0.3 indicate poor correlation with the overall scale and should be removed (Fields, 2009). In addition, Cronbach's alpha-if-item-deleted values were examined. If the Cronbach's alpha increases with the removal of any one item compared to the Cronbach's alpha level for the entire sample of 12-items, this would suggest that the scale would be more reliable without the item and the item should be removed.

*Convergent and Discriminant Validity*

Convergent validity was evaluated by correlating SPTS total scores with the PCL-C using Spearman's correlation coefficient. The PCL-C measures a construct that is theoretically related to the SPTS, Post-traumatic Stress Disorder symptoms, but is unrelated to pain. A high correlation between the SPTS and the PCL-C was defined as ($r > 0.60$) which would indicate adequate convergent validity.

The correlation between total SPTS scores and pain intensity ratings were conducted using Spearman's correlation coefficients. This correlation was examined both using average chronic post-surgical chest pain (CI-NRS) and current post-surgical

chronic pain (S-LANSS). The strength of the relationship between the variables was measured by the magnitude of Spearman's correlation coefficient.

Discriminant validity was evaluated by correlating SPTS total scores with the HADS-D using Spearman's correlation coefficient. Depression is a theoretically distinct construct from the SPTS and the pain-related anxiety concepts that informed the development of the SPTS scale. Low to moderate correlations ($r < 0.60$) between SPTS total and HADS-D were taken to indicate adequate discriminant validity.

Convergent and discriminant validity were also assessed using a computer software program: *Calculation for the Test of the Difference Between two Dependent Correlations with one Variable in Common* (Lee et al., 2013). This statistical software performs a test of the equality of two correlation coefficients, which are obtained from the same sample and that share one variable in common. The product of the computation is a *z*-score which is then compared to the normal distribution. Scores greater than |1.96| are considered significant using a 2-tailed test. This test allows for a comparison between the correlations computed to assess convergent validity ($r_{SPTS/PCL\text{-}C}$) with the correlation computed to assess discriminant validity ($r_{SPTS/HADS\text{-}D}$) to determine if their difference is statistically significant. Finding a significantly larger correlation between the coefficients assessing convergent validity compared with the one assessing discriminant validity would support good convergent and discriminant validity.

*Sample Size*

Disagreement exists on how to assess for adequate sample size in factor analysis, and several authors have pointed to the lack of generally accepted guidelines (Costello et al., 2005; Fields, 2009; Schmitt, 2011). Recommendations are often based on either case

number alone or on subject-to-variable (STV) ratios. Highly inconsistent figures have been put forward within both approaches. On the low end, it has been recommended that for reliable results the STV ratio should be 5 times the number of variables (Bryant & Yarnold, 1995) and a minimum of 100 subjects (Gorsuch, 1983) or the greater of the two figures (Hatcher, 1994).

However, some researchers have called for a nuanced approach to evaluating sample size requirements for factor analysis. In a simulation study by Guadagnoli and Velicer (1988) it was found that regardless of sample size, if a factor is represented by at least 4 variables and these variables have loadings greater than 0.6, it is reliable. Velicer and Fava (1998) also recommended considering the number of variables defining each factor and number of factor loadings to determine adequate sample size. In an empirical study by MacCallaum, Widaman, Zhang, & Hong (1999) it was suggested that sample size requirements depend on communalities and number of factors, such that under ideal conditions of high communalities (over .60) and few factors (e.g. 3 factors) defined by multiple test variables (e.g. 6) a small sample (e.g. 60) would be sufficient. The general recommendations reviewed above and those that highlight the specific characteristics of the dataset were considered for the present analysis (see Results section).

## Results

### *Data Preparation*

Prior to conducting the statistical analysis, the dataset was inspected for missing values, data integrity, and violation of the assumption of normality as relevant to the proximate analysis. The sample used for the analysis consisted of 104 participants, after removing 4 participants due to missing values from the SPTS scale (3 participants were

missing one-item each, 1 participant was missing all 12-SPTS items). The results of the tests of normality are reviewed at the outset of each of the sections of the analyses.

*Participant Characteristics*

Demographic and clinical variables are summarized in Table 1. The majority of the participants were male (n = 74, age 25 to 82 years [*M* = 60.99, *SD* = 11.24]) and the minority female (women: n = 30, age 27 to 81 years [*M* = 64.10, *SD* = 12.12]). The mean age of the sample was 61.88 years (*SD* = 11.53). The majority of participants self-identified as Caucasian (53.8%), with the minority identifying as other specific groups (5.8%; e.g. Asian, Black, Hispanic), a smaller group selected "other" (4.8%), and many participants did not provide ethnicity data (33.7%). Twenty three percent (23.1%) of the sample reported that the CABGS procedure was their first surgery, and 18.3% reported an intervening surgery of various types between the CABGS and the psychometric assessment. Mean time since surgery was 33.53 months (SD = 23.20). There were no correlations between time since surgery and the measures of interest.

Post-surgical pain variables are listed in Table 2. In describing their average post-surgery chest pain (pain lasting at least three months), participants reported an average CI-NRS of 3.47 (SD=2.49) on a 0-10 scale. The majority reported the duration of a pain episode lasting seconds or minutes (49%), or hours (15.4%). Fewer reported their pain lasting days (6.7%), or weeks or months (4.8%). However several participants reported their pain as constant (14.4%). In terms of frequency of pain episodes, most reported experiencing pain weekly (42.3%), and fewer hourly (26.0%), monthly (15.4%), or every 3-6 months (6.7%). Most participants reported that their pain was still present (54.8%), with less reporting it lasting for 6 months (25%) or 1-year (10%).

A large minority of participants reported that the surgeon used a vein from their leg during the CABG surgery (42.3%). Of the total sample, 19.2% of participants reported they were experiencing chronic leg pain resulting from their operation, 23.1% reported no chronic pain from the leg surgery, and 57.7% of the did not have leg surgery.

In terms of current pain intensity (see Table 3), participants reported an average S-LANSS of 3.41 (SD=2.27) on an 11-point scale (men [$M = 3.07$, $SD = 1.80$] and women [$M = 4.02$, $SD = 2.87$]). Considering the entire sample, a minority of participants were currently taking pain medication at the time of the survey (36.5%). However, the majority of women (60%) reported currently using medication, while the minority of men (27%) reported the current use of medication. Similarly, while about half of the sample reported other ongoing pain problems unrelated to their surgery (49%), the majority of women (70%) and the minority of men (40.5%) reported the presence of other pain. A significantly higher proportion of women than men reported the current use of pain medications ($z=-3.16$, $p<.002$) and the presence of other pain conditions ($z=-2.72$, $p<.007$).

Tables 4 shows means, standard deviations, medians, score ranges, and inter-quartile ranges for the SPTS, PCL-C, HADS-D, CI-NRS, and S-LANSS by sex and across the sample.

Total scores on the SPTS ($r =.007$, $p = .944$), PCL-C ($r = -.014$, $p=.89$), CI-NRS ($r = -.105, .291$), and S-LANSS-NRS ($r = .100$, $p = .424$) did not significantly correlate with age (see Table 9). The HADS-D ($r = .206$, $p = .036$) showed a low, significant, positive correlation with age. Total scores on the PCL-C ($r = .169$, $p = .087$), HADS-D ($r = .148$, $p = .133$), CI-NRS ($r = .173, .079$), and S-LANSS-NRS ($r = .105$, $p = .403$) did

not significantly correlate with sex (see Table 9). However, the SPTS ($r$ = .283, p = .004) showed a low significant positive correlation with sex. Mean SPTS scores were significantly higher in females (M = 13.07, SD = 9.14) then in males (M = 8.42, SD = 8.90) [t (102) = -2.394, p = .018].

Mean scores on the PCL-C (M = 29.40, SD = 11.54) were at the low-end of the clinical cutoff for symptoms severity score. According to the US Department of Veteran Affairs, National Centre for PTSD, for the civilian version of the PCL, clinical cut off scores for symptom severity range between 30 and 50 depending on the specific setting (e.g. in primary care settings severity cutoff score is lower than in trauma or substance rehabilitation settings) (Weathers, Litz, Keane, Palmieri, Marx, & Schnurr, 2013). Thirty five percent (35.1 %) of the sample scored above the low-end of the clinical cutoff score for symptom severity of PTSD symptoms in a civilian setting.

The measures of average and current pain approached the moderate to severe clinical cutoff [CI-NRS (M = 3.47, SD = 2.50); S-LANSS (M = 3.41, SD = 2.27)]. An NRS rating of 0-2 is considered mild; 3-4 moderate; and 5-10 severe, with pain at the moderate-severe threshold warranting treatment interventions (Gerbershagen, Rothaug, Kalkman & Meissener, 2011). Forty three (43.2%) of the sample scored at or above 4 for average chronic chest pain intensity on the CI-NRS, and 39.3% for current pain on the S-LANSS.

Fourteen percent (14.4%) of the sample scored above the range of at least sub-threshold symptoms of depression on the HADS-D, 6.7% above the clinic cutoff for a Major Depressive Episode (MDE) (>=8 or >=11, respectively; Zigmond et al., 1983; Bjelland et al., 2002).

Table 1.

*Demographic and Clinical Variables*

| | Total |
|---|---|
| *n (%)* | 104 (100) |
| Male | 74 (71.2) |
| Female | 30 (28.8) |
| *Age, M (SD)* | 61.88 (11.53) |
| Male | 60.99 (11.24) |
| Female | 64.10(12.12) |
| *Ethnicity, n (%)* | |
| Caucasian | 56 (53.8) |
| Asian | 1 (1.0) |
| Black | 4 (3.8) |
| Hispanic | 1 (1.0) |
| Other | 5 (4.8) |
| Unknown | 35 (33.7) |
| *CABG First Surgery, n (%)* | |
| Yes | 24 (23.1) |
| No | 80 (76.9) |
| *Subsequent Surgery, n (%)* | |
| Yes | 19 (18.3) |
| No | 85 (81.7) |
| Time Since Surgery, M (SD) | 33.53(23.20) |

Table 2.

*Post-Surgical Pain Variables*

| | Total |
|---|---|
| Average Pain Lasting > 3 Months | |
| *Cardiac Pain Intensity, M (SD)* | 3.47 (2.49) |
| *Chronic Pain Duration Chest, n (%)* | |
| Sec/Minutes | 51 (49.0) |
| Hours | 16 (15.4) |
| Days | 7 (6.7) |
| Weeks/Months | 5 (4.8) |
| Constant | 15 (14.4) |
| *Chronic Pain Frequency Chest, n (%)* | |
| Hourly | 27 (26.0) |
| Weekly | 44 (42.3) |
| Monthly | 16 (15.4) |
| 3-6 Months | 7 (6.7) |
| *Chronic Pain Length Chest, n (%)* | |
| 6 months | 26 (25.0) |
| 1 year | 11 (10.6) |
| Still present | 57 (54.8) |
| *Leg Surgery with CABG, n (%)* | |
| Yes | 44 (42.3) |
| No | 60 (57.7) |
| *Chronic Pain from Leg Operation, n (%)* | |
| Yes | 20 (19.2) |
| No | 24 (23.1) |

Table 3.
*Current Pain Variables*

| | Men | Women | Total |
|---|---|---|---|
| Current Pain | | | |
| *Current Cardiac Surgery Pain (S-LANSS), M (SD)* | 3.07 (1.80) | 4.00 (2.87) | 3.41 (2.27) |
| *Current Pain Medication, n (%)** | | | |
| Yes | 20 (27.0) | 18 (60.0) | 38 (36.5) |
| No | 54 (73.0) | 11 (36.7) | 65 (62.5) |
| *Other Chronic Pain, n (%)** | | | |
| Yes | 30 (40.5) | 21 (70.0) | 51 (49.0) |
| No | 43 (58.1) | 7 (23.3) | 50 (48.1) |

*Note:* * denotes significant difference between sexes, $p < 0.01$ (2-tailed).

Table 4.

*Means, SD, median, score range, and inter-quartile range, of the, SPTS, PCL-C, HADS-D, CI-NRS and S-LANSS*

| | Men | Women | Total |
|---|---|---|---|
| *SPTS** | | | |
| Mean | 8.42 | 13.07 | 9.76 |
| SD | 8.90 | 9.14 | 9.17 |
| Median | 5.00 | 10.50 | 7.50 |
| Min-Max | 0-48 | 2-35 | 0-48 |
| IQR | 11.0 | 12.0 | 11.0 |
| *PCL-C* | | | |
| Mean | 28.28 | 32.17 | 29.40 |
| SD | 11.08 | 12.40 | 11.54 |
| Median | 24.50 | 28.50 | 26.00 |
| Min-Max | 17-65 | 17-62 | 17-65 |
| IQR | 14.00 | 16.75 | 15.75 |
| *HADS-D* | | | |
| Mean | 3.37 | 4.13 | 3.59 |
| SD | 3.54 | 3.49 | 3.52 |
| Median | 2.00 | 3.50 | 2.00 |
| Min-Max | 0-14 | 0-14 | 0-14 |
| IQR | 4.00 | 3.25 | 4.00 |
| *CI-NRS* | | | |
| Mean | 3.18 | 4.20 | 3.47 |
| SD | 2.34 | 2.73 | 2.50 |
| Median | 3.00 | 4.00 | 3.00 |
| Min-Max | 0-10 | 1-10 | 0-10 |
| IQR | 3.00 | 4.00 | 3.00 |
| *S-LANSS* | | | |
| Mean | 3.07 | 4.00 | 3.41 |
| SD | 1.80 | 2.87 | 2.27 |
| Median | 3.00 | 4.00 | 3.00 |
| Min-Max | 0-8 | 1-10 | 0-10 |
| IQR | 2.00 | 6.00 | 3.00 |

*Note:* * denotes significant difference between sexes, $p < 0.02$ (2-tailed).

*EFA*

For the EFA procedure, the sample size of 104 was deemed sufficient given it meets the basic requirements of a STV ratio of 5 (Bryant et al., 1995) and the recommended 100 minimum number of subjects (Gorsuch, 1983). Following the EFA procedure, the analysis yielded results which aligned with Guadagnoli and Velicer (1988) and Velicer and Fava's (1998) recommendations as the data was represented by few factors (e.g. 1), each factor defined by multiple test variables (e.g. 12), and with one exception had factor loadings which exceeded .6 (0.551 to 0.886; see Table 7).

ML is considered ideal unless the assumptions of normality are severely violated (Costello et al., 2005; Fabrigar et al., 1999). The criteria recommended for evaluating such a violation is skewness >2 and kurtosis >7 (West, Finch, Curran, 1995). The distributions of the 12-SPTS items were examined for normality, and found to be right skewed (see *Reliability* section below). Skewness scores for the 12-SPTS items ranged from .298 to 2.506, and kurtosis scores from -1.126 to 5.125. Given that no one item exceeded the dual cutoff and only 3 of the 12-SPTS items were only slightly above 2 it was deemed appropriate to proceed with the analysis. In addition, PA with polychoric matrix is robust to skewness (Garrido, Abad, & Ponsoda, 2013).

Following the procedure outlined in the data analysis section, both PA and MAP were run on the 12–item SPTS and the results compared. Results of the PA suggested a 1-factor solution (see Table 5). In the MAP analysis, the second step had the lowest average squared partial correlation (see Table 6), however the revised MAP test (2000) suggested a 1-factor solution. In addition, considering the rule-of-thumb for determining

the number of factors to retain, the eigenvalues from the PA and MAP tests were considered (see Table 5) and a Cattell's Scree plot was examined (see Figure 1).

In both PA and the MAP test the first eigenvalue far exceeds 1 (7.23), while the second is slightly above 1 (1.18). It is clear from both analyses that the contribution of the second and succeeding factors is small compared with the first. This trend is visually represented by Cattell's Scree plot of the eigenvalues (see Figure 1). We can see that the contribution of the first factor is considerable higher and then a sharp slope connects the first to the second value. Following Cattell's inflection point cut-off, we would clearly retain the first factor and discard the rest.

Table 7 shows the factor loadings of the 12-items SPTS for the MAP analysis. The 1-factor solution suggested by the MAP test explains 56.9% of the variance.

Table 5.

*MAP and Parallel Analysis Eigenvalues from PCA and bootstrapping (Mean, Percentile) using the PA procedure*

| Step | PCA Eigenvalue | Mean | Percentile |
|---|---|---|---|
| 1 | 7.23 | 1.58 | 1.73 |
| 2 | 1.18 | 1.42 | 1.51 |
| 3 | 0.78 | 1.30 | 1.38 |
| 4 | 0.71 | 1.20 | 1.27 |
| 5 | 0.53 | 1.11 | 1.17 |
| 6 | 0.46 | 1.01 | 1.07 |
| 7 | 0.38 | 0.93 | 0.99 |
| 8 | 0.29 | 0.85 | 0.91 |
| 9 | 0.20 | 0.77 | 0.83 |
| 10 | 0.11 | 0.70 | 0.76 |
| 11 | 0.10 | 0.61 | 0.68 |
| 12 | 0.03 | 0.52 | 0.58 |

*Note.* Step 1 PCA eigenvalue exceeds generated values suggesting factor retention.

Table 6.

*MAP Average Squared Correlations*

| Step | Average Squared Correlation |
|---|---|
| 0 | 0.32966 |
| 1 | 0.06216 |
| 2 | 0.06127 |
| 3 | 0.07746 |
| 4 | 0.09557 |
| 5 | 0.12732 |
| 6 | 0.14697 |
| 7 | 0.21137 |
| 8 | 0.24677 |
| 9 | 0.34037 |
| 10 | 0.51497 |
| 11 | 1.00000 |

*Note.* Step 2 has the smallest average squared correlation.

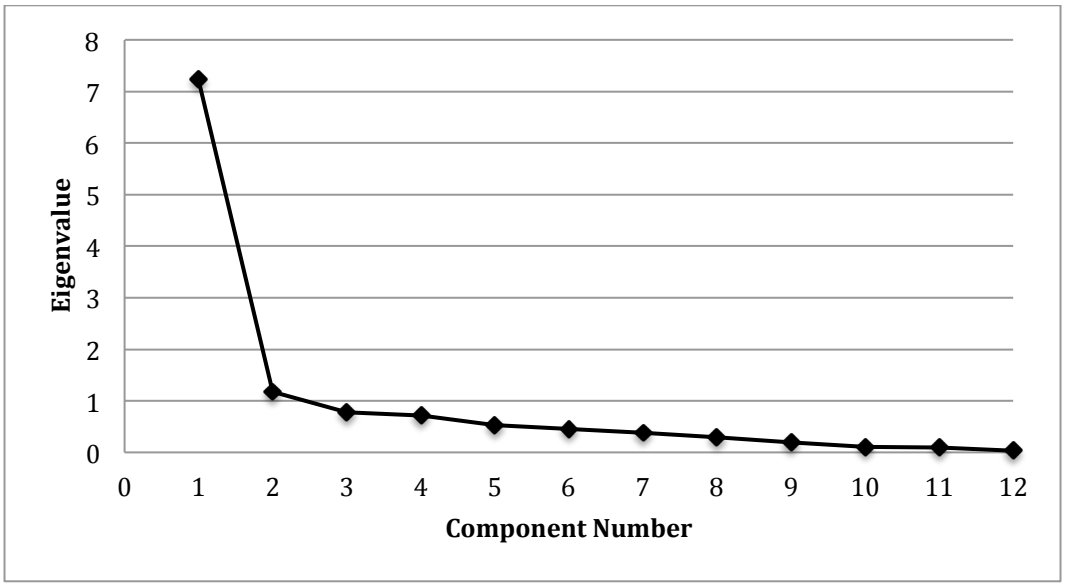


*Figure 1*. Cattell's Scree Test with MAP eigenvalues suggesting a 1-factor (component) solution.

Table 7.

*Factor Loadings Extracted by ML*

| SPTS Item # | Factor Loading | Communalities |
|---|---|---|
| 1 | 0.618 | 0.382 |
| 2 | 0.885 | 0.783 |
| 3 | 0.700 | 0.490 |
| 4 | 0.778 | 0.605 |
| 5 | 0.861 | 0.741 |
| 6 | 0.793 | 0.629 |
| 7 | 0.739 | 0.546 |
| 8 | 0.835 | 0.697 |
| 9 | 0.551 | 0.304 |
| 10 | 0.777 | 0.604 |
| 11 | 0.784 | 0.615 |
| 12 | 0.654 | 0.428 |

*Reliability*

The 12-items of the SPTS were tested for normality using the Kolmogoroz-Smirnov and Shapiro-Wilk test. SPTS scores were non-normally distributed, significant at the $p < .001$ level. In a normal distribution, values of skewness should be zero. However, the 12-SPTS items range from SK = .677 to 2.506 (*SE* = .237). A visual inspection of the Frequency and Q-Q plots demonstrates the positive skew of the distributions. This indicates that the majority of participants favored the low/moderate range of the scale (e.g. “Not at all true” and “Slightly true”).

Within the literature, disagreement exists whether the assumption of normality must be met for reliability analysis with Cronbach’s alpha (Sheng & Sheng, 2012). The normality assumption is often considered unrealistic for latent variables in psychological research (Woods, 2008), and normalizing data makes the results difficult to interpret

(Fields, 2009). For these reasons, the analysis was conducted without normalizing the data.

Unstandardized Cronbach's alpha coefficients were used to estimate the reliability of the SPTS. The SPTS showed excellent overall internal consistency ($\alpha$ = .908). Item-deleted Cronbach's alpha and corrected item-total correlations are presented in Table 8. Deletion of any single item did not significantly affect the internal consistency of the overall scale ($\alpha$ = .893 – .906). Corrected item-total correlations ranged from .496 to .785. Despite having a low number of women in the study, the reliability analysis was also run separately for men (n = 74) and women (n = 30). The SPTS showed excellent internal consistency for both groups (men: $\alpha$ = .922, women: $\alpha$ = .860).

*Validity*

Prior to the validity analyses, the assumption of normality was tested for all of the variables included: SPTS Total Score, PCL-C Total Score, HADS Depression Subscale Score, CI-NRS Score, and S-LANSS. The Kolmogoroz-Smirnov and Shapiro-Wilk tests determined that all total scale and subscale scores were non-normally distributed ($p \leq$ .01). A visual inspection of the frequency plots and Q-Q plots describe the positive skew of the distributions (*SK* = .651 – 1.500, *SE* = .237-.295). This indicates that the majority of participants selected answers from the low/moderate range of the scales.

The Spearman's correlation coefficient (Spearman, 1910) is a non-parametric statistic that measures of the strength of the linear relationship between two variables. It is appropriate when violations of the normality exist or for correlations including categorical variables (Fields, 2009). For the subsequent correlation analyses, Spearman's correlation coefficients ($r_s$) were used.

When considering whether to run partial correlations to control for the effects of sex and age on the variables of interest, a correlation matrix was examined to determine the contribution of sex and age to the validity analysis (see Table 9). The correlation between sex and age with the other variables was either non-significant or low (e.g. $r_{S_{Sex\&SPTS}} = .283, p = .004$; $r_{S_{Age\&HADS\,Dep}} = .206, p = .036$). Due to the low or insignificant correlations, as well as the unequal sample sizes it was decided not to partial out for age and sex in the validity analysis. Table 9 provides a summary the correlation statistics for all measures.

Convergent and Discriminant Validity

The correlations between the 12-item SPTS Total Score and the PCL-C ($r_S = .595, p < .001$), support preliminary convergent validity. The correlations between the 12-item SPTS Total Score and the S-LANSS ($r_S = .383, p = .002$) was in the moderate range, while the correlation between the 12-item SPTS Total Score and CI-NRS was non-significant ($r_S = .170, p = .084$). This suggests that current pain ratings, but not average pain ratings are correlated with SPTS total scores. Looking at current pain ratings and SPTS Total scores, high pain raters (S-LANSS Score >= 4) compared with low pain raters (S-LANSS-NRS Score < 4) had significantly higher SPTS scores (high: M = 15.54, SD=12.55; low: M = 7.68, SD = 6.51; t(33.9) = -2.947, p = .006) (see Table 10).

The 12-item SPTS Total Score correlated to a lesser extent with the HADS-D ($r_S = .41,\ p < .001$) than the PCL-C. Convergent and discriminant validity was also assessed by testing the equality of the two correlation coefficients ($r_{SPTS/PCL\text{-}C}$ vs. $r_{SPTS/HADS\text{-}D}$) (Lee et al., 2013). Results indicated that the magnitude of the correlation between the SPTS and PCL-C is significantly greater than with the HADS-D (z = 2.32, p

= .02), supporting good convergent and discriminant validity. This suggests a significantly greater overlap between the SPTS and a measure of PTSD symptoms, than with a measure of depression symptoms.

Table 8.

*Reliability Analysis Item-Total and Alpha-if-Item-Deleted Statistics*

| SPTS Item # | Corrected Item-Total Correlation | Cronbach's Alpha if Item Deleted |
|---|---|---|
| 1. Pain keeps me awake at night. | .538 | .906 |
| 2. When I am in pain, everything I see or do reminds me of the pain. | .785 | .893 |
| 3. I try to avoid activities that cause pain. | .621 | .902 |
| 4. When I feel pain, I'm scared that it's the beginning of a terrible problem. | .649 | .900 |
| 5. Pain seems to bother me more than it does other people. | .689 | .898 |
| 6. When I feel pain, I think about it even when I don't mean to. | .703 | .897 |
| 7. I can't stand pain. | .662 | .899 |
| 8. When I'm in pain, I feel distant from people even when I'm talking to them. | .749 | .895 |
| 9. As soon as the pain comes on, I take medications to reduce it. | .496 | .907 |
| 10. Pain sensations terrify me. | .630 | .901 |
| 11. When I'm in pain, things don't feel real. | .649 | .900 |
| 12. I feel sick to my stomach when I am in pain. | .565 | .904 |

Table 9.

*Correlation coefficients between sex, age, SPTS, PCL-C, HADS-D, and CPI.*

| | Age | Sex | SPTS | PCL-C | HADS-D | CI-NRS | S-LANSS |
|---|---|---|---|---|---|---|---|
| Age | | | | | | | |
| Sex | .150 | | | | | | |
| SPTS | .007 | .283** | | | | | |
| PCL-C | -.014 | .169 | .595** | | | | |
| HADS-D | .206* | .148 | .408** | .510** | | | |
| CI-NRS | -.105 | .173 | .170 | .203* | .001 | | |
| S-LANSS | .100 | .105 | .383** | .368** | .224 | .661** | |

*Note:* All correlation coefficients are measured with Spearman's Rho.
* $p < 0.05$ (2-tailed).
** $p < 0.01$ (2-tailed).

Table 10.

*STPS Scores of High vs. Low Pain Raters*

| | Total | |
|---|---|---|
| *S-LANSS Pain Group* | Low | High |
| *SPTS* | | |
| Mean | 7.68 | 15.54 |
| SD | 6.51 | 12.55 |
| Median | 6.00 | 11.50 |
| Min-Max | 0-26 | 1-48 |
| IQR | 11 | 18 |

*Note.* Chronic Pain Group was split at the median L-SLANSS score to form a "Low" and a "High" group.
* denotes significant difference between pain rater levels, $p < 0.01$ (2-tailed).

# Discussion

The aims of the present study were to evaluate the factor structure, reliability, and validity of the preliminary version of the 12-item SPTS in a sample of patients who had (or had had) chronic pain after undergoing CABGS a minimum of 6 months earlier. Results of the EFA suggested a one-factor solution for the 12-SPTS items. All four methods used to evaluate the number of factors to retain suggested a one-factor solution was the best fit to the data (PA, MAP Revised Test [2000], Cattell's Scree Plot, Eigenvalues > 1). This factor includes all 12-SPTS items and explains 56.9% of the variance.

These results mirror the findings from the preliminary study on the development of the 12-item SPTS in which an EFA, using undergraduate students as participants, yielded a one-factor solution that explained 53.7% of the total variance (Roosen, 2009). The one-factor solution is also consistent with the findings from preliminary research on the SPT construct in which using a higher-order analysis identified a single factor which explained 68.3% of the common variance underlying the major pain-related anxiety constructs (Kleiman et al., 2011). Taken together, these findings suggest that the Sensitivity to Pain Traumatization construct, as measured by the SPTS, represents a higher-order factor which describes a predisposition to respond to pain with somatic, cognitive, emotional, and behavioral response that are similar to a traumatic reaction (Kleiman et al., 2011). These findings support previous research that has suggested a higher-order factor underlies the pain-related anxiety constructs (e.g. Keogh et al., 2004).

Results of the reliability analysis suggest the SPTS has excellent overall internal consistency. Deletion of any one item did not improve the internal consistency of the scale, indicating that each item is consistent with the reliability of the overall scale. The moderate to high corrected item-total coefficients suggests that all SPTS items relate strongly to the construct of SPT. The high level of internal consistency suggests that the SPTS is reliable for use in a clinical setting. These findings concur with the initial study which tested the SPTS in a community setting of university students (Roosen, 2009) and found high internal consistency of the overall scale ($\alpha = 0.9167$) and that deletion of any one of the 12-SPTS items did not improve the overall reliability of the scale (Roosen, 2009). Taken together, this provides preliminary evidence that the scale is reliable for use in various settings.

In the present study, the SPTS also demonstrated excellent internal consistency for both men and women. However, due to the small number of women in the study this result should be interpreted tentatively especially in light of the almost ubiquitous findings that women and men differ in terms of their responses to acute experimental (Bartley & Fillingim, 2013; Cruz-Almeida, Riley & Fillingim, 2013), clinical (Tighe, Riley & Fillingim, 2014) and chronic pain (Tsang et al., 2008). Reliability within subgroups such as sex should be examined in future studies with larger samples from various settings.

Examination of the convergent validity of the SPTS showed a moderate to high correlation with PTSD symptomatology as measured by the PCL-C. Additionally, the SPTS was moderately correlated with current pain ratings as measured by the S-LANSS. The results of the analysis of discriminant validity yielded a lesser correlation between

the 12-item SPTS and depressive symptomatology as measured by the HADS-D compared to its correlation with PTSD symptoms. A significance test of the magnitude of the difference between the correlation coefficients representing convergent (SPTS/PCL-C) and discriminant validity (SPTS/HADS-D) demonstrated a significant difference in the magnitude of these relationships. This suggests that the SPTS shares significantly more variance with a measure of PTSD symptoms, than with a measure of depressive symptoms. Taken together, the results of the validity analysis suggest good preliminary construct validity of the 12-item SPTS. This also suggests that despite the variable overlap between the SPT construct with PTSD and pain these constructs are distinct.

In sum, the results of the present study supported the initial hypotheses. The SPTS was found to have a one-factor structure, which was consistent with results of the prior EFA analyses of Kleiman et al. (2011) and Roosen (2009). The SPTS demonstrated good preliminary psychometric properties including high internal consistency, and good preliminary construct (convergent and discriminant) validity.

The remainder of the present discussion is organized into nine sections. In Section 1, the findings regarding pain are examined and the psychometric challenges of measuring pain are discussed. In Section 2, the main findings pertaining to the SPTS scale are reviewed within the context of the development of the SPT construct. In Sections 3 – 5 the relationship between the SPTS and pain, PTSD, and sex are discussed respectively. Section 6 evaluates limitations associated with the present study. In Section 7, recommendations for future directions of the SPTS scale are reviewed. Section 8 discusses the implications of the present study and its contribution to research in this

field. In the final section, the discussion will conclude with a summary of the present study.

*1. Psychometric Challenges, Findings, and Comorbidities related to Pain*

Chronic pain post-surgery is a considerable and pervasive concern, although it is under-acknowledged (Choinière, Watt-Watson, Costello, Guerriere, Carrier, Cogan, Bussieres, Guertin, Lespérance, Basket, McFetridge-Durdle, Sullivan, 2009). Prevalance rates of post-surgical chronic pain resulting from cardiac surgery vary from 18-61% (Gjeilo, Klepstad, Wahba, Lydersen, Stenseth, 2010). Further, recovery from CABGS is complex and informed by physical, psychological, and social factors, suggesting that normative healing trajectories may not apply to all individuals (Lopez, Ying, Poon, Wai, 2007). While the importance of studying post-surgical chronic pain is clear, the inherent challenges of studying the complex phenomenon of pain in this population are considerable.

The present study attempted to quantify the nature of pain through several target questions regarding pain experience. This required participants to distinguish acute from chronic pain and present chronic pain from pain history, as well as rate pain descriptors that aimed to capture objective (rather than subjective and experiential) information. Acute pain was operationalized as pain following surgery that reflected normal healing times while the process of recovery from surgery was still underway (e.g. under 3 months). In contrast, chronic pain was considered to be pain that exceeds this time limit and persists (e.g. pain beginning at least 3 months post-surgery and lasting greater than 3 months). This important distinction was aimed at separating pain that reflects tissue damage from pain that becomes its own disorder and perpetuates disability. The present

study emphasized these time frames when asking about all aspects of pain experience, to distinguish chronic pain from acute and historical pain issues. Pain location, intensity, frequency, duration, and length were collected in order to characterize the nature of the chronic pain.

Despite the specificity of the definitions used, participant's ability to report pain objectively and the limits of memory challenge these psychometric aims. While it is clear that acute peri-operative pain predicts the severity of post-operative chronic pain (Choinière et al., 2009), it is also well established that pain disability results from interpretation and adjustment to pain not only based on pain severity (Burns et al., 2015; McCracken, Spertus, Janeck, Sinclair & Wetzel, 1999; Turk & Wilson, 2010). The connection between participant reports of pain intensity (e.g. high and low pain raters) and SPTS scores in the present study raises the possibility that the impact of pain and perception of pain severity varies with individual sensitivity, thus resisting objective measure.

In kind, participants were asked to rate "average pain intensity" of pain beginning at least 3 months post-surgery and lasting 3 months or more. Such a measure relies on memory. Contextual features of assessment such as memory complicate measurement (Jensen & Karoly, 1995). An additional challenge of the present study was that that some participants were currently experiencing chronic pain at the time of assessment while for others, whose chronic pain had resolved, were reporting past chronic pain. It is possible that the experience of current pain may change the report of pain. In addition, there was great variation in the time between survey and assessment (i.e. 7 months to 7 years).

Although time since surgery was not correlated with the measures of interest, we do not know the effect of memory, or the presence or absence of current pain on memory.

In contrast to these challenges, the post-surgical sample also afforded distinct benefits to the present study. Surgery is a controlled trauma that allows the examination of pain among individuals who have shared a common tissue-damaging event. The focused nature of the surgical procedure allows for a comprehensive study of the inception and maintenance of chronic pain. However, the specificity of the trigger that the participants shared also harbors the possibility that the results of the present study describe the distinct trajectory of chronic pain in a CABGS post-surgical population. For this reason, future studies of post-surgical nature should assess additional populations or groups undergoing different procedures.

In the present study, mean scores of average and current pain approached the moderate to severe clinical cutoff warranting treatment interventions (e.g. NRS of 4; Gerbershagen et al., 2011). The majority of the sample characterized their pain at the time of assessment as lasting seconds or minutes, occurring weekly, and still ongoing. Surprisingly, there were no significant differences between men and women on pain ratings, in contrast to the literature (for review see Section 5). A minority of participants were taking pain medication at the time of the survey and about half of the sample reported other ongoing pain problems unrelated to their surgery. A significantly higher proportion of women than men reported the current use of pain medications and the presence of other pain conditions. Unfortunately, women were underrepresented in the current sample, and thus these results should be interpreted with caution. Researchers have suggested that we know much less about the surgery related pain and functional

status of women than men undergoing CABGS, thus sex differences in healing trajectories is an important area warranting future research (Routledge, Tsuyuki, Hervas-Malo, LeBlanc, McFetridge-Durdle, King, 2009).

Comorbid PTSD symptoms and PTSD as measured by PCL-C scores (i.e. 30 and 44, respectively; Weathers et al., 2013; Weathers, et al., 1993; Blanchard et al., 1996) were slightly higher than the typical prevalence rates in chronic pain samples. The prevalence rate of PTSD symptoms was 35.1% compared with 10-30% (Asmundson et al., 2002; McWilliams, Cox & Enns, 2003) and that of PTSD was 13.8% compared with 9.1% (see Section 4). Comorbid depression was lower than the prevalence of this disorder in chronic pain samples, 6.7% were above the clinic cutoff for a Major Depressive Episode (MDE) in the present sample compared with 12.6% (Von Korff et al., 2005). However, 14.4% of the sample had at least sub-threshold symptoms of depression on the HADS-D (i.e. >=8; Zigmond et al., 1983; Bjelland et al., 2002). Although the presence of psychiatric comorbidity was evaluated by self-report, these results suggest that PTSD was overrepresented while depression underrepresented in the current study.

*2. SPTS Main findings*

The development and validation of the SPTS represents an effort to clarify the nature of a higher-order factor that underlies the major pain-related anxiety constructs. The origin of the SPTS began with the finding of Kleiman et al. (2011) that one higher order factor described the shared variation of the major pain-related anxiety constructs as represented by Anxiety Sensitivity (Anxiety Sensitivity Index; ASI), Pain Anxiety (Pain Anxiety Symptoms Scale; PASS-20), and Pain Catastrophizing (Pain Catastrophizing

Scale; PCS). Kleiman and colleagues (2011) conducted a hierarchal analysis in two steps. In the first-order analysis, the initial EFA suggested three separate factors substantiating the construct independence of PA, PC, and AS. A second-order analysis revealed one factor on which 47 of the 49 items loaded, more strongly than the three lower-order factors. These findings suggested a bi-factor model best fit the pool of items, which both described the separate dimensions of AS, PA, and PC, and their underlying commonality.

This single underlying factor was termed Sensitivity to Pain Traumatization (SPT) to reflect the content of the items, which described somatic, cognitive, emotional, and behavioral responses to pain that are similar to a traumatic reaction (Kleiman et al., 2011). Interestingly, it was found that 20 of the items loaded significantly only on the general higher-order SPT factor. Thus, this item set was seen to better represent the higher-order factor than the first-order factors. These 20 items were classified to into 4 symptom groups: re-experiencing, avoidance, increased arousal, and other (hopelessness and social anxiety relating to physical symptoms). This initial pool of 20-items suggested strong similarity with PTSD symptomatology although the authors pointed out that the item pool, not originally aimed at measuring traumatic stress, was missing important groups of symptoms (e.g. emotional numbing; Kleiman et al., 2011).

Building on these findings, Roosen et al. (2009) methodically surveyed the research and clinical literature to develop a pool of items in which a traumatic stress reaction relevant to pain was represented comprehensively. The initial SPT scale was developed to include 6 subscales: Sensitivity to Pain, Fear of Pain, Pain Experiencing, Pain and Hyperarousal, Pain Avoidance, Pain and Emotional Numbing. Mirroring the

findings from Kleiman et al. (2011), Roosen (2009) found that all 12-SPTS items loaded on one factor.

In the present and third study on the SPT construct, the results of the EFA also support the existence of a cohesive factor that represents a propensity to respond to pain with a traumatic stress reaction. This was the first opportunity to test the SPTS in a clinical population and yielded promising preliminary results. The present EFA followed the current recommendations for EFA of ordinal data (Costello et al., 2005). Although the sample size was just above the minimum recommended number of subjects and subject-to-variable ratio (e.g. Bryant et al., 1995; Gorsuch, 1983; Hatcher, 1994), the data was represented by few factors (e.g. 1), each factor defined by multiple test variables (e.g. 12), and with one exception had factor loadings which exceeded .6, suggesting the analysis was reliable (Guadagnoli et al., 1988; Velicer et al., 1998).

The 1-factor solution was favored by all four of the methods used to determine factor retention (PA, MAP Revised Test [2000], Cattell's Scree Plot, Eigenvalues > 1). Examining the eigenvalues associated with the factors, it was clear that the first factor offered the greatest contribution to the model (i.e. was above 7), while subsequent factors contributed little additional variance (1.18 or below). Taken on the whole, the 1-factor solution that explains 56.9% of the variance in the 12-item SPTS can be considered a robust finding.

The internal consistency of the scale was supported by the reliability analysis ($\alpha$ = .908) suggesting the accuracy of the 12-item SPTS as a test instrument to measure the SPT construct. Each item supported the reliability of the overall measure: deletion of any item did not improve the internal consistency of the scale; and the moderate-to-high

corrected item-total correlations suggested that all SPTS items related strongly to the construct of SPT. These findings are similar to the original SPTS study by Roosen (2009) which also found excellent reliability and that all items supported the internal consistency of the scale ($\alpha = 0.918$). This suggests the robustness of the scale for use in a wide range of groups and settings.

Comparing between the initial (Roosen, 2009) and present SPTS study, while the samples sizes where almost identical (initial: n = 105; present n = 104), the samples differed greatly by age range, sex composition, pain histories, and setting. The initial SPTS study participants were young adults (men [*M* = 21.75 years, *SD* = 4.49] and women [*M* = 20.68 years, *SD* = 4.22]), whereas the present sample was composed of older aged adults (men [*M* = 60.99 years, *SD* = 11.24] and women [*M* = 64.10 years, *SD* = 12.12]). Women represented the majority in the initial study (80.8%) and the minority in the present sample (28.8%). The initial sample was a community sample of undergraduate student participants reporting mixed pain histories (24.8% reported having experienced chronic pain persisting for more than one month) whereas the present sample was composed of post-surgical patients who had all experienced pain persisting 3 months or longer post-surgery.

While the number of variables potentially impacting differences between the samples (e.g. age, sex, pain history, setting, etc.) precludes drawing inferences, the striking discrepancy between mean SPTS scores raises important questions regarding the SPT construct. On average, the undergraduate sample scored at the very low end of the scale (total possible score of 60; men [*M* = 1.65, *SD* = 0.46] and women [*M* = 2.39, *SD* = 0.95]). The clinical sample had much higher average scores (total possible score of 60;

men [$M = 20.42$, $SD = 8.90$] and women [$M = 25.07$, $SD = 9.14$])[1]. These initial findings raise the possibility that the prevalence of the SPT levels may vary greatly across different groups and settings, and may be related to chronic pain exposure.

If SPT is a vulnerability factor which predisposes individuals to develop chronic pain or extend pain chronicity, we would expect to find higher SPTS scores in chronic pain groups. Roosen (2009) did not find any differences in SPTS scores between the minority of participants who reported pain problems, chronic pain, or current pain as compared to the majority who did not. However, the overall low mean scores of the SPTS in the community sample compared with the higher scores of the current chronic pain sample suggest that the predisposition to react to pain with symptoms resembling a traumatic stress reaction may be seen at higher prevalence rates within chronic pain samples. This raises the question of whether the vulnerability described by SPT is responsible for individual pain levels, if the pain itself is triggering the SPTS reaction, or if both form a mutually maintaining system.

In addition, considering the SPT construct is measured on a spectrum, the variation between groups in the two studies highlights the importance of identifying clinically impactful levels of the SPT trait. It is possible that below a certain point, the range of SPTS scores represent typical variability in pain responses in the general population. This should be investigated through future studies including larger samples of different groups. One specific area of pertinent interest would be in clinical settings of a psychological nature, to observe SPT levels in a population with psychiatric

[1] Roosen (2009) used a 0-4 SPTS while the present study used a 1-5 SPTS. The scores from the current project are converted here for comparison

comorbidities given the association between SPT and psychological factors (e.g. the pain-related anxiety constructs) and disorders (e.g. PTSD).

*3. SPTS and Pain*

The main findings of the present study offer the opportunity to consider the theoretical relationship between chronic pain and the SPT construct. Current post-surgery pain intensity (e.g. as measured by the S-LANSS) had a low to moderate correlation with the 12-item SPTS ($r_S = .383, p = .002$), whereas a measure of average post-surgical chronic chest pain intensity (e.g.CI-NRS) was not significantly correlated ($r_S = .170, p = .084$). While the two pain scales are identical in their structure (11-point Numeric Rating Scale) one asks about current pain, the other average pain lasting 3 months or longer. The discrepancy between these measures of pain and relationship to the SPTS may be attributed to the different demands of the questions. While a measure of current pain relies on individual ability to assess one's current state, reporting average pain depends on memory and ability to generalize across time. Contextual features impacting the psychological assessment of pain, such as memory, can decrease the reliability of pain measurements (Jensen et al., 1995).

The magnitude of the correlation between the 12-item SPTS and current pain levels was lower than expected. However, high pain raters (S-LANSS Score >= 4) compared with low pain raters (S-LANSS-NRS Score < 4) had significantly higher SPTS scores (high: M = 15.54, SD=12.55; low: M = 7.68, SD = 6.51; t(33.9) = -2.947, p = .006). An NRS of four is the clinical cut-off for pain intensity, below which pain is considered mild, and above which is considered moderate-to-severe and warrants

treatment (Gerbershagen et al., 2011). Thus, in the present study higher mean SPTS scores are associated with participants who report pain above the threshold for moderate-to-severe pain requiring treatment compared with participants experiencing pain that is considered mild and manageable without treatment.

The distinction found between high and low pain raters is interesting considered alongside the results of the initial SPTS study. As discussed in Section 2, Roosen (2009) did not find a difference between SPTS total scores for those reporting pain problems (chronic pain or current pain) compared to those not reporting pain. However SPTS scores were significantly different across high compared with low pain raters, where groups were defined by participants' ratings of common pain problems (e.g. "toothache", "paper cut", "stubbed toe", etc.) on a 11-point NRS scale from 0 (not at all intense) to 10 (as intense as can be). "High pain raters" scored ≥ 5/10. While all participants rated the same items, those with higher mean scores on the 12-item SPTS reported the subjective experience of these common pain events as more intense. This suggests a relationship between subjective sensitivity to pain and the SPTS scale.

The association between high SPTS scores and higher pain ratings in Roosen's (2009) study supports the interpretation that the SPTS is measuring the greater physiological and psychological impact of pain on certain individuals. In Roosen's (2009) study, having participants rate everyday pains provided "pain anchors" by which the experience of an objective pain event could be compared with subjective experience (e.g. intensity). Those who rated a similar objective pain more intensely (e.g. paper cut) had higher SPTS scores, indicating they also experience pain more traumatically.

It is also possible that higher SPTS scores are linked to a greater subjective sensitivity to pain in the present study. While all participants in the present sample went through a similar surgical procedure, different levels (or absence) of chronic pain resulted. The higher mean SPTS ratings of those whose current pain was equal to or above the clinical threshold for treatment ("high pain raters") may suggest a propensity to experience an equivalent pain event more traumatically and possibly more intensely compared with others. Although it is not possible to comprehensively compare a highly complex and multifaceted phenomenon such as pain across individuals, the association between SPTS scores and higher pain ratings across the two studies may suggest that SPTS describes a disposition toward experiencing pain more with more cognitive, emotional, behavioral and somatic distress.

Considering participants who gave higher ratings on the SPTS, the item-content provides a picture of the experience of an individual with who is greatly impacted by pain: Sensitivity to Pain (e.g. "Pain seems to bother me more than it does other people"); Fear of Pain (e.g. "Pain sensations terrify me"); Pain Experiencing (e.g. "When I am in pain, everything I see or do reminds me of the pain"); Pain and Hyperarousal (e.g. "When I am in pain, it keeps me awake at night"); Pain Avoidance (e.g. "I try to avoid activities that cause pain"); Pain and Emotional Numbing (e.g. "When I'm in pain, I feel distant from people even when I'm talking to them").

Another possibility for interpreting the results of the present study is that more intense pain is simply more traumatic than less intense pain. Current pain level and pain history may have directly informed participants' SPTS scores. For example, an individual who had recently, or over their lifetime, experienced lower levels of pain (e.g. 0, 1, 2;

where zero equals no pain) may have only had pain experiences that were mild in intensity and manageable to them. If these participants answered the SPTS items based on their mild historical experience of pain it may not be associated with high SPTS scores. On the other hand, if an individual had, or was currently experiencing, severe pain closer to the maximum of the scale (e.g. 10 corresponding to "pain as severe as it could be") their experience may be more congruent with statements such as: "As soon as the pain comes on, I take medications to reduce it", "When I am in pain, it keeps me awake at night", and "I feel sick to my stomach when I am in pain". In other words for most people, more intense pain causes greater distress and disruption than does less intense pain.

Considering the two studies together it is hard to reconcile that the higher mean SPTS scores of high pain raters can be explained on the basis of greater somatic intensity alone. Roosen's (2009) findings discussed above do not support this interpretation, but instead suggest experiencing pain more intensely is influenced by a heightened sensitivity to pain. Similarly, Kleiman and colleagues (2011) found that pre-surgery SPT scores differentiated persistent pain (ongoing pain problems, interference of pain daily life, no subsequent surgeries) from no-pain groups (no ongoing pain problems and no history of pain) at the one year post-surgery follow-up ($t[288]=2.27$, $p = .02$), connecting a preexisting vulnerability to traumatic reactions to pain to the later development of pain problems. It is critical that future studies involving the SPTS examine the SPTS predisposition prior to a trauma, such as surgery, so that the relationship of the SPTS vulnerability to pain and pain chronicity can be clarified.

Regardless of whether higher pain ratings are due to the objective pain or sensitivity toward experiencing greater impact from pain, the distressing reaction to pain indicated by higher SPTS scores is traumatic in itself and could perpetuate the experience of pain. The avoidance, rumination, physiological dysregulation, fear and dissociative features that the SPTS describes are closely related to the various pain-related anxiety constructs which have been linked in the literature to extending the chronicity of pain and ultimately pain-related disability (McCracken et al., 1992; Sullivan et al., 1998; Vlaeyen et al., 1995; Zvolensky et al., 2001). The Kleiman et al. (2011) study examined an initial definition of the SPT construct, which was composed of 20 items from the main pain-related anxiety scales (e.g. ASI, PA, and PCS), directly relating the SPT construct to these psychological measures.

Taken together, the present study offers preliminary evidence to suggest that the SPTS differentiates between high and low pain scores. This complements the findings of Kleiman et al. (2011), which show the SPT construct differentiates pain groups pre- and post-surgery. Importantly, the results of the present study are also compatible with the preliminary finding of Roosen (2009) that the SPTS may differentiate between those more or less sensitive to the experience of pain. The relationship between the SPTS and severity of reactions to pain highlight the potential clinical utility of this scale. In the present study, the high and low pain groups are separated based on clinical cutoffs for pain treatment. If higher pain ratings are, in part, explained by a higher sensitivity to react to pain with a traumatic stress reaction, this would suggest that psychological interventions should be considered independently or alongside biological pain treatments. It has been highlighted that post-operative psychological interventions are lacking (Katz

et al., 2009), although considerable evidence links increased protective psychological factors such to better pain outcomes (e.g. self-efficacy; Turk, 2002).

*4. Prevalence of PTSD and Relationship to SPTS*

Within the present sample thirty five percent (35.1 %) scored above the low-end of the clinical cutoff for symptom severity of PTSD symptoms in a civilian setting (i.e. 30; Weathers et al., 2013) and thirteen percent (13.8%) at or above the DSM-IV cutoff score on the PCL-C for a diagnosis of PTSD (i.e.. 44; Weathers, et al., 1993; Blanchard et al., 1996). Additionally, mean scores on the PCL-C (M = 29.40, SD = 11.54) were at the low-end of the clinical cutoff for symptoms severity score. As the presence of PTSD symptoms was ascertained by self-report inventory, these rates should be interpreted cautiously. However, these findings do suggest that the present sample exhibits rates of PTSD/PTSD symptoms well above general population levels.

The prevalence of PTSD and PTSD symptoms are elevated in chronic pain samples compared to the general population (Asmundson et al., 2002). The 12-month prevalence rate of PTSD in the general population is 3.5 % as estimated by the US National Comorbidity Survey Replication (Kessler, Chiu, Demler &Walters, 2005). Research suggests that the 12-month prevalence rate of PTSD among chronic pain samples is 2.6 times higher than the general population of the US (Von Korff et al., 2005) and internationally (Demyttenaere et al., 2007). The comorbidity rate of PTSD symptoms in civilian (non-veteran) chronic pain samples ranges from 10-30% (Asmundson et al., 2002; McWilliams et al., 2003). The prevalence rates of PTSD symptoms and PTSD in

the present chronic pain sample are slightly above those in chronic pain populations (e.g. 35.1% compared with 10-30%; 13.8% compared with 9.1%, respectively).

In the present study, total scores on the 12-item SPTS showed a strong positive correlation with PCL-C scores ($r = .595$, $p < .001$), substantiating the hypothesis of adequate convergent validity. This aligns with the earlier finding of Kleiman et al. (2011) that the higher-order SPT factor has a moderate positive correlation with the PCL-C in both the chronic pain ($r = .488$, $p < .001$) and non-chronic pain ($r = .484$, $p < .001$) groups. This finding of Kleiman and colleagues complemented their observation that the 20 pain-related anxiety items of the SPT fit into the DSM-IV PTSD symptom categories: re-experiencing, avoidance, and hyperarousal. The similarity in symptom organization between SPT and PTSD suggests the comorbidity between these constructs may be related through a predisposition toward experiencing traumatic stress reactions. Whereas PTSD symptoms are a clinically significant and enduring response to traumatic stressors of varied natures, the SPTS contains symptoms that describe a traumatic stress reaction specifically to pain (Kleiman et al., 2011).

The multi-cluster symptom representation of the SPT construct allows for the possibility that individual symptoms expression of the SPT vulnerability may be diverse (as represented by different combinations of cognitive, behavioral, and affective symptoms) but that each presentation refers back to a core vulnerability to experience a traumatic response to pain. This aligns with the suggestion that the various pain-related anxiety constructs are related through an overarching organizational factor (e.g. Keogh et al., 2004). Similarly, while PTSD symptom expression varies widely (Breslau, Chase, & Anthony, 2002) the various presentations refer back to the overarching disorder. The

robustness of the finding that the SPTS has a one-factor structure in combination with the high correlation between the SPTS and PCL-C, is suggestive of a shared vulnerability. A lesser correlation between the 12-item SPTS/HADS-D compared to the magnitude of the SPTS/PCL-C correlation ($z = 2.32$, $p = .02$) suggests that the overlap between the SPTS and PTSD is not simply a result of both describing general psychological distress, thus supporting the discriminant validity of the SPTS.

However, the correlation between SPTS and the PCL-C ($r = .595$, $p < .001$) can also be seen to support the uniqueness of these constructs. Despite the overlap between the SPT construct with PTSD there is variability that is not shared between their respective measures. Relatedly, it was found the original SPT study that the SPT factor not the PCL-C was correlated with pain versus no pain groups pre-surgery and at a 1-year follow-up (Kleiman et al., 2011). This underscores the pain-specific traumatization that the SPTS was designed to describe.

Although the present study did not evaluate SPTS scores in a non-pain sample, the results of Kleiman et al. (2011) suggest a relationship between the SPT construct and PCL-C that may not depend on pain history. This is consistent with recent research that synthesizes previous shared vulnerability and mutual maintenance models and suggests the independent contribution of the pain-related anxiety constructs and pain history toward a cumulative influence on PTSD (Rosenbloom, 2014). In this light, the predisposition linking SPTS and PTSD that the present study suggests may be separate from pain history. This possibility is compatible with the findings discussed in Section 3 that suggest the SPT construct may be linked to a heightened sensitivity to pain rather than pain history (e.g. pain history is not requisite to produce high scores on the SPTS).

*5. SPTS and Sex*

In the present analysis, 12-item SPTS total scores demonstrated a low significant positive correlation with sex ($r$ =.283, p=.004). Mean SPTS scores were significantly higher in women (M = 13.07, SD = 9.14) than in men (M = 8.42, SD = 8.90) [t (102) = -2.394, p = .018]. This aligned with the findings from the initial SPTS study, in which mean SPTS total scores were higher in women ($M$ = 2.39, $SD$ = 0.95) compared with men ($M$ = 1.65, $SD$ = 0.46) [$F_w$ (1, 64.09) = 11.37, $p < 0.001$] with an observed power of 0.92. In addition, women tended to score higher on most of the 12-items of the SPTS and were more likely to rate every day pain events as more painful compared with men (men [M=3.76, SD=1.90] and women [M=5.07, SD=2.00], t(79)=-2.321, p = .023] (Roosen, 2009).

These findings reflect a substantial body of research suggesting that women and men differ in their response to experimental pain, and demonstrate that women show greater sensitivity to multiple pain modalities (Bartley et al., 2013). For example, in a recent study of almost 300 hundred participants (n = 291) undergoing a variety of experimental pain manipulations (heat, cold, pressure, and ischemic pain assessments), the group with the highest sensitivity to all of the stimuli was composed of 83% women (Cruz-Almeida et al., 2013; Sullivan, Tripp, & Santor, 2000). Other studies have found that sex is the only variable consistently affecting measures of heat, pressure, and cold pain (Hastie, Riley, Robinson, Glover, Campbell, Staud, Fillingim, 2005; Neziri, Scaramozzino, Andersen, Dickenson, Arendt-Nielsen & Curatolo, 2011).

In addition to exhibiting a heightened sensitivity to experimental pain manipulations, women also tend to score higher on measures of pain-related anxiety

including the ASI (Stewart, Taylor, & Baker, 1997), PASS-20 (Osman, Barrios, Osman, Schneekloth & Troutman, 1994) and PCS (D'Eon, Harris & Ellis, 2004). In a review on the fear of pain construct, Keogh et al. (2004) suggest that there are "important difference in the emotion-pain relationship" between men and women (pg. 107). While the precise etiology of sex differences in pain sensitivity and response are not yet clear, several psychosocial (e.g. pain history, childhood abuse) and biological variables (e.g. sex hormones, genetics) are being investigated (Bartley et al., 2013; Racine, Tousignant-Laflamme, Kloda, Dion, Dupuis, Choinière, 2012).

The findings of the present study add to this body of literature by supporting the distinction between men and women in sensitivity to pain as well as linking this sensitivity to a broader disposition to traumatic reactions to pain as measured by the 12-item SPTS. In light of the established sex differences in pain sensitivity, it is interesting that the SPTS demonstrated excellent internal consistency for both men (men: $\alpha$ = .922) and women ($\alpha$ = .860). However, due to the small number of women in the study this result should be interpreted tentatively and reliability within subgroups such as sex should be examined in future studies with larger samples from various settings.

In the present sample, neither current pain nor average post-surgical chronic chest pain was correlated with sex. This is surprising, given the well-established finding that men and women differ in terms of clinical and chronic pain prevalence (Fillingim, 2000). In a recent retrospective cohort study of over 300,000 patients, women reported greater mean NRS pain scores and higher proportion of severe pain events following surgery compared with men (Tighe et al., 2014). An international survey study of chronic pain

prevalence of over 40,000 participants found a higher prevalence of chronic pain conditions in women (Tsang et al., 2008).

The proportion of women who reported currently using pain medication (60%) was significantly higher than the proportion of men (27%; $z=-3.16$, $p<.002$). It is possible that women's true pain scores are tempered by the effective use of medication. This would explain why while overwhelming empirical evidence suggests that women have greater pain sensitivity than men, this study did not demonstrate any significant difference between men and women's pain ratings. In addition, the proportion of women who reported other ongoing pain problems unrelated to their surgery (70%) was significantly higher than the proportion of men (40.5%; $z=-2.72$, $p<.007$). Taken together, the interpretation of these findings is not straightforward. Given that women have higher mean SPTS scores than men, it is possible that the differences in current pain medication use and additional pain problems are indicative of greater pain sensitivity. It is also possible that the current use of pain medication is directed at the additional pain problems, rather than post-operative pain. The relationship between sex, SPTS, pain, medication use, and additional pain problems is complex but should be investigated in future studies.

*6. Limitations*

The limitations associated with the present study inform directions for future research with the SPTS. The sample size, aspects of the study design, and issues surrounding the psychometric assessment of pain will be discussed. The relatively small sample size restricts the generalizability of the results of the present study. To begin, all of the psychological measures of interest were described by non-normal distributions. It

is possible that the results of this study are not relevant to the larger population of post-surgical patients but instead reflect the particularities of the present sample. While deviations from normality are common in research, the violation of normality also imposed limits to the statistical analyses of the present study.

EFA is an exploratory procedure that assists in building a foundation to direct future research, which was the premise for choosing it for the present study. However, it is also a multi-step procedure that includes several subjective choice points for the researcher (Fabrigar et al., 1999). While the present sample was large enough to conduct the EFA, it was not sufficient for a complementary CFA that is also recommended for scale development (Netemeyer, Bearden, & Sharma, 2003). The advantage of a CFA is it begins with a factor model that is then fit to the data (Schmitt, 2011). This procedure supports hypothesis testing and inference, and should be used after an initial EFA is preformed (Costello et al., 2005). Thus, based on the EFA method, the results of the present study may not be extrapolated to the general population unless the same results are obtained over several samples (Fields, 2009).

The non-normal distributions associated with the present sample also indicate that the results should be interpreted tentatively. Non-parametric correlations were used for the validity assessment, and this restricted the possibility of using partial correlations to control for additional variables. It also reduces confidence in the results of the z-test used to evaluate the difference in magnitude between the convergent and discriminant validity correlation coefficients. Nevertheless, this study yielded promising preliminary results that highlight important areas for future research.

This prefatory analysis suggested significant differences in STPS levels with respect to sex, pain levels, medication use, and additional pain problems. While the sample size was too small to compare between subgroups with confidence, the differences suggested by the present analysis highlight the potential importance of these variables in evaluating the nature of the STPS. The unequal subsamples of men and women participants yielded a somewhat contrary result of significant sex differences in SPTS levels, yet high reliability coefficients for both groups. Surprisingly, sex was not related to pain levels or PTSD in contrast to the literature on these topics (see discussion Section 5). Ideally, future studies would use a sample of at least 200 with more equality between the subsamples of men and women to allow for comparison between these groups. In addition a sample of this size would more closely approximate the normal distribution, as well as allow for complementary EFA and CFAs.

The design of the present study precluded assessing additional forms of validity. No pre-surgery assessment was collected in the sample, thus the pre-existing psychometrics of the sample could not be taken into account. This prevented testing the predictive validity of the SPTS in the present sample (e.g. the extent to which the SPTS measured later chronic pain disability or PTSD levels). The lack of a pre-surgery assessment also prevented the examination of pre-morbid SPT levels prior to a controlled pain event, compared with post-surgery levels of SPTS, pain, and PTSD. In addition, collecting only one post-surgery assessment did not allow for an evaluation of test-retest reliability. While multiple assessments increases participant burden, it can also provide important information about the reliability of a test and the relative state-independence of the SPT construct.

The timing of the assessments introduced a complexity to the study. As the full assessment was divided over 2 time points, measures of chronic pain were not taken at the same time as the SPTS. In addition, the inclusion criteria for the study were such that participants were required to have experienced chronic post-surgical pain after surgery, but not all were presently experiencing pain (e.g. their pain may have resolved). The advantage of this definition of a "chronic pain sample" is that it allows for the evaluation of SPT levels in a group distinguished from patients who completed surgery and followed the expected healing trajectory. However, the disadvantage of this sample and assessment structure is that some participants were reporting on their current state during the pain assessment and others regarding their memory of pain. Furthermore, given the association between the SPTS and pain, it is possible that currently being in pain while completing the scale would alter ones pattern of responding or ability to generalize their typical attitude toward pain.

The assessment of pain is necessarily complex as it aims to record an inherently subjective and diverse phenomenon. The present study relied on self-report rather than medical record. While this was necessary to capturing the variation in pain scores and allowed for hypothesis to be made regarding differential sensitivity to pain, its disadvantage lies in the lack of objective reference for pain. For example, from the self-report scale it is not possible to infer if the chronic pain was clinically significant or impacted the participants daily functioning. Moreover, the liberal inclusion criteria facilitated a highly heterogeneous sample with those across the spectrum in pain ratings. While the absence of stricter criteria for the presence of chronic pain may have diluted the sample in some regards, it allowed for an important and unexpected exploration of

STP levels in high and low pain-rating groups which was an important finding from this study.

7. *Future Directions*

The limits of the present study indicate a diverse program of research for the future of the SPTS. The SPTS should be tested in larger samples to facilitate the statistical assumptions of normality, allow for both EFA and CFA procedures, and allow for comparisons between subgroups such as sex. Future studies would benefit from including a pre-surgery, and multiple post-surgery assessments to allow for additional tests of validity and to examine the relationship between pre-morbid SPTS levels and post-surgery outcomes. Ongoing research would benefit from including objective pain measures such as medical record data or self-report questions on pain impact and disability. As several studies have found that differences in the pattern of pain-related anxiety constructs between pain and non-pain populations, the SPTS should be examined in non-pain settings, as well as non-surgical chronic pain groups, and psychiatric clinical populations.

With these general recommendations in mind, there are also several specific tasks to pursue in future studies. The initial SPT study by Kleiman et al. (2011) found that 20-items from the ASI, PCS, and PASS-20 formed a cohesive factor representing sensitivity to pain traumatization. Given the substantiation of the positive preliminary reliability and validity of the 12-item SPTS, this measure should be tested alongside the original pain-anxiety measures. Germane to the present findings is to conduct this investigation in groups that can be split by sex and pain level. Future studies should investigate the

possibility that the etiology and maintenance of chronic pain and PTSD may be different across various subgroups.

It is also critical to assess the reliability and validity of the 12-item SPTS in nonclinical samples. Of particular importance is collecting a normative sample, which represents the range of SPT levels in a community setting. The literature suggests differences between clinical and non-clinical populations on the pain-related anxiety measures (e.g. Olatunji et al., 2009). The differences between the mean SPTS scores of Roosen (2009) and the present study also suggest this. A normative sample would allow for a meaningful comparison with clinical populations to support the development of clinical cut-offs for the SPT trait. Critical to the evaluation of mental disorders according to the DSM-IV (American Psychiatric Association, 1993), is the non-content criterion for diagnosis including an evaluation of distress and impairment resulting from conditions. Evaluating the relationship between SPTS scores and impairment will be an important aspect of refining the clinical applicability of the scale.

The close relationship between the SPTS and PTSD symptoms (as measured by the PCL-C) demonstrated by the present study warrants further investigation. While the presence of PTSD symptoms in the present sample reflected the general prevalence rates of PTSD symptoms in chronic pain samples, it is important to evaluate the SPTS in a clinical psychiatric population composed of individuals who meet all clinical criteria for the disorder. This will assist in furthering clarifying the shared predisposition between chronic pain and PTSD as well as discriminating between trauma responses to pain from general stressors.

It was originally thought that 12-item SPTS could potentially differentiate between individuals with and without a history of pain problems. However, the results of the present study are more compatible with the interpretation that SPTS scores differentiate those with higher verses lower pain sensitivity. One potential area of investigation that could assist in clarifying this is to assess the SPTS with measures of pain intensity as well as psychophysical testing which captures sensitivity to pain. In order to assess whether this hypothesized sensitivity toward greater impact from pain does increase risk for developing chronic pain, longitudinal assessments would be of benefit, to compare pre-surgical SPTS scores to post-surgical pain outcomes.

Until recently, theoretical models describing shared vulnerability, mutual maintenance, and combined influence for comorbid chronic pain and PTSD had not been empirically tested, although the relationship between the pain-related anxiety constructs and chronic pain and PTSD has been independently examined (see Introduction Section 2). However, Martin at al. (2010) used structural equation modeling (SEM) to test Turk's diathesis-stress model of chronic pain as well as a second model that incorporated the potential contribution of PTSD symptoms to this model of chronic pain disability in response to the growing body of research that highlights this connection (e.g. Asmundson et al., 2002; Otis et al., 2003). The first SEM analysis supported a model that included several of relationships proposed by Turk. The second analysis provided preliminary support for role of PTSS in the model, as it accounted for a significant proportion of the variance in disability. The present findings of the SPTS can contribute to future empirical evaluation of models such as this by synthesizing the relationship between the pain-

related anxiety constructs as well as providing an parsimonious way to assess their underlying vulnerability factor.

There is a need to strive towards a nuanced understanding of the factors that contribute to the inception and maintenance of these disorders. For example, in a study assessing factors related to the transition from acute to chronic pain Katz et al., (2009) assessed patients 6 and 12 months post-surgery. It was found that the factors affecting the inception of chronic pain and the maintenance of chronic pain differed at these time-points. Comparing predictors of pain disability at 6 and 12-month follow-ups, pain disability became less related to pain intensity over time, whereas emotional numbing become more related to pain disability. The authors propose that pain itself may be a traumatic stressor as it reminds individuals about their trauma. The results of the present study highlight the possibility that it may be the reaction to pain that is the traumatic stressor. Including the SPTS in future studies can assist in clarifying this question.

8. *Study Implications*

The importance of the pain-related anxiety constructs in describing the co-occurrence, shared etiology, and reinforcing comorbidity of chronic pain and PTSD cannot be understated. This present study represents an effort to clarify the nature of this shared vulnerability both to elaborate the origin of these related psychological constructs and to contribute to informed care for individuals who present with both disorders. The results of the present study suggest the SPTS scale is a promising instrument which both captures a sensitivity to react to the experience of pain traumatically and the association between chronic pain conditions and PTSD symptoms. As such, it warrants future

research and already in its present form captures information that is critical for directing informed clinical care.

The preliminary results of the present study represent a culmination of the findings of Kleiman et al. (2011) that a general sensitivity to experience pain as a trauma underlies the major pain-related anxiety measures and of Roosen (2009), that the SPTS scale may measure this predisposition. The apparent cohesiveness of the SPT factor is suggested by its one-factor structure, good preliminary reliability and validity; and shared variation with a measure of PTSD (e.g. the PCL-C) and pain rating levels. This finding is compatible with shared vulnerability (Asmundson et al., 2002), and combined models (Rosenbloom, 2014) that suggest a shared predisposition toward chronic pain and PTSD. It has been demonstrated that levels of pain-related anxiety constructs are higher in anxiety disorder patients (e.g. Carleton et al., 2009b) and chronic pain patients (e.g. Asmundson et al., 2009) compared with community samples. The results of the present study suggest further investigation to clarify if a higher-order factor can parsimoniously describe this trend or if the SPT factor should be measured alongside these constructs.

While the theoretical modeling of this comorbidity and its paired empirical validation are important ongoing pursuits, the clinical management of affected individuals is an urgent current need. A staggering body of evidence suggests those with comorbid PTSD and chronic pain fair more poorly on a variety of health and recovery indicators. This comorbidity is associated with more intense pain, greater physical health challenges (Asmundson et al., 2009), functional limitation (Duckworth et al., 2005), occupational impact (Bosco, Gallinati & Clark, 2013; Kessler, 2000), and emotional distress (Geisser et al., 1996; Asmundson et al., 2002 for review). Researchers within this

field advocate for the assessment of PTSD in chronic pain populations and vice versa (e.g. Pagé, Kleiman, Asmundson & Katz, 2009), and the targeting of psychological interventions toward mechanisms thought to be responsibility for the shared vulnerability (e.g. avoidance and fear behaviors; Bosco et al, 2013).

It is recommended that appropriate assessment for individuals with comorbid chronic pain and PTSD include interview and self-report measures, a comprehensive inventory of symptoms (e.g. pain intensity, location, etc.), as well as questions on emotional coping behaviors and level of functional impairment (Asmundson et al., 2002). Indices of pain-related anxiety such as the PCS and PASS are recommended (e.g. Asmunsdon et al., 2002). In terms of treatment, Cognitive Behavioral Therapy (CBT) has been shown to be effective for PTSD (e.g. Taylor, 2004) and chronic pain (e.g. Edhe, Dillworth & Turner, 2004) independently. When treated together, a modified approach is recommended, for example CBT adapted to include pain management strategies (Asmunsdon et al. 2002). Anxiety sensitivity has been highlighted an important vulnerability factor for explaining this comorbidity and the extreme emotional reactions to trauma and pain associated with injury (Asmundson et al., 2009; Turk, 2002). Evidence suggests the treatment of AS may improve both PTSD (Taylor et al., 2004) and chronic pain (Ocañez et al., 2010).

This literature and the suggestion of a higher-order factor underlying the pain-related anxiety constructs, indicates a role for the 12-item SPTS in future research. The three studies representing the SPT construct (e.g. Kleiman et al., 2011; Roosen, 2009; and the present study) suggest that AS may be subsumed under the SPT factor and that this factor represents sensitivity to pain, the disposition to be greatly impacted by pain, and to

respond traumatically to pain. This highlights the potential of the SPT construct to contribute to our understanding of the shared etiology and maintenance of chronic pain and PTSD; both through representing the disposition toward traumatic responses to pain and by creating a parsimonious representation of the primary pain-related anxiety constructs critical to this comorbidity. Further, the brevity and preliminary reliability and validity of the SPTS suggest it will be an asset in assessment to identify individuals at higher risk, and to direct treatment planning or incorporate into treatment protocols.

### *9. Conclusion*

The present study was the first to examine the factor structure, reliability, and validity of the 12-item SPTS in a clinical sample of patients who experienced chronic pain after undergoing surgery. Preliminary results suggest that the SPTS is a reliable and valid tool for an adult post-surgical population that may assist in identifying those at higher risk for developing greater trauma responses to pain that support the development of chronic pain and disability. In response to the suggestion in the literature that a higher-order factor underlies the various pain-related anxiety constructs, this study represents an effort to clarify the nature of that factor which is thought to describe a propensity toward a trauma response to pain (Kleiman et al., 2011). Additionally, results suggest that the SPT has a single-factor structure that is associated with a higher sensitivity to pain, PTSD symptoms, and presents at higher rates in women.

The present study highlights several future directions for the SPTS including testing the scale in larger samples; clarifying the presentation of the SPT trait in different

clinical and non-clinical populations and subgroups (e.g. sex); testing it alongside the pain-related anxiety constructs; and including it in empirical investigations of models of chronic pain and PTSD and studies on the inception and maintenance of chronic pain. In the long term, the continued development of the SPTS will contribute to clinically relevant assessment and treatment planning. In the short-term, the present study suggests that the SPTS is already able to identify those who are more psychologically impacted by pain, which is information germane to the assessment and recovery planning for post-surgical patients.

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Appendix A:

**12-item SPTS**

INSTRUCTIONS: The statements listed below describe beliefs, thoughts, feelings and actions that people have or do when they are in physical pain (i.e., when a part of their body hurts). Read each statement carefully and place a check mark (✓) in the box that best reflects how true that statement is for you.[2]

| | Not at all true | Slightly true | Somewhat true | Very true | Entirely true |
|---|---|---|---|---|---|
| 1. Pain keeps me awake at night. | | | | | |
| 2. When I am in pain, everything I see or do reminds me of the pain. | | | | | |
| 3. I try to avoid activities that cause pain. | | | | | |
| 4. When I feel pain, I'm scared that it's the beginning of a terrible problem. | | | | | |
| 5. Pain seems to bother me more than it does other people. | | | | | |
| 6. When I feel pain, I think about it even when I don't mean to. | | | | | |
| 7. I can't stand pain. | | | | | |
| 8. When I'm in pain, I feel distant from people even when I'm talking to them. | | | | | |
| 9. As soon as the pain comes on, I take medications to reduce it. | | | | | |
| 10. Pain sensations terrify me. | | | | | |
| 11. When I'm in pain, things don't feel real. | | | | | |
| 12. I feel sick to my stomach when I am in pain. | | | | | |

[2] SPTS (Roosen, 2009)

Appendix B:

**Chronic Post-Cardiac Surgery Pain Questionnaire: Short Survey**

STUDY ID NUMBER AND INITIALS (F/L): _____

Date: _____/_____/_____ DD / MM / YYYY

Telephone: _____

1) Sex: M ☐ F ☐

2) Age: _____

3) Mother's country of origin: _____

4) Father's country of origin: _____

**C. PAIN AFTER THE CARDIAC SURGERY**

7) Now we are interested in finding out if you currently have, or previously had, chronic pain as a result of your cardiac surgery. We are interested in cardiac-related chronic pain even if it has now gone away.
Listed below you will see a list of BODY PARTS that may have been ***chronically painful*** AFTER your cardiac surgery (pain for 3 months or longer). Please complete the PAIN INTENSITY with a number from 0-10. Where 0 is 'no pain' and 10 is the 'most intense pain imaginable.' This number represents the **average pain** you experienced from 3 months **after the cardiac operation,** and then put that number next to the appropriate BODY PART. Next, for the same BODY PART also select a number from 0 – 10 that relates to PAIN UNPLEASANTNESS, where 0 is 'Not unpleasant' and 10 – 'Most unpleasant pain imaginable'. The unpleasantness of the pain describes how distressing this pain was/is for you. The distinction between pain intensity and pain unpleasantness might be made clearer if you think of listening to music. The intensity of the pain is like the loudness of the music, whereas the unpleasantness of the pain is how much you like or dislike the music.
Next, please recall how long each episode of pain lasted, using the choices: 'sec/min', 'hours', 'days', 'weeks/months', or 'constant'.

| | PAIN INTENSITY | PAIN UNPLEASANTNESS | HOW LONG DID EACH PAIN EPISODE LAST? | | | | |
|---|---|---|---|---|---|---|---|
| Limbs / Shoulder/ Hip | 0 1 2 3 4 5 6 7 8 9 10 | 0 1 2 3 4 5 6 7 8 9 10 | Sec/Min | Hours | Days | Weeks/ Months | Constant |
| Head/ Neck/ Face/ Mouth | 0 1 2 3 4 5 6 7 8 9 10 | 0 1 2 3 4 5 6 7 8 9 10 | Sec/Min | Hours | Days | Weeks/ Months | Constant |
| Chest pain from your cardiac surgery | 0 1 2 3 4 5 6 7 8 9 10 | 0 1 2 3 4 5 6 7 8 9 10 | Sec/Min | Hours | Days | Weeks/ Months | Constant |
| Lower Trunk (Abdomen/ Genitalia/ Lower Back) | 0 1 2 3 4 5 6 7 8 9 10 | 0 1 2 3 4 5 6 7 8 9 10 | Sec/Min | Hours | Days | Weeks/ Months | Constant |

8) Now, for the same BODY PART(S) please fill in **how often you had/have these pain episodes** using the choices: several times 'in an hour' / 'a week' / 'a month' / '3-6 months' / '6-12 months' / '1-2 years' / 'in 2 yrs or more'.
Next, please respond to the question FOR HOW LONG HAVE YOU EXPERIENCED THESE PAIN EPISODES, using the choices: 'Half a year (within 6 months after the operation)', 'A year (within 12 months)', or 'Still have it (the pain has not gone away completely)'.
Lastly, please tell us WHERE IS THE PAIN LOCATED, using the choices: 'skin', 'scar', 'deep tissue (such as muscle/joint)'.

| **BODY PART** | **FREQUENCY OF THE PAIN**<br><br>**How often did you have such pain episodes?**<br><br>Please circle one below | | | | | | | **For how long have you experienced these pain episodes?**<br><br>Please circle one below | | | **Where is the pain located?**<br><br>Please circle all that apply | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Limb/ Shoulder/ Hip | Hourly | Weekly | Monthly | 3-6 months | 6-12 months | 1-2 years | 2 years or more | Half a year | A year | Still have it | Skin | Scar | Deep inside (muscle/ joint) |
| Head/ Neck/ Face/ Mouth | Hourly | Weekly | Monthly | 3-6 months | 6-12 months | 1-2 years | 2 years or more | Half a year | A year | Still have it | Skin | Scar | Deep inside (muscle/ joint) |
| Chest pain from your cardiac surgery | Hourly | Weekly | Monthly | 3-6 months | 6-12 months | 1-2 years | 2 years or more | Half a year | A year | Still have it | Skin | Scar | Deep inside (muscle/ joint) |
| Lower Trunk (Abdomen/ Genitalia/Lo wer back) | Hourly | Weekly | Monthly | 3-6 months | 6-12 months | 1-2 years | 2 years or more | Half a year | A year | Still have it | Skin | Scar | Deep inside (muscle/ joint) |

9) Do you have numbness around your chest scar? Y ☐ N ☐

10) Did the surgeon use a vein from your leg as part of your CABG operation? Y ☐ N ☐ Don't know ☐

If yes, have you experienced chronic pain (that lasted more than 3 months) at or around the site of this leg operation? Y ☐ N ☐

11) Where exactly was/is that pain felt?

☐ Scar ☐ Skin ☐ Deep Tissue (Tendons/Muscles/Joint)

12) Do you have numbness around your leg scar? Y ☐ N ☐

Appendix C:

**Chronic Post-Cardiac Surgery Pain Questionnaire: Long Survey**

We would like you to do three things and indicate whether they cause you any pain or discomfort. Please rate your pain using a scale where 0 means "no pain" and 10 means "most intense pain imaginable":

1) Please take a deep breath, expanding your chest. Does that hurt? Please rate the pain:

0 1 2 3 4 5 6 7 8 9 10

No Pain Most intense pain imaginable

2) Please run your finger lightly along your chest scar. Does that hurt? Please rate the pain:

0 1 2 3 4 5 6 7 8 9 10

No Pain Most intense pain imaginable

3) Please run your finger lightly along your leg scar. Does that hurt? Please rate the pain:

0 1 2 3 4 5 6 7 8 9 10

No Pain Most intense pain imaginable

Not Applicable ☐

4) We would like to know how intense the following everyday pains are for you. Rate the intensity of the following pains from 1 (not at all intense) to 5 (as intense as can be).

Toothache _____
Paper cut _____
Stubbed toe _____
Biting your tongue _____
Earache _____

5) If applicable, when did the <u>chronic</u> **chest** pain from cardiac surgery, which you currently feel or have previously felt begin?

_____

If the chronic chest pain from cardiac surgery has since gone away, when did it end?

_____

6) If applicable, when did the <u>chronic</u> **leg** pain from cardiac surgery, which you currently feel or have previously felt begin?

_____

If the chronic leg pain from cardiac surgery has since gone away, when did it end?

_____

7) Are you currently experiencing any other on going pain problems? YES NO

Location of other pain? _____

When did this other pain begin? _____
What is the intensity of this other pain? Please use a scale of 0 (no pain) to 10 (worst pain imaginable): _____

In the next set of questions we want to learn if your pain is severe enough to necessitate treatment.

8) Do you take any pain medication currently? Y ☐ N ☐

Please fill in the chart below by specifying the name of medications and other treatments you are or have taken for chronic pain and their effect on your chronic pain by selecting from the following 4 levels:

| **Name of the treatment** | | **Pain Relieving Effect on the Chronic Pain** | | | | **Did you stop because of side effects?** | |
|---|---|---|---|---|---|---|---|
| Type | Enter names below | No relief | Slight relief | Some relief | Complete relief | Yes | No |
| **A. Pharmacological medications** (if you take but don't know the name - write "don't know name" and describe effects ) | 1 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 2 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 3 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 4 | 1 | 2 | 3 | 4 | 1 | 2 |
| **B. Natural health products** e.g., vitamins, mineral supplements, dietary supplements, herbs, alcohol, marihuana | 1 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 2 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 3 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 4 | 1 | 2 | 3 | 4 | 1 | 2 |
| **C. Physical approaches** e.g., acupuncture, physiotherapy, massage, exercise, pressure, | 1 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 2 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 3 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 4 | 1 | 2 | 3 | 4 | 1 | 2 |
| **D. Psychological approaches** e.g., hypnosis, relaxation, distraction, talking to a religious/spiritual authority, prayer, meditation, breathing techniques | 1 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 2 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 3 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 4 | 1 | 2 | 3 | 4 | 1 | 2 |
| **E. Medical-invasive / Surgical treatments** e.g., injections to the painful site; resection of the nerves | 1 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 2 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 3 | 1 | 2 | 3 | 4 | 1 | 2 |
| | 4 | 1 | 2 | 3 | 4 | 1 | 2 |