

PCS

The Pain Catastrophizing Scale

User Manual

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Pain Catastrophizing

The Construct

In 'Sur L'eau, novelist Maupassant (1875) writes,

“Migraine is atrocious torment, one of the worst in the world, weakening the nerves, driving one mad, scattering one’s thoughts to the winds and impairing the memory. So terrible are these headaches that I can do nothing but lie on the couch and try to dull the pain by sniffing ether.”

Maupassant’s words describe the torment of his pain, his emotional distress, and the disability that pain brings to his life. He feels overwhelmed by his pain, and helpless to deal with it. He surrenders to the pain, and seeks chemical means of dulling it. Today’s specialists on the psychology of pain would argue that Maupassant’s ‘catastrophic thinking’ about his pain likely played a role in heightening the intensity of the pain he experienced.

In order to grasp the essence of current conceptualizations of catastrophizing, it is useful to consider four papers that have provided a foundation for the literature on catastrophizing (Chaves and Brown, 1978; Spanos et al, 1979; Rosenstiel and Keefe, 1983; Sullivan et al., 1995). In an early study, John Chaves and Judith Brown (1978) asked dental patients to report thoughts and images they experienced, or the strategies they engaged in, during a stressful dental procedure. The content of the interview records was then examined. Chaves and Brown (1978) noted that individuals differed markedly in the thoughts they experienced during the dental procedure. They found that individuals who engaged catastrophic thoughts were particularly likely to experience high levels of distress during the dental procedure. Catastrophizers were described as individuals who had a tendency to magnify or exaggerate the threat value or seriousness of the pain sensations (i.e., “I’m afraid that my pain might get worse”).

Nicholas Spanos and his colleagues from Carleton University were also interested in the psychological factors that influenced the experience of pain (Spanos et al., 1979). In their research, they asked university

students to immerse one arm in a container of very cold water and to report the degree of pain they experienced. Cold water immersion is often used as a means of inducing pain in the laboratory. The procedure is referred to as a 'cold pressor procedure' and can induce significant pain without causing any tissue damage. Spanos et al (1979) interviewed their subjects about their pain experience following participation in the cold pressor procedure. Individuals who reported thought content reflecting worry, fear, and the inability to divert attention away from pain were classified as catastrophizers (i.e., "I kept thinking I can't stand this much longer, I want to get out"). Similar to the findings of Chaves and Brown (1978) individuals who engaged in catastrophic thinking reported the highest levels of pain.

Frank Keefe and his colleagues from Duke University (Rosenstiel and Keefe, 1983; Keefe et al., 1989) were interested in the pain-related thoughts experienced by individuals with chronic pain. They developed the Coping Strategies Questionnaire (CSQ), which consists of 7 coping subscales, including a catastrophizing subscale. The items on the catastrophizing subscale reflect elements of helplessness and pessimism in relation to one's ability to deal with the pain experience (i.e., "Its terrible and its never going to get any better"). Numerous investigations conducted by Keefe and his colleagues have shown that individuals who obtained high scores on the catastrophizing scale of the CSQ experienced higher levels of physical and emotional distress associated with their pain condition.

In 1995, my colleagues and I (Sullivan et al., 1995) developed the Pain Catastrophizing Scale (PCS) in an effort to develop a comprehensive evaluation instrument that would encompass the different perspectives on catastrophizing that had been discussed by previous investigators. The PCS is currently one of the most widely used measures of catastrophic thinking related to pain. It has been translated into several languages and has been incorporated in the assessment protocol of pain clinics and rehabilitation centres throughout North America and Europe. The PCS taps three dimensions of catastrophizing: rumination ("I can't stop thinking about how much it hurts"), magnification ("I worry that something serious may happen"), and helplessness ("It's awful and I feel that it overwhelms me").

Catastrophizing is currently defined as:

"an exaggerated negative mental set brought to bear during actual or anticipated painful experience" (Sullivan et al., 2001).

The Development of the PCS

The PCS was developed in 1995 at the University Centre for Research on Pain and Disability in order to facilitate research on the mechanisms by which catastrophizing impacts on pain experience. The items on the PCS were drawn from previous experimental and clinical research on catastrophic thinking in relation to pain experience (Chaves and Brown, 1987; Rosenstiel and Keefe, 1983; Spanos et al., 1979).

Factor analyses of the PCS have shown that catastrophizing can be viewed as a multidimensional construct comprising elements of rumination (“I can’t stop thinking about how much it hurts”), magnification (“I worry that something serious may happen”), and helplessness (“There is nothing I can do to reduce the intensity of the pain”). The factor structure of the PCS has been replicated in several investigations (Osman et al., 1997, 2000; Sullivan et al., 1995, 2000; Van Damme et al., 2002).

A copy of the PCS is appended to this document. Electronic copies can be obtained through the web site provided below. The PCS is presently a public forum instrument and as such, no costs are associated with its use or with duplication.

<http://sullivan-painresearch.mcgill.ca/>

Assessment of Catastrophizing Using the PCS

The PCS can be completed and scored in less than 5 minutes, and thus is easily amenable to inclusion within standard clinical practice. Prior knowledge of a patient’s level of catastrophic thinking, in addition to other pain-related variables, enables treatment plans to be more individually tailored.

The PCS is a 13-item instrument derived from definitions of catastrophizing described in the literature (Chaves & Brown, 1987; Spanos et al., 1979) as well as items from the catastrophizing subscale of the CSQ (Rosenstiel & Keefe, 1983). The PCS requires a reading level of approximately Grade 6.

The PCS instructions ask participants to reflect on past painful experiences, and to indicate the degree to which they experienced each of 13 thoughts or feelings when experiencing pain, on 5-point scales with the end points (0) not at all and (4) all the time. The PCS yields a total score and three subscale scores assessing rumination, magnification and helplessness. The PCS has been shown to have adequate to excellent

internal consistency (coefficient alphas: total PCS = .87, rumination = .87, magnification = .66, and helplessness = .78; Sullivan et al., 1995).

The PCS total score is computed by summing responses to all 13 items. PCS total scores range from 0 – 52. The PCS subscales are computed by summing the responses to the following items:

- Rumination: Sum of items 8, 9, 10, 11
- Magnification: Sum of items 6, 7, 13
- Helplessness: Sum of items 1, 2, 3, 4, 5, 12

Interpretation of PCS Scores: Clinical Samples

Appendix A provides a table of PCS raw scores and associated percentile scores. The percentile scores are derived from a sample of injured workers who had initiated a time loss claim with the Nova Scotia Workers Compensation Board. General characteristics of the sample are as follows:

- Sample size: 851
- Sex distribution: 438 men, 413 women
- Age: 42.2 years (range 17 to 63 years).
- Mean duration work absence: 6.9 months
- Injury Type: 75% soft tissue back injury

Indices of central tendency and distribution are as follows:

	PCS
Mean	20.90
Median	20.00
Std. Dev.	12.50
Skewness	0.26
Kurtosis	-0.87
Minimum	0
Maximum	50.00

The percentile equivalents listed in Appendix A should be interpreted with caution when applied to asymptomatic samples, or individuals experiencing acute pain.

Although PCS scores are normally distributed, suggesting that individuals vary in degree in their level of catastrophizing, it has been useful to consider 'cut-off scores' for clinically relevant levels of catastrophizing. Research at the University Centre for Research on Pain and Disability indicates that a total PCS score of 30 represents clinically relevant level of catastrophizing. A total PCS score of 30 corresponds to the 75th percentile of the distribution of PCS scores in clinic samples of chronic pain patients. Appendix A provides the 75th percentile cut-off scores for the three PCS subscales.

For the normative database described above, in the subsample of patients who scored above 30 (75th percentile) on the PCS;

70% remained unemployed one year post injury

70% described themselves as totally disabled for occupationally-related activities

66% scored above 16 (moderate depression) on the BDI-II

Implications for Screening

It is becoming increasingly clear that catastrophic thinking in relation to pain might be a risk factor for chronicity. In other words, catastrophizing not only contributes to heightened levels of pain and emotional distress, but also increases the probability that the pain condition will persist over an extended period of time. Janet Pavlin and her colleagues from the University of Washington (Pavlin et al, 2004) found that high scores on a measure of catastrophizing predicted the degree of pain that individuals experienced following surgery, and contributed to a higher level of disability in the weeks that immediately followed surgery. Michael Forsythe and his colleagues at Dalhousie University (Forsythe et al., 2008) followed a group of arthritis patients for two years following knee replacement surgery. Their study showed that individuals who obtained high scores on a measure of catastrophizing had a higher probability of experiencing persistent knee pain and disability two years following their surgery. A number of recent investigations have shown that catastrophizing, assessed shortly following occupational injury, predicted the development of chronic pain and disability (Picavet et al., 2002; Waddell et al., 2003). Findings such as these suggest that if catastrophic

thinking can be minimized, then the probability of the persistence of pain and disability might be reduced.

In recent work on the prevention of chronicity, the PCS has been used as a screening measure for risk of prolonged pain and disability. Individuals who score between the 50th and 75th percentiles on the PCS are considered at moderate risk for the development of chronicity. Individuals who score above the 75th percentile would be considered at high risk for the development of chronicity. Individuals who obtain high scores on the PCS would be considered suitable candidates for a risk-factor targeted intervention program (described in more detail below).

A Determinant of Pain-Related Outcomes

Research has supported a multidimensional conceptualization of catastrophizing comprising elements of rumination, magnification, and helplessness (Sullivan et al., 1995; Osman et al., 2000; Van Damme et al., 2002). Individuals who score high on measures of pain catastrophizing report more intense pain (Sullivan et al., 1995, 2006), more severe depression and anxiety (Keefe et al., 1989; Martin et al., 1996), show higher levels of pain behaviour and disability (Sullivan et al., 1998, 2000, 2006; Keefe et al., 2000; Sullivan and Stanish, 2003), consume more analgesic medication (Bedard et al., 1997; Jacobsen and Butler, 1996) and have more prolonged stays when hospitalized (Gil et al., 1992).

To date, nearly 600 studies have been published documenting a relation between catastrophizing and pain. A significant relation between catastrophizing and pain-related outcomes has been observed in numerous pain samples. These have included patients with rheumatoid arthritis (Keefe et al., 1989), osteoarthritis (Keefe et al., 1997), fibromyalgia (Martin et al., 1996), sickle cell disease (Gil et al., 1992), soft tissue injuries (Sullivan et al., 1998; Sullivan and Stanish, 2003), neuropathic pain (Sullivan et al., 2005), dental patients (Sullivan & Neish, 1999), and patients recovering from surgery (Jacobsen & Butler, 1996). A relation between catastrophizing and pain-related outcomes has been observed in children as young as 7 years (Crombez et al., 2002; Gil et al., 1993).

The relation between catastrophizing and pain appears to emerge early in life, has been observed across a wide range of clinical and experimental pain-eliciting situations, and shows a remarkable consistency. Implicit in this work is the view that catastrophizing is causally related to pain, and the pattern of findings appears to support the causal or, at least, antecedent status of catastrophizing. For example, catastrophizing, assessed while individuals are in a pain-free state, prospectively predicts

pain ratings made in response to aversive stimulation. Catastrophizing scores obtained one week (Sullivan and Neish, 1999) or 10 weeks (Sullivan et al., 1995) prior to a painful procedure predicted pain ratings. Catastrophizing prospectively predicted pain ratings in arthritis patients 6-months later, even when controlling for initial pain ratings (Keefe et al., 1989). Reductions in catastrophizing have been shown to prospectively predict reductions in pain and disability (Sullivan et al., 2006; Adams et al., 2007).

Theory and Mechanisms

It is not clear when the term catastrophizing was first used in the psychological literature, but there are references to catastrophizing in psychological writings dating back to the beginning of this century. In the 1960s, the term catastrophizing was used to describe the excessively negative thinking of individuals with depression. For example, Beck (1967) described catastrophizing as a 'cognitive distortion' that could contribute to the development or exacerbation of symptoms of depression. The term catastrophizing has also been used to describe the mental set of individuals suffering from various anxiety disorders (Beck and Emery, 1985). It is possible that the essential features of catastrophizing in depression and anxiety may be similar to those of catastrophizing as discussed in the pain literature (Turner and Aaron, 2001). But there has been some reluctance to consider them equivalent. Catastrophizing in the literature on depression and anxiety has been characterized in rather 'pathological' terms. This is not a view that is necessarily held in the pain literature (Sullivan et al., 2004). There are indications that catastrophizing may serve very useful coping functions in the day to day life of certain individuals. It may be only in situations where individuals develop chronic pain conditions that catastrophizing truly becomes problematic.

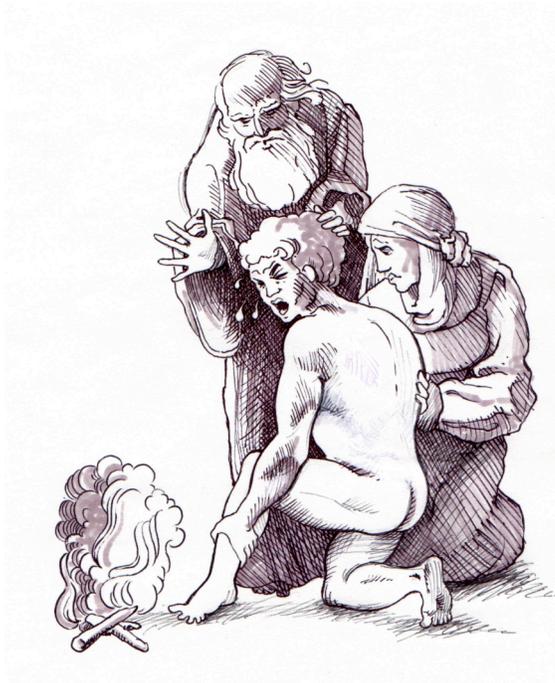
A number of theoretical models of catastrophizing have been put forward in order to increase our understanding of the manner in which catastrophic thinking might influence pain (Sullivan et al., 2001). These theories include a Beckian model of cognitive errors in which catastrophizing is compared to the dysfunctional thinking present in depressives, an appraisal model characterized by exaggerated perception of the threat value of pain sensations, and a coping model in which catastrophizing is described as a method of eliciting social support from others.

If one conceptualizes catastrophizing as a cognitive error, such as those proposed by a Beckian model of depression, interventions should be similar to those used in the treatment of depression. Cognitive restructuring is the typical strategy for reducing dysfunctional thinking

among depressives. This approach focuses on identifying automatic, maladaptive cognitions and replacing these with more rational and realistic thoughts (Beck, 1995).

If one views catastrophizing as an appraisal process that results in heightened attention to pain, perceptions of threat, and expectations of heightened pain, interventions would most likely entail attempts to change attentional focus from catastrophic thinking to distraction strategies and other coping strategies. This would theoretically result in a reduction in attentional resources devoted to catastrophic thinking and pain perception (Eccleston & Crombez, 1999).

Although it may seem counterintuitive to adopt the theory that catastrophizing is a method of coping, there have been recent suggestions that catastrophizing may indeed be employed in an effort to garner social support from others (Sullivan et al., 2000). Sullivan and his colleagues have proposed that catastrophizing might represent a 'communal' approach to dealing with the distress of pain. This model emerged from research showing that individuals who catastrophized not only experienced more pain, but were also more expressive during their pain experience (Sullivan et al., 2000).



A Communal
Coping Model of
Catastrophizing

Individuals differ in the manner in which they express or display their pain experience. Some individuals experience high levels of pain but show

little outward evidence that they are in pain. Others are very expressive of their pain experience. When individuals express their pain through various behaviours such as grimacing or distress vocalizations they are likely to attract the attention of other people in their social environment. It is possible that non-catastrophizers may prefer to deal with their pain in a solitary fashion and may minimize their display of distress in order to prevent social attention from being drawn to them. Catastrophizers however might prefer a 'communal' approach to coping; in other words they might prefer to deal with distress in the presence of others from their social environment. The expression of distress may be a necessary component of an interpersonal or communal approach to coping. It is only through the clear communication of distress that others in one's social environment will be able to determine that assistance is required.

Although there has been considerable discussion of the 'maladaptive' nature of catastrophizing, it is also necessary to consider that there may be adaptive dimensions to catastrophizing. Research that has emerged for the past two decades indicates that individuals who catastrophize are more attentive to pain signals and more expressive of their current physical and emotional distress (Sullivan et al., 2001). If we consider that pain is often a signal of tissue damage, increased attention to pain signals may be quite adaptive. Increased attention to pain signals and effective communication of pain signals may facilitate early detection and treatment of serious illness.

Sullivan et al. (2001) suggested that catastrophizers may engage in exaggerated pain expression in order to maximize proximity, or to solicit assistance or empathic responses from others in their social environment. Unfortunately, in attaining these social goals, catastrophizers' may inadvertently make their pain experience more aversive. Catastrophizers' increased attention to their pain and their exaggerated display of pain behavior may become maladaptive by actually contributing to heightened pain experience. In addition, others' solicitous or reinforcing responses may serve to trigger, maintain, or reinforce catastrophizers' exaggerated pain expression.

Although the coping style of high catastrophizers may appear maladaptive, it is important to consider that a communal coping style may only become truly maladaptive under chronic pain or chronic illness conditions. In response to acute pain, exaggerated pain displays may result in a precarious, but sustainable, balance between satisfying support or affiliative needs, and increasing pain-related distress. Under acute pain conditions, overall benefits may outweigh costs, and reinforcement contingencies (e.g., increased support, attention, empathic responses) may actually serve to maintain the expressive style of high catastrophizers. When conditions become chronic, this balance may be

disrupted such that costs begin to outweigh the benefits. Social environmental responses may become increasingly negative when distress displays extend over a period of time. The disrupted balance may find expression as increased interpersonal conflict, social rejection and depression. (see Thorn et al., 2004; Cano, 2004)

Peterson and Moon (1999) have raised the interesting possibility that catastrophizing may initially emerge as a result of exposure to traumatic life events. In other words, catastrophic thinking may have its origins in reality. Peterson and Moon (1999) propose that life traumas such as major losses, severe accidents, and abuse experiences may sensitize individuals to distress reactions to future stressors.

The Role of Attention

Attention to pain symptoms appears to be one of the mechanisms by which catastrophizing contributes to increased physical and emotional distress. For example, the rumination subscale of the Pain Catastrophizing Scale has been shown to be most highly correlated with pain-outcomes. In other words, the endorsement of items, such as 'I keep thinking about how much it hurts' and 'I can't seem to keep it out of my mind' have been most consistently associated with more severe pain symptoms. Sullivan and Neish (1998) found that only the rumination component of pain catastrophizing contributed significant unique variance to the prediction of pain intensity during dental hygiene treatments. In addition, Sullivan et al. (1998) reported that only the rumination contributed significant unique variance to the prediction of pain-related disability in a sample of patients with soft tissue injuries.

The importance of attentional mechanisms associated with pain catastrophizing has been discussed by several investigators. Crombez et al (1997) reported that pain catastrophizers showed greater interference on attention-demanding task than non-pain-catastrophizers in anticipation of a pain stimulus onset. Heyneman et al (1990) showed that pain catastrophizers were unsuccessful in using cognitive attention diversion coping strategies to reduce their pain. Similarly, Sullivan et al (1997) provided data suggesting that pain catastrophizers may be impaired in their ability to divert attention away from pain. Eccleston and his colleagues (1997) have suggested that excessive focus on pain sensations may lead to the facilitation of pain access into consciousness and the magnification of painful sensations. Recent neuro-imaging studies have shown that brain areas responsible for attentional modulation are more likely to be activated in high catastrophizers during the experience of pain (Gracely et al., 2004; Seminowitz et al., 2006)

Catastrophizing and Attention to Pain Sensations



Attentional mechanisms have frequently been cited as a significant mediator of heightened somatic complaints. Watson and Pennebaker (1989) have suggested that internally focused attention may be one of the factors that underlies the relation between negative emotional states and heightened physical complaints. Artzn and his colleagues (1994) have reported that attention may be the primary mediator of anxiety effects on pain experience. It has been suggested that attention to pain sensations might actually increase sensory flow of pain signals to the brain (Eccleson and Crombez, 1999). It is possible that attentional focus may represent one of the final common pathways through which many cognitive and affective variables impact on pain experience.

Catastrophizing and the Development of a Chronic Hyperalgesic State



Ronald Melzack and his colleagues of McGill University have recently proposed a 'neural matrix' model of pain suggesting that although the processing of pain by the brain is genetically specified, such processing is modified by experience. Factors that increase sensory flow of pain signals, may, over time, actually alter central thresholds of excitability, thereby increasing sensitivity to pain. By engaging in cognitive activity that amplifies pain signals, catastrophizers' central neural mechanisms may become more sensitized, yielding a chronic hyperalgesic state.

Neuroimaging research has shown that focusing attention on pain may activate a distributed network of brain regions, including prefrontal and parietal areas, parts of the anterior cingulate cortex, and the thalamus (Bushnell et al. 2004; Derbyshire et al. 1997; Peyron et al. 2000). During painful stimulation, some regions of the "attentional network" have been shown to be significantly more activated in high pain catastrophizers, particularly the dorsolateral prefrontal cortex, the anterior cingulate cortex, and the inferior parietal cortex (Gracely et al. 2004; Seminowicz and Davis 2006). These findings provide neural evidence that attentional mechanisms might account, at least in part, for the relation between catastrophic thinking and pain experience (Seminowicz and Davis 2006; Sullivan et al. 2001b).

Research may ultimately reveal that the relation between catastrophizing and central nociceptive mechanisms is bi-directional. This line of reasoning suggests that although the processes that underlie the relation between catastrophizing and pain may initially be psychological in nature, experience-based changes in neural sensitivity may be such that these processes come increasingly under physiological control. The potential self-sustaining nature of a bi-directional relation between catastrophizing and nociceptive processing may be one of the factors that contributes to the chronicity of many pain conditions.

The findings highlighting a relation between catastrophic thinking and attention to pain symptoms suggests that interventions aimed at minimizing catastrophic thinking will need to incorporate strategies for assisting catastrophizers in disengaging their attention from their pain symptoms.

The Role of Emotion

A basic tenet of cognitive theories of emotion is that negative cognitions can lead to negative emotions (Banks and Kerns 1996; Beck et al. 1978; Lazarus and Folkman 1984). Researchers have appealed to variations of this general framework to understand the relation between catastrophic thinking and negative emotional reactions (Turner and Aaron 2001;

Vlaeyen and Linton 2000). The relations among pain catastrophizing, fear and depression have been the focus of numerous investigations (Keefe et al. 2005; Sullivan and D'Eon 1990). Research has been consistent in showing that measures of catastrophic thinking are significantly correlated with measures of depression, anxiety, and fear (Borsbo et al. 2008; Drahovzal et al. 2006; Edwards et al. 2006a; Edwards et al. 2006b; Leeuw et al. 2007). Keefe et al (Keefe et al. 1989) reported that pain catastrophizing prospectively predicted depressive symptoms in a sample of individuals with arthritis. The pattern of findings that has emerged suggests that catastrophic thinking might contribute to the development or maintenance of anxiety, fear or depression associated with pain.

The study of the relation between emotion and pain dates back several decades (Craig 1989; Schwarz 1962). There is a sizeable literature that has examined the relation between trait measures of emotional distress and pain outcomes. Numerous investigators have reported significant cross-sectional and prospective relations between trait measures of depression, anxiety, fear and anger, and heightened pain experience (Banks and Kerns 1996; Leeuw et al. 2007; Rudy et al. 1988; Sullivan and Neish 1998; Turk 1996; Turk and Okifuji 2002; Vlaeyen et al. 1995). For example, Smith and Zautra (2008) reported that anxiety was prospective related to heightened pain intensity in a sample of women with arthritis (Smith and Zautra 2008). Carroll et al (2004) reported that depressive symptoms might increase susceptibility to exacerbation of musculoskeletal pain symptoms (Carroll et al. 2004).

Fewer studies have addressed the role of situation-specific or experimentally induced emotional distress on responses to painful stimulation. Findings from experimental studies are not entirely consistent with the pattern of findings using trait measures of emotional distress. For example, Meagher et al (Meagher et al. 2001) examined the effects of viewing emotional slides prior to participating in an experimental pain procedure. Their findings indicated that viewing slides of fear or disgust resulted in a decrease as opposed to an increase in pain intensity. However, consistent with the research using trait measures of emotional distress, Carter et al (Carter et al. 2002) reported that experimental induction of negative emotions (i.e., anxiety, depression) led to increased pain severity during a cold pressor task. Tang et al (Tang et al. 2008) reported that listening to sad music led to more intense pain experience and lower pain tolerance in chronic back pain patients. Thus, although the research on the effects of situation-specific negative mood on pain is not as consistent as the literature using trait measures of emotional distress, the findings point to a possible hyperalgesic effect of emotional distress in both healthy individuals and chronic pain patients.

Studies using functional brain imaging techniques have identified a number of brain areas responsible for producing emotional/affective responses associated with pain, including feelings of unpleasantness and distress. For example, studies have been consistent in showing that painful stimulation leads to increased neural activity in the anterior cingulate and insular cortices, both part of the limbic system (for a review, see (Apkarian et al. 2005). It is generally assumed that neural activity in limbic areas contributes to heightened pain experience by increasing the emotional valence attributed to pain sensations.

Recent efforts have been made to examine the neural mechanisms underlying the effects of emotional states on pain processing. For example, Phillips et al (Phillips et al. 2003) have shown that experimentally induced negative mood can enhance neural activity in cingulate and insular cortices during visceral stimulation, leading to increased levels of pain-related discomfort. Similarly, Ploghaus et al (Ploghaus et al. 2001) have shown that experimentally induced anxiety can lead to hyperalgesic responses and increased neural activity in a number of brain areas associated with pain processing. Specifically, it has been shown that high levels of anxiety prior to painful heat stimulation can increase activity in the medial prefrontal cortex, the anterior cingulate cortex and parts of the hippocampal formation. These areas are considered to be directly involved in the amplification of pain experience and provide a neural basis for the effects of emotion on pain (Schweinhart et al. 2008; Tracey and Mantyh 2007).

There is reason to believe that pain catastrophizing might influence pain experience through similar neural mechanisms to those involved in the relationship between emotional distress and pain. During painful stimulation, Seminowicz and Davis (Seminowicz and Davis 2006) found that pain catastrophizing was significantly associated with activity in the medial prefrontal cortex, the anterior cingulate cortex, the insula and parts of the hippocampal formation. Pain-evoked neural activity in some of these regions has been associated with negative affect (Phillips et al. 2003) (Ploghaus et al. 2001), suggesting that pain catastrophizing is likely to overlap with other emotional processes in modulating brain responses to pain. These neuroimaging findings also suggest that pain catastrophizing might emotional distress, facilitating nociceptive processing in cortico-cortical circuits and augmenting the overall pain experience.

Endogenous pain modulation

There are some indications that pain catastrophizing might have a direct impact on endogenous pain modulation mechanisms. As noted above,

research suggests that pain catastrophizers might benefit less from rehabilitation interventions for chronic pain. There is also research to suggest that pain catastrophizing might interfere with the effectiveness of pharmacological interventions for pain. Haythornthwaite et al (Haythornthwaite et al. 2003) reported the findings of a study assessing the efficacy of an opiate medication for post-herpetic neuralgia. Analyses revealed that initial pain catastrophizing scores predicted higher post-treatment pain ratings, even when controlling for baseline pain. Sullivan et al (Sullivan et al. 2008b) reported that catastrophizing was associated with poor response to a topical analgesic for neuropathic pain. In an experimental study investigating psychological factors related to pain perception and analgesia, Fillingim et al. (Fillingim et al. 2005) found that catastrophizing in men was associated with poor overall analgesic responses to intravenous pentazocine.

The mechanisms by which psychological factors interfere with response to analgesics remain unclear. It has been suggested that individuals high in catastrophizing might produce endogenous nocebo-like responses due to their negative cognitions (Fillingim et al. 2005). It has also been suggested that catastrophizing might compromise processes involved in descending inhibition of pain (Edwards and Fillingim 2001). For example, in a temporal summation paradigm, Edwards et al (Edwards et al. 2006c) found that individuals with high levels of catastrophizing reported significantly greater increases in pain ratings than individuals with low levels of catastrophizing during the application of repeated painful heat stimulations. Similarly, George et al (George et al. 2006) found that pain catastrophizing was a significant predictor of increases in pain ratings across repeated noxious heat pulses, even when controlling for sex and pain-related fear. These findings suggest that pain catastrophizing may facilitate processes involved in temporal summation of pain or 'windup' (Price et al. 2002). The findings also suggest that pain catastrophizing might interfere with descending pain-inhibitory systems, facilitate neuroplastic changes in the spinal cord during repeated painful stimulation, subsequently promoting sensitization in the CNS.

Other studies have also established a link between pain catastrophizing and the operation of endogenous pain-modulatory systems. For example, two recently published papers have reported a negative association between pain catastrophizing and diffuse noxious inhibitory controls, a psychophysical measure of endogenous pain inhibition (Goodin et al. 2008; Weissman-Fogel et al. 2008). On the basis of findings such as these, it has been suggested that pain catastrophizing might directly interfere with the efficacy of endogenous pain-inhibitory mechanisms (Goodin et al. 2008).

Intervention Implications

Research examining potential mediators of the relation between catastrophizing and pain outcomes might have important implications for the development of targeted interventions aimed at reducing pain catastrophizing, or minimizing the negative impact of pain catastrophizing on pain outcomes. Although numerous treatment studies have been shown to have an effect on catastrophic thinking, the critical elements of effective treatments for yielding meaningful change have yet to be identified.

Considerable research supports the view that pain catastrophizing is a modifiable variable (Keefe et al. 2005; Sullivan et al. 2005a). In the absence of intervention, pain catastrophizing shows some degree of stability over time (Sullivan et al. 2001b). However, numerous intervention studies have shown that catastrophic thinking decreases as a result of participation in treatment aimed at facilitating recovery or adaptation to chronic pain (Jensen et al. 2001; Smeets et al. 2006; Spinhoven et al. 2004). Many of these studies have pointed to importance of reducing pain catastrophizing as a key factor in determining the success of interventions for chronic pain (Spinhoven et al. 2004; Sullivan et al. 2005b).

Jensen et al (2001) reported that participation in a 3-week (82 hours) multidisciplinary pain treatment program led to a 40% reduction in scores on a measure of catastrophizing (Jensen et al. 2001). Treatment-related changes in pain catastrophizing rose significantly at 6-month follow-up, but remained below baseline levels. Sullivan et al (2003) reported a 33% reduction in catastrophizing scores following participation in a 10-week (10 hours) psychological intervention (led by psychologists) designed to target psychosocial risk factors for pain and disability (Sullivan and Stanish 2003). Sullivan et al (Sullivan et al. 2006a) reported a 43% reduction in catastrophizing following participation in a 10-week program (50 hours) consisting of exercise and a psychosocial intervention (led by occupational therapists and physiotherapists) targeting risk factors for pain and disability. Adams et al (Adams et al. 2007) reported that reductions in pain catastrophizing following a 10-week (50 hours) treatment program consisting of exercise and a psychosocial intervention varied as a function of level of chronicity. For patients in the subacute (4 weeks to 3 months) and early chronic period (3 – 6 months) of recovery, pain catastrophizing scores showed a reduction of 39%. For patients whose condition had become chronic (+ 6 months), pain catastrophizing scores decreased by only 10%.

There are also indications that psychological intervention might not be essential to yield reductions in catastrophic thinking. Sullivan et al (Sullivan et al. 2006a) reported a 24% reduction in pain catastrophizing scores following participation in a 10-week (45 hours) physical therapy intervention. Another study reported a 27% decrease in pain catastrophizing scores following a 4-week (100 hours) functional restoration exercise program (Sullivan et al. 2008a). Smeets et al (Smeets et al. 2006) reported no significant difference in the magnitude of reduction in pain catastrophizing scores for patients who participated in active physiotherapy (-12%), problem-solving therapy (-10%) or combined treatment (-10%). In the latter study, each treatment program consisted of approximately 11 hours of intervention.

The research conducted to date suggests that catastrophic thinking associated with pain can be reduced through a variety of means. However, some degree of caution must be exercised in the interpretation of the results of the studies described above. Studies vary in terms of the nature of the population being treated (recent onset versus long term disability; low back pain versus whiplash), the intensity of treatment (10 to 100 hours), the insurance context within which clients are treated (no fault versus tort), and the objectives of the intervention (pain management, functional improvement, or return to work). Initial values (high versus low) on measures of pain catastrophizing will play a role in determining the magnitude of reductions that will be observed and the relation between reductions in pain catastrophizing and clinical outcomes. In a related manner, there is currently limited information about the magnitude of reduction in pain catastrophizing scores that is required to impact in a clinically meaningful manner on pain outcomes.

Education, activity resumption and instruction in self-management skills characterize the content of most multidisciplinary programs for the management of chronic pain (Gatchel et al. 2007). It is not unreasonable to assume that each of these elements might impact directly or indirectly on catastrophic thinking. As noted earlier, catastrophizing has been discussed as a multidimensional construct comprising rumination, magnification and helplessness. Intervention techniques that impact on any of these dimensions might yield therapeutic benefit. Education might permit individuals to re-evaluate or re-appraise the degree of threat they associate with their condition or their participation in activity (Moseley 2004; Turk 2004). Participation in exercise or other physical activity might yield benefit by reducing the cognitive resources that can be allocated to catastrophic thinking. Activity participation and instruction in self-management skills might increase self-efficacy and, in turn, reduce the helplessness dimension of catastrophizing.

In recent years, several treatment programs have been developed to specifically target psychological risk factors for pain and disability (Hasenbring et al., 1999; Linton & Andersson, 2000; Sullivan & Stanish, 2003; Thorn et al., 2001; Van den Hout, Vlaeyen, Heuts, Zijlema, & Wijnen, 2003; Vlaeyen, De Jong, Onghena, Kerckhoffs-Hanssen, & Kole-Snijders, 2002). Treatment programs have typically taken the form of structured cognitive and/or behavioural interventions aimed at minimizing levels of psychological risk factors. Numerous investigations have shown that risk factor targeted interventions can have a significant impact on improving function and facilitating return to work. Linton and his colleagues have shown that a 6-week cognitive-behavioural intervention was effective in reducing absenteeism in a sample of workers considered at risk for the development of chronic pain (Linton, 2002; Linton & Andersson, 2000; Linton & Ryberg, 2001). Hasenbring and her colleagues have reported significant reductions in pain following a risk factor targeted intervention for patients with sciatica (Turk, 2002). Graded activity interventions aimed at reducing fear of movement have been shown to be effective in facilitating return to work and reducing absenteeism (Van Den Hout et al., 2003; Lindstrom et al., 1992; George, Fritz, Bialosky, & Donald., 2003). Sullivan and Stanish (2003) have shown that an intervention program targeting pain catastrophizing, fear of re-injury, perceived disability and depression can significantly increase the probability of return to work following occupational injury.

The rationale that has driven the development and implementation of risk-factor targeted interventions is that the reduction in risk factors should facilitate rehabilitation progress and return to work potential following occupational injury (Pincus, Burton, et al., 2002; Pincus, Vlaeyen, et al., 2002; Feuerstein, Berkowitz, & Huang, 1999).

Our Centre has developed a community-based risk factor targeted intervention program aimed at minimizing psychological risk factors for chronicity. The Progressive Goal Attainment Program is a 10-week standardized behavioural-cognitive intervention that aims to increase activity involvement during the post injury period, and minimize psychological barriers to rehabilitation progress (Sullivan & Stanish, 2003). Psychological risk factors specifically targeted in the intervention include fear of movement/re-injury, catastrophizing, perceived disability and depression.

Overall, participation in the PGAPP Program has been associated with return to work rates of approximately 62% in individuals absent from work for over 6 months. For individuals off work between 3 and 6 months, return to work rates were 78%; the probability of return to work decreased as the duration of work absence increased. Univariate analyses revealed that pre- to post-treatment reductions in all risk factors assessed are

associated with higher probability of returning to work. However, there was a high degree of shared variance among predictors such that not all variables contributed unique variance to the prediction of return to work. Logistic regression revealed that initial levels of pain catastrophizing and fear of movement/re-injury contributed uniquely to predicting return to work. Reductions in catastrophizing were also related to increased likelihood of returning to work. Initial levels of and reductions in pain, depression, and perceived disability did not contribute uniquely to prediction of return to work. Although reductions in all risk factors assessed were significantly related to treatment outcome, the findings indicated that only reductions in catastrophizing contribute significant unique variance to the prediction of return to work. The results of this research suggest that interventions that aim to reduce levels of pain catastrophizing may be associated with the best return to work outcomes.

For more information information on intervention programs aimed at reducing pain catastrophizing, please consult our website:

www.pdp.pgap.com

The results showing that risk factor reduction is associated with higher probability of returning to work have important implications for the nature of early interventions for pain-related occupational injury. Psychosocial interventions have been under-represented in secondary prevention programs. It has been common practice to involve psychosocial service providers primarily in the treatment of individuals with long standing pain and disability, where treatment goals are often more palliative in nature, with a focus on the consequences of injury as opposed to risk factors for chronicity. The incorporation of risk-factor targeted psychosocial interventions in the early stages of recovery from injury holds promise of yielding significant improvement in rehabilitation outcomes for individuals who are at risk of following a trajectory of prolonged pain-related disability.

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APPENDIX A

PCS Raw Scores and Percentiles

PCS Total	Rumination	Magnification	Helplessness	Percentile
				1
0				2
	0			3
				4
2				5
3	1		0	6
				7
				8
4				9
			1	10
5				11
				12
	2			13
		0		14
6				15
			2	16
7				17
				18
8	3			19
				20
9				21
			3	22
				23
10				24
				25
	4			26
		1		27
11				28
			4	29
				30
12	5			31
				32
				33
13				34
14				35
			5	36
				37
	6			38
15				39
				40
16				41
		2	6	42
				43
17	7			44
				45
				46
18			7	47
19				48
				49
-20-	-8-	-3-	-8-	-50-

PCS Total	Rumination	Magnification	Helplessness	Percentile
				51
				52
21				53
				54
22			9	55
				56
23				57
	9			58
				59
24			10	60
				61
25				62
		4		63
			11	64
26	10			65
				66
27				67
				68
28				69
			12	70
				71
				72
29				73
				74
30	**11**	**5**	**13**	75
				76
				77
31				78
		6	14	79
32	12			80
33				81
				82
			15	83
34				84
35				85
	13	7		86
36			16	87
37				88
			17	89
38		8		90
39	14			91
40			18	92
41		9		93
42				94
43			19	95
44	15			96
45		10	20	97
46		11	21	98
47-48			22	99
49-52	16	12	23-24	100

APPENDIX B

PCS: English and French Versions



PCS

Client No.: _____ Age: _____ Sex: M() F() Date: _____

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

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

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

When I'm in pain ...

- 1 I worry all the time about whether the pain will end.
- 2 I feel I can't go on.
- 3 It's terrible and I think it's never going to get any better.
- 4 It's awful and I feel that it overwhelms me.
- 5 I feel I can't stand it anymore.
- 6 I become afraid that the pain will get worse.
- 7 I keep thinking of other painful events.
- 8 I anxiously want the pain to go away.
- 9 I can't seem to keep it out of my mind.
- 10 I keep thinking about how much it hurts.
- 11 I keep thinking about how badly I want the pain to stop.
- 12 There's nothing I can do to reduce the intensity of the pain.
- 13 I wonder whether something serious may happen.

... Total



PCS-CF

Nom: _____ Age: _____ Sexe: _____ Date: _____

Chacun d'entre nous aura à subir des expériences douloureuses. Cela peut être la douleur associée aux maux de tête, à un mal de dent, ou encore la douleur musculaire ou aux articulations. Il nous arrive souvent d'avoir à subir des expériences douloureuses telles que la maladie, une blessure, un traitement dentaire ou une intervention chirurgicale.

Dans le présent questionnaire, nous vous demandons de décrire le genre de pensées et d'émotions que vous avez quand vous avez de la douleur. Vous trouverez ci-dessous treize énoncés décrivant différentes pensées et émotions qui peuvent être associées à la douleur. Veuillez indiquer à quel point vous avez ces pensées et émotions, selon l'échelle ci-dessous, quand vous avez de la douleur.

0 – pas du tout 1 – quelque peu 2 – de façon modéré 3 – beaucoup 4 – tout le temps

Quand j'ai de la douleur ...

- 1 j'ai peur qu'il n'y aura pas de fin à la douleur.
- 2 je sens que je ne peux pas continuer.
- 3 c'est terrible et je pense que ça ne s'améliorera jamais.
- 4 c'est affreux et je sens que c'est plus fort que moi.
- 5 je sens que je ne peux plus supporter la douleur.
- 6 j'ai peur que la douleur s'empire.
- 7 je ne fais que penser à d'autres expériences douloureuses.
- 8 avec inquiétude, je souhaite que la douleur disparaisse.
- 9 je ne peux m'empêcher d'y penser.
- 10 je ne fais que penser à quel point ça fait mal.
- 11 je ne fais que penser à quel point je veux que la douleur disparaisse.
- 12 il n'y a rien que je puisse faire pour réduire l'intensité de la douleur.
- 13 je me demande si quelque chose de grave va se produire.

... Total