The Clinical Journal of Pain
17:52–64 © 2001 Lippincott Williams & Wilkins, Inc., Philadelphia

Theoretical Perspectives on the Relation Between Catastrophizing and Pain

*Michael J. L. Sullivan, Ph.D., †Beverly Thorn, Ph.D., ‡Jennifer A. Haythornthwaite, Ph.D., §Francis Keefe, Ph.D., ‖Michelle Martin, Ph.D., ‖Laurence A. Bradley, Ph.D., and ‖¶John C. Lefebvre, Ph.D.

**Dalhousie University, Halifax, Nova Scotia; †University of Alabama at Tuscaloosa, Tuscaloosa, Alabama; ‡Johns Hopkins University, Baltimore, Maryland; §Duke University, Durham, North Carolina; ‖University of Alabama at Birmingham, Birmingham, Alabama; ¶Wofford College, Spartanburg, South Carolina*

Abstract:
The tendency to "catastrophize" during painful stimulation contributes to more intense pain experience and increased emotional distress. Catastrophizing has been broadly conceived as an exaggerated negative "mental set" brought to bear during painful experiences. Although findings have been consistent in showing a relation between catastrophizing and pain, research in this area has proceeded in the relative absence of a guiding theoretical framework. This article reviews the literature on the relation between catastrophizing and pain and examines the relative strengths and limitations of different theoretical models that could be advanced to account for the pattern of available findings. The article evaluates the explanatory power of a schema activation model, an appraisal model, an attention model, and a communal coping model of pain perception. It is suggested that catastrophizing might best be viewed from the perspective of hierarchical levels of analysis, where social factors and social goals may play a role in the development and maintenance of catastrophizing, whereas appraisal-related processes may point to the mechanisms that link catastrophizing to pain experience. Directions for future research are suggested.
Key Words: Catastrophizing—Pain—Depression—Disability

In "Sur L'eau," novelist Maupassant[1] 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 he is helpless to deal with it. He surrenders to the pain and seeks chemical means of dulling it. Maupassant's words emphasize the psychological components of pain perception; the sensory, cognitive, affective, and behavioral dimensions of his experience. Specialists of the psychology of pain would argue that Maupassant's "catastrophic" orientation to his pain likely played a role in heightening the intensity of the pain he experienced.[2–4]

It is of interest that early theories of pain focused almost exclusively on the role of physiologic processes. For example, Descartes' pain specificity hypothesis addressed pain in purely mechanistic terms, where pain intensity was posited to be a direct function of the degree of tissue damage.[5] But anecdotal reports and scientific investigations showed that a purely physiologic view of pain could not account for the wide range of reactions observed in response to painful stimulation.[6–8] In recent years, it has become increasingly clear that psychological factors are important determinants of pain experience.[7–10]

A growing amount of literature shows that the tendency to "catastrophize" during painful stimulation contributes to more intense pain and increased emotional

Received April 20, 2000; revised August 18, 2000; accepted September 10, 2000.
Address correspondence and reprint requests to Dr. Michael Sullivan, Department of Psychology, Dalhousie University, Halifax, Nova Scotia, Canada, B3H 4J1; e-mail: sully@is.dal.ca

distress.[2,3,11] Catastrophizing has been broadly conceived as an exaggerated negative "mental set" brought to bear during actual or anticipated pain experience. In the literature that has emerged during the past 2 decades, catastrophizing has risen to the status of one of the most important psychological predictors of pain experience. Surprisingly however, research in this area has proceeded in the relative absence of a guiding theoretical framework. Theoretical accounts of catastrophizing have not been elaborated substantively beyond investigators' operational definitions of the construct. The absence of a guiding theoretical framework has impeded systematic inquiry into the antecedents, consequents, determinants, and mechanisms of action of catastrophizing. Questions also remain regarding the contextual determinants of catastrophizing, the malleability of catastrophizing, the behavioral dimensions of catastrophizing, and the relation between catastrophizing and the effectiveness of different coping strategies. These questions are of interest not only for their theoretical relevance but also for their relevance to the clinical management of pain.

The main purpose of this article is to provide an overview of the research that has been performed in regard to catastrophizing and pain. From a review of available findings, efforts will be made to address the theoretical models that are able to account for the body of existing literature. The relative strengths and limitations of these models will be examined, and future directions for research will be discussed.

The nature of catastrophizing

To grasp the essence of current conceptualizations of catastrophizing, it is useful to consider four articles that have provided a foundation for the literature on catastrophizing.[12–15] Chaves and Brown[12,13] asked dental patients to report thoughts and images they experienced, or the strategies they engaged in, during a stressful 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 wonder whether something serious may happen"). Spanos et al.[15] interviewed individuals about their pain experience after a cold pressor task. 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"). Rosenstiel and Keefe[14] reported on the development of the Coping Strategies Questionnaire (CSQ), which consists of seven coping subscales, including a catastrophizing subscale. The items on the catastrophizing subscale reflect elements of helplessness and pessimism in relation to ability to deal with the pain experience (i.e., "It's terrible and it's never going to get any better").

Two issues arise from this early work. First, although the different studies show consensus in construing catastrophizing in terms of negative pain-related cognitions, they differ in their emphasis on the content of these cognitions. To address this issue, Sullivan et al.[3] developed the Pain Catastrophizing Scale (PCS) using examples of catastrophic thinking drawn from each of these earlier studies.[12–15] Factor analysis yielded a correlated three-factor solution, suggesting that catastrophizing could be viewed as a unitary construct comprising three different dimensions (i.e., magnification, rumination, helplessness). Subsequent studies replicated the factor structure of the PCS.[16,17]

A second issue, which has not been addressed empirically, concerns the conceptualization of catastrophizing as a continuous versus dichotomous variable. Most studies have treated catastrophizing as a continuous variable. In research using patients with chronic pain, the distribution of CSQ and PCS scores does not depart significantly from normality (examination of raw data[14,18,19]). In research that used a dichotomous typology,[12,15,20] data on the frequency distribution have not been presented; therefore, it is unclear whether a dichotomous typology would be supported.

The evidence

The evaluation of conceptual frameworks for catastrophizing must proceed by determining the degree to which available frameworks can account for available findings. The following section reviews the findings examining the relation among catastrophizing, pain, and pain-related outcomes.

Catastrophizing and pain

One of the most consistent findings has been that catastrophizing is associated with heightened pain experience. In zero-order correlations, catastrophizing accounts for 7 to 31% of the variance in pain ratings. The relation between catastrophizing and pain has been observed across measures and in diverse patient groups, including mixed chronic pain,[18] low back pain,[21] rheumatoid arthritis,[2] aversive diagnostic procedures,[3] (Study 3), surgery,[20,22] dental procedures,[23] burn dressing changes,[24] whiplash injuries,[25] and survey samples of young adults,[26] asymptomatic individuals participating in experimental pain procedures,[3,27] and varsity athletes.[17]

Other studies have used factor analysis to identify composite measures that include catastrophizing. Factor analysis of the CSQ yielded a Pain Control and Rational Thinking factor that contained the catastrophizing

subscale and the two coping effectiveness items and accounted for significant variance in pain.[4] Similar findings have been reported in patients with rheumatoid arthritis,[28] chronic low back pain,[29] and sickle-cell disease.[30]

The relation between catastrophizing and pain also has been reported in younger samples. In a survey of junior high school students, Bédard et al.[31] found that individuals who catastrophized reported more intense pain than noncatastrophizers for all types of pain examined. Responses of patients with juvenile rheumatoid arthritis to the Pain Coping Questionnaire[32] yielded a factor containing the catastrophizing subscale that correlated positively with pain intensity ratings. A similar procedure using the CSQ adapted for children (CSQ-C) showed that the factor containing catastrophizing correlated significantly with pain intensity and pain location in juvenile rheumatoid arthritis[33] and juvenile primary fibromyalgia.[34] Catastrophizing also has predicted pain threshold and pain intensity in juvenile arthritis patients exposed to a cold pressor procedure.[35]

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[36] or 10 weeks[3] before a painful procedure predict pain ratings. Catastrophizing prospectively predicted pain ratings in patients with arthritis 6 months later, even when controlling for initial pain ratings.[2]

Given that the bulk of research examining the relation between catastrophizing and pain has been cross-sectional in design, it is necessary to consider that intense pain may cause catastrophic thinking. The reactive nature of catastrophizing has not been systematically investigated. Nevertheless, in support of a reactivity position, there are indications that catastrophizing scores in asymptomatic undergraduates or dental patients[3,23] are less than those seen in patients with chronic pain patients.[19] Although suggestive, it is important to note that cohort factors, as opposed to the presence of pain, may be responsible for cross-sample differences in catastrophizing. More research is needed to address how catastrophic thinking is influenced by pain experience.

Catastrophizing, pain behavior, and illness behavior

Catastrophizing has been associated with a wide range of pain and illness behaviors. Pain behavior refers to the different motor and verbal responses emitted in response to the experience of pain.[8] Pain behaviors that take the form of help-seeking or excessive preoccupation with symptom management have been referred to as illness behavior. The Pain Control and Rational Thinking factor of the CSQ has been associated with higher observed frequency of pain behaviors in patients with osteoarthritis of the knee[11,37] and patients who underwent knee replacement surgery.[38] Sullivan et al.[39] showed associations between catastrophizing and the total duration of pain behavior during a cold pressor test. Catastrophizing concurrently and prospectively (after treatment) predicted self-reported pain behavior in a sample of patients with fibromyalgia.[40]

Catastrophizing has also been associated with illness behaviors, including the frequency and duration of hospital stay,[41] use of staff-administered analgesics after breast cancer surgery,[20] more frequent visits to healthcare professionals,[42] and use of over-the-counter medication.[31] It is interesting to speculate that more frequent, more pronounced, or longer duration of pain behaviors may prompt health professionals to pursue more intensive and more invasive approaches to pain assessment and treatment.

Little is known about the relation between catastrophizing and pain behavior in the natural environment of the individual. It is possible that the *help seeking* context of clinical settings may give rise to exaggerated displays of pain and illness behavior. But it is equally possible that because clinical and experimental settings do not contain the environmental or social cues that typically trigger or reinforce displays of pain behavior, these settings may actually render an underestimate of the relation between catastrophizing and pain behavior. More research is needed to explore the social and contextual determinants of catastrophizing and pain behavior.

Catastrophizing and disability

Disability refers to the activity restrictions or limitations that are associated with a physical or mental impairment.[43] In the context of most persistent pain disorders, *pain* is considered to be the impairment that contributes to disability, significantly affecting social and occupational functioning.[44] As a result of the high costs associated with pain-related disability, considerable research[45,46] has been aimed at identifying determinants of disability in individuals with persistent pain.

A number of studies have examined the relation between specific measures of catastrophizing and indices of disability across diverse clinical settings. The association between catastrophizing and disability has been observed in a mixed group of patients with chronic pain,[47]

patients with fibromyalgia,[48] and patients after soft-tissue injury,[19,25] and these relations are observed across multiple measures of disability. Catastrophizing has been associated with heightened disability, even when controlling for depression, anxiety, neuroticism, disease severity,[48] and pain severity.[19,25] Finally, in a prospective study patients with rheumatoid arthritis, initial levels of catastrophizing predicted perceived disability scores obtained 6 months later, even when controlling for initial perceived disability, age, gender, compensation status, and duration of illness duration.[2]

Several investigations have also shown that factors including catastrophizing are associated with objective and subjective indices of disability. The CSQ factor containing the catastrophizing subscale correlates with patient downtime,[30,49] dexterity, mobility, and household activities scales of the Arthritis Impact Measurement Scale,[4] higher levels of perceived disability,[28] and activity reduction.[29] The CSQ factor containing the catastrophizing subscale also has been shown to predict functional impairment in adults with fibromyalgia,[40] with rheumatoid arthritis,[50] and after knee replacement surgery,[11,38] and in children with fibromyalgia.[34]

The relation between specific components of catastrophizing and disability may vary as a function of duration of pain. For example, the magnification subscale of the PCS was the best predictor of pain and disability in a sample of patients with whiplash who were approximately 1 year postinjury,[25] whereas the rumination subscale was the best predictor of severity of disability in patients who had been experiencing pain for approximately 3 years.[19] Later in the course of chronic low back pain, the helplessness subscale has been shown to be the best predictor of severity of disability.[51] Although cross-study differences in sample composition limit the strength of conclusions that can be drawn, these findings suggest that the nature of catastrophic cognitions associated with disability may change as the pain condition becomes more chronic.

Catastrophizing and gender

A number of studies have reported that women score higher than men on measures of catastrophizing. Gender differences in levels of catastrophizing have been observed among patients with musculoskeletal pain[52] or osteoarthritis of the knee,[37] undergraduates exposed to a cold pressor task,[17,39] and junior high school students.[30] Although asymptomatic undergraduate women scored higher than men on the total score of the PCS, gender differences were only observed on the rumination and helplessness subscales, not the magnification subscale.[3] Similar findings were reported by Osman et al.[16] and by Sullivan et al.[39]

The relation between gender and catastrophizing is of particular interest in the context of the growing literature showing that women are more likely than men to report high levels of pain (for a review, see [53]). Numerous investigations have shown that in comparison with men, women report more intense pain,[54,55] show higher rates of healthcare utilization,[56] and display more pain behavior.[37,39] Until recently, the factors responsible for gender differences in pain were largely unknown, although experimental artifacts, differences in physiology, and socialization have been discussed.[53,57,58]

Three recent studies provide evidence that catastrophizing might account for gender differences in pain. Women reported more intense pain and displayed more pain behavior in a sample of asymptomatic undergraduates participating in a cold pressor procedure[17,39] and a sample of patients with osteoarthritis of the knee.[37] In all three studies, after controlling for catastrophizing scores, gender differences in pain and pain behavior were no longer significant.[17,37,39]

In contrast to these three studies, it is important to note that the majority of studies examining the relation between catastrophizing and pain have not examined gender differences in catastrophizing. The robustness of gender differences in catastrophizing is difficult to ascertain, given that in most studies, gender differences have not been analyzed. No study has reported higher levels of catastrophizing in men.

Findings showing that catastrophizing might mediate gender differences in pain and pain behavior have potentially far-reaching implications. Increased research on the mechanisms by which catastrophizing impacts pain may shed light on the factors that underlie gender differences in pain and pain behavior. In addition, research on the factors that contribute to the evolution of catastrophizing may hold promise for the development of more effective interventions aimed at reducing pain and emotional distress experienced in response to injury, illness, or aversive medical procedures.

Stability and situational specificity of catastrophizing

An emerging literature suggests that catastrophizing is a relatively enduring mode of responding to painful experiences. Test-retest correlations of 0.70–0.80 have been reported in samples of undergraduates over a 6- to 8-week period[3] (studies 2 and 4) and in patients with rheumatoid arthritis during a 6-month period.[2] Although test–retest findings suggest a high degree of stability, catastrophizing may change as a function of age. Lower levels of catastrophizing have been associated with older age in patients attending a university dental clinic[23] and in women after breast cancer surgery.[20] However, the

opposite relation was found in a study of junior high school students, with more students classified as catastrophizers in Grade 9 in comparison with earlier grades.[31] Although the basis for these discrepant findings is unclear, age differences in young adolescents might not be comparable to age differences in adults.

Because catastrophizing is assessed through self-report, it has not been possible to study its development in young (i.e., preliterate) children. However, observational studies suggest that differences in the socialization of male and female children in regard to pain are apparent early in life. For example, observational studies of children indicate that girls were much more likely than boys to react to pain by crying, screaming, or showing anger.[59] In this study, although male and female children did not differ in the frequency or severity of pain-causing incidents, adult caregivers provided more physical comfort to female children who were expressing distress. Among women attending a Planned Parenthood clinic, retrospective reports of reinforcement and modeling of illness behavior in response to pain and other physical symptoms during adolescence predicted illness behavior during adulthood.[60] It is possible that the provision of excessive support and physical comfort in response to pain-eliciting situations may shape the development of alarmist reactions to pain experience and become entrenched over time as a catastrophic orientation to pain.

Contrary to the trait conceptualization, early views regarded catastrophizing as a readily modifiable, situation-specific cognitive style. For example, following instructions not to engage in catastrophic thinking, undergraduates who had previously been identified as catastrophizers were no longer classified as catastrophizers.[61] After a brief stress inoculation procedure, the majority of individuals who had initially been classified as catastrophizers no longer reported catastrophic thoughts during a cold pressor procedure.[62] Although these studies showed decreases in catastrophic thinking with minimal intervention, it is important to consider the strong demand characteristics associated with asking participants to report on mental activity in which they were specifically instructed not to engage.

More intensive cognitive-behavioral interventions can lead to reductions in catastrophizing, which are in turn associated with better adjustment to chronic pain (e.g., [49,50,63]). Therefore, catastrophizing does not appear to have the immutable character ascribed to personality traits, but, at least in the absence of intervention, it appears to remain stable in chronic pain and in asymptomatic populations.

Questions regarding the degree to which catastrophizing is a general phenomenon or one that is restricted to pain-related outcomes have yet to be examined. Nevertheless, there are grounds for proposing a general as opposed to a pain-specific view of catastrophizing. For example, catastrophizing has been discussed as a cognitive component of depression and anxiety.[64,65] The significant relations between catastrophizing, depression, and anxiety (discussed below) are consistent with the view that individuals who catastrophize in pain-related situations might also catastrophize in problem situations that do not involve pain.

Confounded measurement and construct redundancy

In experimental and clinical samples, catastrophizing has been shown to be significantly correlated with depression, state and trait anxiety, fear of pain, and coping-effectiveness.[3,14,19] At times, the magnitude of correlations among these measures has been sufficiently high to question their operational and conceptual distinctiveness.[3,18] In factor analyses of the CSQ, the catastrophizing and coping-effectiveness scales have frequently loaded on the same factor, which suggests that they might be measuring the same underlying construct. In addition, the item content of scales measuring catastrophizing, depression, and anxiety are markedly similar, although they are intended to measure distinct constructs. Therefore, it is not surprising that issues related to redundant or confounded measurement have been raised in research on catastrophizing and pain.[3,18,67–69]

Sullivan and D'Eon[18] first addressed issues of conceptual and measurement confounds in research on the relation between catastrophizing and depression in patients with chronic pain. They asked clinical psychologists to rate the degree to which items of the CSQ "reflected" symptoms of depression. All CSQ catastrophizing items were rated as reflecting symptoms of depression. When these items were removed, the remaining CSQ subscales were not significantly correlated with depression. On the basis of this finding, the authors argued that if catastrophizing was not conceptually or operationally distinct from depression, it could not be invoked as an explanatory construct for high levels of depression in patients with chronic pain.

Sullivan and D'Eon's[18] position was critiqued by Haaga,[68] who argued that the instructional set provided to the raters was sufficiently ambiguous to render the observed findings uninterpretable. Haaga[68] also highlighted that observed relations between catastrophizing and depression were typically in the moderate range and not sufficiently high to be considered supportive of construct redundancy. A similar argument was advanced by Jensen et al.[67]

Keefe et al.[2] directly addressed the issue of construct redundancy between catastrophizing and depression. The authors obtained measures of catastrophizing and depression in a sample of patients with rheumatoid arthritis at two time-points separated by 6 months. They examined whether catastrophizing at time 1 predicted depression measured at time 2 (controlling for time 1 depression). The authors reasoned that if catastrophizing and depression were redundant constructs, controlling for time 1 depression should render the relation between time 1 catastrophizing and time 2 depression nonsignificant. Their findings showed that catastrophizing significantly predicted later depression beyond the variance accounted for by initial depression (see also [63]).

Several investigations have addressed the question of construct redundancy in catastrophizing and depression by examining the concurrent relation between catastrophizing and pain-related outcomes (e.g., pain intensity, disability) while controlling for current levels of depression. Geisser et al.[70] obtained measures of catastrophizing, depression, and pain in a sample of patients with chronic pain. The results of path-analytic procedures revealed that catastrophizing was not redundant with depression; rather, analyses showed that catastrophizing mediated the relation between depression and the evaluative and affective aspects of pain. Walsh et al.[71] reported that catastrophizing was a significant predictor of cold pressor pain, even when controlling for depression. Sullivan et al.[19] reported that catastrophizing was significantly related to perceived disability in patients with soft tissue injuries beyond the variance accounted for by depression. Keefe et al.[37] reported that catastrophizing mediated the relations between gender and pain, pain behavior, and disability, even when controlling for depression.

Discrepant findings have been reported by Affleck et al.[72] These investigators collected daily mood and pain ratings in a sample of patients with rheumatoid arthritis during a period of 72 days. Consistent with previous research, the results of a path analysis revealed that catastrophizing was associated with pain intensity ratings. However, the path linking catastrophizing to pain was no longer significant when levels of depression were controlled.

Research has shown that the relation between catastrophizing and pain is independent of other distress-related variables, such as anxiety and fear of pain. For example, Sullivan et al.[3] administered measures of catastrophizing, fear of pain, and trait anxiety to a sample of asymptomatic undergraduates participating in a cold pressor procedure. Catastrophizing correlated significantly with fear of pain and trait anxiety. However, the results of a regression analysis revealed that only catastrophizing contributed significant unique variance to the prediction of pain.

Clinical studies have also shown catastrophizing to be distinct from neuroticism. Affleck et al.[72] reported that the relation between neuroticism and pain in patients with rheumatoid arthritis was mediated by catastrophizing (but not when depression was controlled for). Martin et al.[48] reported that catastrophizing predicted perceived physical disability in patients with fibromyalgia, even when controlling for level of neuroticism. Sullivan et al.[19] reported that catastrophizing predicted perceived disability in patients with soft-tissue injuries, even when controlling for levels of trait anxiety.

Therefore, despite initial claims of redundancy, research suggests that catastrophizing is distinct from depression. Prospective studies have shown that catastrophizing predicts future depression, even when accounting for initial levels of depression. Cross-sectional studies have shown that catastrophizing predicts pain-related outcomes, even when controlling for depression. Although catastrophizing is correlated with various indices of emotional distress, it appears to contribute unique variance to the prediction of pain and disability.

Summary of evidence

To date, nearly 100 studies have been published addressing the relation between catastrophizing and pain. The results have shown a remarkable level of consistency. Catastrophizing has been associated with heightened pain in clinical and in experimental studies with adults and with children. It has also been shown to be associated with heightened disability and to predict disability better than disease-related variables or pain. In addition, catastrophizing has been associated with increased pain behavior, increased use of health care services, longer durations of hospital stay, and increased use of analgesic medication. In the absence of intervention, catastrophizing seems to be relatively stable over time, although there are indications that it may decrease with age (at least in adult samples). Several investigations have reported that women catastrophize more than men.

Theoretical models and possible mechanisms of action

As noted previously, although considerable evidence has accumulated on the relation between catastrophizing and pain, there have been few attempts to place the emerging pattern of findings within a broader theoretical context. There have been discussions of whether catastrophizing may best be viewed as a coping strategy, a belief, or an appraisal process, but these discussions have not moved beyond operational or definitional

issues.[69,73–75] What is needed at this point is a general framework that will assist in the interpretation of available findings and, more importantly, provide direction for future research.

In this final section of the article, we discuss the explanatory power of a schema-activation theory, an appraisal model, an attentional model, and a communal/interpersonal coping model in accounting for the influence of catastrophizing on pain-related outcomes. Because these models in their original forms were not intended to account for psychological influences on pain experience, they are discussed in a rather "generic" manner to explore the predictions they might yield if applied to catastrophizing and pain. Among the models, there is some conceptual overlap, and they are not to be considered as mutually exclusive. The intent of our discussion is not to support or reject a particular theoretical approach, but to point to directions for research that might ultimately contribute to the development of a comprehensive theory about psychological influences on pain and pain-related outcomes.

Schema-activation model

Before its emergence in the pain literature, catastrophizing had been discussed primarily within the context of cognitive theories of depression. For example, in the cognitive model of emotional disorders of Beck,[76] catastrophizing is viewed as a "cognitive distortion" that might contribute to the precipitation and maintenance of depressive symptoms. Beck et al.[77] propose that "depressive schema" may become activated after the occurrence of negative life events. Once activated, depressive schemas are said to give rise to a variety of cognitive distortions, including catastrophizing, overgeneralization, personalization, and selective abstraction. In the model of Beck et al.,[77] cognitive errors are expected to bias information processing in such a manner as to increase the likelihood of the development of depressive symptoms.

The high rate of comorbidity of depression and pain is consistent with the view that the two conditions may share a common cognitive vulnerability factor. Depression, however, does not appear to be a precondition to the association between catastrophizing and pain. As noted earlier, several investigations[2,3,19,48] have shown that catastrophizing contributes to pain independent of its relation to depression.

Nevertheless, it is possible that catastrophizing may contribute to heightened pain experience through its influence on emotional functioning.[70,71] Multidimensional models of pain perception distinguish between affective and sensory aspects of pain experience.[7,9] It has been shown that catastrophizing is associated with high levels of situational anxiety, anger, and sadness.[3] These transient subclinical states of emotional distress could be the vehicle through which catastrophizing impacts on pain experience. There are data to suggest that catastrophizing may contribute to the affective and evaluative components of pain experience in patients with chronic pain.[70]

It has also been suggested that catastrophizers may possess "pain schema" containing excessively negative information about pain-related experiences, and pessimistic beliefs about pain or the ability to cope with pain.[3,10] As a function of a learning history characterized by heightened pain experience, catastrophizers may develop expectancies about the high threat value of painful stimuli and about their inability to effectively manage the stress associated with painful experiences.[10,75] Once activated, these pain schema may influence emotional or cognitive functioning in a manner that leads to heightened pain experience.

Schema-activation models are ambiguous with respect to the conditions necessary for schema activation, and methodologies are not readily available for discerning whether, or to what degree, a schema has been activated. Prospective studies showing that catastrophizing, measured in a pain-free state, predicts future pain responses suggest that catastrophizing does not require the experience of pain for schema activation.[3,23] However, it must be recognized that pain may not be the only negative life event that can activate a pain schema and that schema activation may not be an all-or-none phenomenon.

Schema-activation models are heuristic in pointing to a number of cognitive processes variables that might mediate the relation between catastrophizing and pain. Cognitive theories of emotional functioning propose that processes related to stimulus interpretation or appraisal, and selective attention to schema-relevant information may be initiated after schema activation. The possibility that processes such as these may underlie the relation between catastrophizing and pain is discussed later in this article.

Appraisal model

A model related to the schema-activation theory is the characterization of catastrophizing as an appraisal.[67,75] The transactional model of stress in Lazarus and Folkman[78] provides a conceptual distinction among the concepts of appraisals, beliefs, and coping. Primary appraisals (judgments about whether a potential stressor is irrelevant, benign–positive, or stressful) interact with secondary appraisals (beliefs about coping options and their possible effectiveness) and influence whether, and which, coping responses will be attempted.[74,75,79]

At a descriptive level, the different components of catastrophizing (magnification, rumination, and helplessness) share features with primary and secondary

appraisal processes.[3] For example, magnification and rumination may be related to primary appraisal processes in which individuals may focus on and exaggerate the threat value of a painful stimulus. Helplessness may be related to secondary appraisal processes in which individuals negatively evaluate their ability to deal effectively with painful stimuli.

The results of several investigations have shown that catastrophizing is closely associated with other appraisal constructs. For example, the CSQ catastrophizing subscale frequently loads on the same factor as coping–efficacy ratings, which suggests that catastrophizing may be related to efficacy appraisals in relation to controlling or decreasing pain.[14,49,50,69] A negative association between catastrophizing and appraisals of control has also been reported by Crisson and Keefe.[80] There is also evidence that catastrophizing may function as an appraisal process linking pain beliefs to pain outcomes. For example, Stroud et al.[81] reported that negative thoughts (including catastrophic thoughts) in patients with chronic pain mediated the relation between their pain beliefs and certain measures of adjustment, including affective distress and perceived interference from pain.

Attentional model

A schema-activation model and an appraisal model both predict that individuals who exaggerate the threat value of pain stimuli or pain sensations will likely increase their attentional focus on pain. The pain schema of catastrophizers may lead them to preferentially process pain-related information and to interpret even ambiguous sensations as being painful.[27,82] Appraisals of threat should direct attention toward the source of the threatening information.[83–85]

Consistent with the view that catastrophizers focus more attention on pain, studies have shown that catastrophizers experience more difficulty controlling or suppressing pain-related thoughts, they ruminate more about their pain sensations, and their cognitive task performance is disrupted by anticipation of pain stimulus onset.[27,85–89] Depth-of-processing or vigilance paradigms that have been used in social–cognitive research may be useful in addressing whether the pain schema and/or appraisals lead catastrophizers to expect negative outcomes, to selectively encode, or preferentially process pain-related information.[90–92]

Additional evidence for the role of attentional factors is derived from findings showing that the rumination subscale of the PCS is most strongly related to pain intensity ratings. Sullivan and Neish[23] found that, of the three PCS subscales, only rumination contributed significant unique variance to the prediction of pain ratings during dental hygiene treatment. That is, patients who endorsed statements such as "I keep thinking about how much it hurts" and "I can't seem to keep it out of my mind" were particularly likely to experience increased levels of pain.

Therefore, attentional focus on pain may be a critical psychological substrate of the relation between catastrophizing and pain experience. Methodologies such as the modified Stroop task,[93] dot-probe paradigms (e.g., [83]), and primary task paradigms (e.g., [85]) might be useful in further exploring pain-related attentional focus in individuals who catastrophize.[94,95]

Coping model

Coping generally refers to the strategies that individuals use to minimize the impact of life stressors on their psychological well-being.[78,96] There has been some debate regarding the conceptualization of catastrophizing as a coping strategy.[67,73,75,97] Specifically, it has been argued that catastrophizing is not strategic or goal-directed and should be considered to be distinct from coping efforts.[74,75] In support of this view, numerous investigations have shown that catastrophizing is rarely correlated with other forms of coping. When CSQ subscales have been factor-analyzed, coping scales have frequently emerged as a separate, independent factor from catastrophizing.[14,49,50] In a large sample factor analysis of CSQ subscales, Lawson et al.[98] found that none of the coping scales loaded on the same factor as catastrophizing.

Furthermore, catastrophizers do not appear to differ from noncatastrophizers in the coping strategies they employ. Spanos et al.[15] reported that catastrophizers and noncatastrophizers did not differ in the number of coping strategies they reported using during a cold pressor procedure. However, for noncatastrophizers, there was an association between number of coping strategies and degree of pain reduction. For catastrophizers, number of coping strategies reported was not associated with pain reduction. Similar findings were reported by Sullivan et al.[3]

To dismiss the coping functions of catastrophizing on the basis of its apparent lack of focus on pain reduction or its independence from other forms of coping may be premature. Recent critiques of experimental coping research have highlighted that distress reduction may not always be the goal of coping efforts.[99–102] In a similar vein, it is important to consider that, for pain patients, pain reduction may not always be the primary goal of coping. When instrumental, caregiving or relational goals are primary, coping efforts may actually be associated with increases rather than decreases in physical or emotional distress.[103] The patient with chronic pain who

takes advantage of a good day to catch up on yard work (e.g., an instrumental goal) will likely experience increased pain. Similarly, the patient with chronic pain who persists in performing heavy household chores to avoid burdening her frail husband (e.g., caregiving goal) will also experience increased pain. In either case, if pain intensity or emotional distress were evaluated as outcome variables, analyses would suggest that the coping efforts of the individuals were maladaptive. In research on the relation between coping and pain, we may have proceeded from the erroneous view that pain reduction or emotional distress reduction are primary coping goals of patients with pain.

Sullivan et al.[39] suggested catastrophizing may represent a broader dimension of a communal or interpersonal approach to coping.[99,103–106] Within this framework, it is assumed that individuals differ in the degree to which they adopt social or relational goals in their efforts to cope with stress. Sullivan et al.[39] suggested that catastrophizers may engage in exaggerated pain expression 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. The increased attention to their pain and the exaggerated display of pain behavior shown by catastrophizers may become maladaptive by contributing to heightened pain experience.[73,107] In addition, solicitous or reinforcing responses from others may serve to trigger, to maintain, or to reinforce the exaggerated pain expression of catastrophizers.

A communal coping model of catastrophizing predicts that women will catastrophize more than men. This prediction follows from research and theory suggesting that women are more likely than men to be emotionally expressive and relationally oriented in their efforts to cope with life stresses including pain.[53,99,103,104,108–110] In light of research showing that catastrophizing mediates gender differences in pain, it is likely that the factors that lead to the development of catastrophizing may be similar to those that give rise to gender differences in pain experience and expression.

A central tenet of a communal coping model of catastrophizing is that catastrophizing serves a social communicative function aimed toward maximizing the probability that distress will be managed within a social/interpersonal context rather than an individualistic context. It has been suggested that the expression of distress may be a necessary component of a communal approach to coping.[39,99] If the goals of coping include seeking proximity, support, or assistance, individuals must be effective in accurately communicating their distress to others in their social environment.

The communicative functions of catastrophizing are supported by the aforementioned research detailing a consistent relation between catastrophizing and both self-reported and observed pain behavior. More compelling support comes from findings regarding spousal perceptions of coping effectiveness.[111] These authors found that the more patients catastrophized, the less their spouses perceived the patient as being able to cope effectively with pain. In addition to highlighting the communicative functions of catastrophizing, these findings suggest that spousal observations of the catastrophizing of a partner might lead to lower expectancies for their participation in home, social, or vocational activities.

Bédard et al.[31] found that adolescents who catastrophized also reported more support-seeking in relation to their pain symptoms than did adolescents who did not catastrophize. In the same study,[31] it was noted that although catastrophizers used more over-the-counter medication to manage their pain symptoms in comparison with noncatastrophizers, they waited until their pain was at a higher intensity before self-administering medication. These findings suggest that catastrophizers may be engaging in displays of distress and pain behavior for longer periods than noncatastrophizers, even when means of reducing pain are available.

Several predictions arise from a communal coping model of catastrophizing. First, if communication goals are primary in catastrophizing, social presence should act as a discriminative cue for the display of pain behavior in individuals who catastrophize. The discriminative cue value of social presence should be greatest for individuals who are part of the social network of the catastrophizer (e.g., spouse, family members) in that these individuals have been the targets of previous pain communication from the catastrophizer and have likely played a role in maintaining the exaggerated orientation to pain expression of the catastrophizer. These predictions have not been examined empirically.

Links to physiology and neuroanatomy

The Gate Control Theory of Pain[7,112] was the first to propose that the brain plays a dynamic role in pain perception rather than simply being the passive recipient of nociceptive signals. The theory proposes that specific brain activity may open or close spinal-gating mechanisms, thereby increasing or decreasing pain, respectively. Psychological factors were postulated to impact on pain experience via their influence on spinal-gating mechanisms. With the advent of technology allowing for the measurement of brain activity during aversive stimulation, it has been possible to gain greater insight into the inter-relations between psychological and physical mechanisms involved in the experience of pain. Research

is emerging indicating that pain-related psychological variables may have specific neurophysiologic and neuroanatomic substrates.

A study by Bandura et al.[113] provides evidence of a link between psychological variables and endogenous opiates. In this study, cold pressor participants were taught a variety of cognitive methods of pain control and were asked to rate their confidence in their ability to tolerate pain (i.e., self-efficacy). The use of cognitive strategies enhanced perceived self-efficacy, which increased pain tolerance. However, the administration of naloxone interfered with the self-efficacy enhancing effects of cognitive strategy use. The observed relations between catastrophizing and coping efficacy suggest that catastrophizing may also be linked in some manner to the action of endogenous opiates.

Recent neuroanatomic investigations have also elucidated possible neural substrates of pain-related psychological variables. Ploghaus et al.[114] used functional magnetic resonance imaging of the brain to show that distinct areas of the brain are involved in pain processing versus pain anticipation. Participants were exposed to different colored lights signaling painful and nonpainful thermal stimulation, and functional magnetic resonance imaging revealed that there was a dissociation of neural regions in response to pain and its anticipation. Perhaps even more importantly, the researchers found that although the level of brain activation in the regions associated with the pain stimulus remained stable across trials, the level of activation in the pain anticipation regions increased over trials. Thus, this study provides evidence of experience-based changes in neural signal patterns. Catastrophizing may be one such behavioral/cognitive marker of pain anticipation.

Similarly, it has been suggested that specific neural changes during the experience of pain may amplify (or reduce) the peripheral signal in response to subsequent pain.[112,115,116–118] In a study comparing patients who underwent lower abdominal surgery and epidural local anesthetic before or after incision (groups underwent standard general surgical anesthesia), patients who underwent the local anesthetic before incision showed lower pain scores and used 22% less patient-controlled morphine after surgery. On the basis of these findings, it was suggested that this "pre-emptive analgesia" might prevent the neural hyperexcitability, which contributes to later postoperative pain.[119]

Melzack[120–122] has recently proposed a "neural matrix" model of pain, which greatly expands the dynamic role of networks within the brain to explain the experience of pain. The neural matrix model suggests that although the processing of pain by the brain is genetically specified, such processing is modified by experience. Therefore, factors that increase sensory flow of pain signals, may alter central thresholds of excitability over time, thereby increasing sensitivity to pain.[120, 122] It is reasonable to propose that by engaging in cognitive activity that amplifies pain signals, central neural mechanisms in catastrophizers may become more sensitized and yielding a chronic hyperalgesic state. Social interactions reinforcing pain and physical symptoms during childhood may have long-term physiological consequences.[60] These early learning experiences, which may be characterized by excessive aversive stimulation such as multiple injuries, illness or abuse, may also alter neural architecture to yield a chronic hyperalgesic state.

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 physiologic 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.

Synthesis and future directions

The review of theoretical models suggests that schema activation, appraisal, attentional, and communal coping models may provide useful frameworks for understanding the relation between catastrophizing and pain. Although these models have been discussed as alternative conceptualizations of the relation between catastrophizing and pain, they are not necessarily incompatible. By broadening the scope of explanation, and proceeding from the perspective of hierarchical levels of analysis, it is conceivable that these models may account for different domains of the relation between catastrophizing and pain.

The schema activation and appraisal models are essentially "proximal" explanations of catastrophizing and pain relations. They address the cognitive variables that precede pain experience and the variables that may lead to the development of enduring beliefs about pain experience. The schema activation and appraisal models help point to basic process mechanisms that may underlie the catastrophizing-pain relation**,** particularly attentional processes, and suggest possible bridges to physiologic mechanisms.

These models may also help to explain the development and maintenance of catastrophizing. Although it is not readily apparent why individuals would adopt or persist in a cognitive style that leads them to experience

heightened pain and emotional distress, fear-avoidance models of pain might be invoked as a potential explanation. Catastrophizing may contribute to pain-related fear, which then leads to avoidance of activity and subsequent disability.[66] In a recent study by Turner et al.,[97] it was found that pain beliefs mediated the relation between catastrophizing and disability, which suggests that catastrophizing may influence disability indirectly through other pain appraisals. Pain-related fear might be one such appraisal.

The communal coping model can be construed as a more "distal" explanation of the relation between catastrophizing and pain. Its focus is primarily on the social-behavioral dimensions of catastrophizing. It is well within current trends that emphasize the need to address interpersonal issues in coping with stress and illness and avoids many of the criticisms that have been levied against overly "individualistic" conceptualizations of adaptational processes.[99,103,104] The communal coping approach can accommodate a "strategic" view of catastrophizing in which the pursuit of interpersonal coping goals inadvertently becomes maladaptive by increasing the aversiveness of pain experience.

The most compelling, and perhaps most heuristic model may be one that incorporates features of communal coping and schema/appraisal processes in which social factors and social goals determine the development and maintenance of catastrophizing, whereas schema/appraisal processes account for the cognitive factors that link catastrophizing to pain experience. The schema/appraisal component of the model predicts a number of attentional and information processing biases that may arise from catastrophizing. The communal component of the model suggests a number of research venues, such as evaluating the discriminative cue value of social presence, solicitous or reinforcing behavior of the spouses of catastrophiziers, and the "communication effectiveness" of the pain displays of catastrophizers. The schema/appraisal component suggests the need to address more closely the basic process mechanisms contributing to pain experience, whereas the communal component calls for more attention to the "ecology" of everyday painful experiences and the social contextual factors within which catastrophizing emerges.

In conclusion, research has shown catastrophizing to be a powerful marker for heightened pain experience. The relative stability of catastrophizing, its amenability to measurement, its presence in clinical and in nonclinical populations, and the magnitude of its relation to pain and pain-related outcomes make catastrophizing ideally suited for basic and process-related research on the psychology of pain. It has been heuristic in generating numerous clinical and experimental investigations and has been central in discussions and controversy on the interrelations among different psychological determinants of pain. Future research on the social, cognitive, emotional, and physiologic correlates of catastrophizing holds promise of contributing in a substantive manner to the development and/or elaboration of comprehensive theories addressing the interplay between psychological and physiologic processes that underlie pain experience.

Acknowledgments: The authors thank Dr. Sophie Bergeron, Universite de Montreal, Quebec, Dr. Joyce D'Eon, The Rehabilitation Centre, Ottawa, Dr. Patrick McGrath, Dalhousie University, Halifax, and two anonymous reviewers for helpful comments on a previous version of this article. Portions of this article were presented in 1999 at the Annual Meeting of the American Pain Society in Fort Lauderdale, Florida.

REFERENCES

1. Maupassant, G. *Sur L'eau* [originally published 1875]. New York: M. Walter Dunne, 1903.
2. Keefe FJ, Brown GK, Wallston KA, et al. Coping with rheumatoid arthritis: catastrophizing as a maladaptive strategy. *Pain* 1989;37:51–6.
3. Sullivan MJL, Bishop S, Pivik J. The Pain Catastrophizing scale: development and validation. *Psychol Assess* 1995;7:524–32.
4. Keefe FJ, Caldwell DS, Queen KT, et al. Pain coping strategies in osteoarthritis patients. *J Consult Clin Psychol* 1987;55:208-12.
5. Descartes R. *The passions of the soul* [originally published 1649]. Indianapolis: Hackett, 1989.
6. Beecher HK. Relationship of significance of wound to pain experienced. *J Am Med Assoc* 1956;161:1609–13.
7. Melzack R, Wall PD. Pain mechanisms: a new theory. *Science* 1965;150:971–9.
8. Fordyce WE. *Behavioral methods for chronic pain and illness*. St. Louis: Mosby, 1976.
9. Leventhal H, Everhart D. Emotion, pain, and physical illness. In: Izard CE, ed. *Emotion and psychopathology*. New York: Plenum Press;1979:263–99.
10. Turk DC, Rudy TE. Cognitive factors and persistent pain: a glimpse into Pandora's box. *Cog Ther Res* 1992;16:99–122.
11. Keefe FJ, Caldwell DS, Queen KT, et al. Osteoarthritis knee pain: a behavioral analysis. *Pain* 1987b;28:309–21.
12. Chaves JF, Brown JM. *Self-generated strategies for the control of pain and stress.* Paper presented at the Annual Meeting of the American Psychological Association, Toronto, Ontario, August 1978.
13. Chaves JF, Brown JM. Spontaneous cognitive strategies for the control of clinical pain and stress. *J Behav Med* 1987;10:263–76.
14. Rosenstiel AK,.Keefe FJ. The use of coping strategies in chronic low back pain patients: relationship to patient characteristics and current adjustment. *Pain* 1983;17:33–44.
15. Spanos NP, Radtke-Bodorik HL, Ferguson JD, et al. The effects of hypnotic susceptibility, suggestions for analgesia, and utilization of cognitive strategies on the reduction of pain. *J Abnorm Psychol* 1979;88:282–92.
16. Osman A, Barrios FX, Kopper BA, et al. Factor structure, reliability, and validity of the Pain Catastrophizing Scale. *J Behav Med* 1997;20:589–605.
17. Sullivan MJL, Tripp D, Rodgers W, et al. Catastrophizing and pain perception in sports participants. *J Applied Sport Psychol* 2000;12:151–67.
18. Sullivan MJL, D'Eon J. Relation between catastrophizing and depression in chronic pain patients. *J Abnorm Psychol* 1990;99:260–3.
19. Sullivan MJL, Stanish W, Waite H, et al. Catastrophizing, pain, and disability following soft tissue injuries. *Pain* 1998;77: 253–60.

20. Jacobsen PB, Butler RW. Relation of cognitive coping and catastrophizing to acute pain and analgesic use following breast cancer surgery. *J Behav Med* 1996;19:17–29.
21. Flor H, Behle DJ, Birbaumer N. Assessment of pain-related cognitions in pain patients. *Behav Res Ther* 1993;31:63–73.
22. Butler RW, Damarin FL, Beaulieu C, et al. Assessing cognitive coping strategies for acute post-surgical pain. *Psychol Assess* 1989;1:41–5.
23. Sullivan MJL, Neish N. Catastrophizing, anxiety and pain during dental hygiene treatment. *Comm Dent Oral Epidemiol* 1998;37: 243–50.
24. Haythornthwaite JA, Lawrence JW, Fauerbach JA. Brief cognitive interventions for burn pain. *Ann Behav Med*; In press.
25. Sullivan MJL, Stanish W, Sullivan ME, et al. Differential predictors of pain and disability in patients with whiplash injuries. *Pain Res Manag*; In press.
26. Lefebvre JC, Lester N, Keefe FJ. Pain in young adults. II: the use and perceived effectiveness of pain-coping strategies. *Clin J Pain* 1995;11:36–44.
27. Sullivan MJL, Rouse D, Bishop S, et al. Thought suppression, catastrophizing, and pain. *Cog Ther Res* 1997;21:555–68.
28. Beckham JC, Keefe FJ, Caldwell DS, et al. Pain coping strategies in rheumatoid arthritis: relationships to pain, disability, depression and daily hassles. *Behav Ther* 1991;22:113–24.
29. Spinhoven P, Ter Kuile MM, Linssen ACG, Gazerdam B. Pain coping strategies in a Dutch population of chronic low back pain patients. *Pain* 1989;37:77–83.
30. Gil KM, Abrams, MR, Phillips G, et al. Sickle cell disease pain: relation of coping strategies to adjustment. *J Consult Clin Psychol* 1989;57:725–31.
31. Bédard GB, Reid GJ, McGrath PJ, et al. Coping and self-medication in a community sample of junior high school students. *Pain Res Manage* 1997;2:151–6.
32. Reid GJ, Gilbert CA, McGrath PJ. The Pain Coping Questionnaire: preliminary validation. *Pain* 1998;76:83–96.
33. Schanberg LE, Lefebvre JC, Keefe FJ, et al. Pain coping and the pain experience in children with juvenile chronic arthritis. *Pain* 1997;73;181–9.
34. Schanberg LE, Keefe FC, Lefebvre JC, et al. Pain coping strategies in children with juvenile primary fibromyalgia syndrome: correlation with pain, physical function, and psychological distress. *Arthr Care Res* 1996;9:89–96.
35. Thastum M, Zacharias R, Scholer M, et al. Cold pressor pain: comparing responses of juvenile arthritis patients and their parents. *Scand J Rheumatol* 1997;26:272–9.
36. Sullivan MJL, Neish N. The effects of disclosure on pain during dental hygiene treatment: the moderating role of catastrophizing. *Pain* 1999;79:155–63.
37. Keefe FJ, Lefebvre JC, Egert JR, et al. The relationship of gender to pain, pain behavior, and disability in osteoarthritis patients: the role of catastrophizing. *Pain* 2000;87:325–34.
38. Keefe FJ, Caldwell DS, Martinez S, et al. Analyzing pain in rheumatoid arthritis patients: pain coping strategies in patients who have had knee replacement surgery. *Pain* 1991;46:153–60.
39. Sullivan MJL, Tripp DA, Santor D. Gender differences in pain and pain behavior: the role of catastrophizing. *Cog Ther Res* 2000;24:121–34.
40. Nicassio PM, Schoenfeld-Smith K, Radojevic V, et al. Pain coping mechanisms in fibromyalgia: relationship to pain and functional outcomes. *J Rheumatol* 1995;22:1552–8.
41. Gil KM, Abrams MR, Phillips G, et al. Sickle cell disease pain 2. Predicting health care use and activity level at 9-month follow-up. *J Consult Clin Psychol* 1992;60:267–73.
42. Gil KM, Thompson RJ, Keith BR, et al. Sickle cell disease pain in children and adolescents: change in pain frequency and coping strategies over time. *J Ped Psychol* 1993;18:621–637.
43. World Health Organization (WHO). *Expert committee on disability prevention and rehabilitation.* WHO Technical Reports Series. 1981;68B:6–37
44. Sullivan MD, Loeser JD. The diagnosis of disability: treating and rating disability in a pain clinic. *Arch Intern Med* 1992;152: 1829–35.
45. Cats-Baril WL, Frymoyer JW. Identifying patients at risk of becoming disabled due to low back pain: the Vermont Rehabilitation Engineering Center model. *Spine* 1991;16:605.
46. Fordyce WE. *Back pain in the work place: management of disability in nonspecific conditions*. Seattle: IASP Press, 1995.
47. Robinson ME, Myers C, Sadler IJ, et al. Bias effects in three common self-report assessment measures. *Clin J Pain* 1997;13: 74–81.
48. Martin MY, Bradley LA, Alexander RW, et al. Coping strategies predict disability in patients with primary fibromyalgia. *Pain* 1996;68:45–53.
49. Turner JA, Clancy S. Strategies for coping with chronic low back pain: relationships to pain and disability. *Pain* 1986;24:355–64.
50. Parker JC, Smarr KL, Buesher KL, et al. Pain control and rational thinking: implications for rheumatoid arthritis. *Arthr Rheumatol* 1989;32:984–90.
51. Vienneau TL, Clark AJ, Lynch ME, et al. Catastrophizing, functional disability and pain reports in adults with chronic low back pain. *Pain Res Manage* 1999;4:93–96.
52. Jensen I, Nygren A, Gamberale F, et al. Coping with long-term musculoskeletal pain and its consequences: is gender a factor? *Pain* 1994;57:167–72.
53. Unruh AM. Gender variations in clinical pain experience. *Pain* 1996;65:123–67.
54. Verbrugge LM, Lepkowski JM, Konkol LL Levels of disability among U.S. adults with arthritis. *J Gerentol Soc Sci* 1991;46: 57–83.
55. Hasvold T, Johnsen R. Headache and neck and shoulder pain: frequent and disabling complaints in the general population. *Scan J Prim Health Care* 1993;11:219–24.
56. Taylor H, Curran NM. *The Nuprin pain report*. New York: Louis Harris & Associates, 1985.
57. Levine FM, De Simone LL. The effects of experimenter gender on pain report in male and female subjects. *Pain* 1991;44:69–72.
58. Lautenbacher S, Rollman GB. Gender differences in response to pain and non-painful stimuli are dependent upon stimulation method. *Pain* 1993;53:255–64.
59. Fearon I, McGrath PJ, Achat H. 'Booboos': the study of everyday pain among young children. *Pain* 1996;68;55–62.
60. Whitehead WE, Crowell MD, Heller BR, et al. Modeling and reinforcement of the sick role during childhood predicts adult illness behavior. *Psychosom Med* 1994;56:541–50.
61. Spanos NP, Henderikus JS, Brazil K. The effects of suggestion and distraction on coping ideation and reported pain. *J Ment Imagery* 1981;5:75–90.
62. Vallis TM. A complete component analysis of stress inoculation for pain tolerance. *Cog Ther Res* 1984;8:313–29.
63. Keefe FJ, Caldwell DS, Williams DA, et al. Pain coping skills training in the management of osteoarthritis knee pain: a comparative study. *Behav Ther* 1991;21:49–62
64. Beck AT. *Depression: causes and treatment*. Philadelphia: University of Pennsylvania Press, 1967.
65. Borkovec TD, Inz J. The nature of worry in generalized anxiety disorder: a predominance of thought activity. *Behav Res Ther* 1990;28:153–8.
66. Vlaeyen JWS, Kole-Snijders AMJ, Boeren RBG, et al. Fear of movement/(re) injury in chronic low back pain and its relation to behavioral performance. *Pain* 1995;62:363–72.
67. Jensen MP, Turner JA, Romano, JM, et al. Coping with chronic pain: a critical review of the literature. *Pain* 1991;47:249–83.
68. Haaga DAF. Catastrophizing, confounds, and depression: a comment on Sullivan and D'Eon (1990). *J Abnorm Psychol* 1992; 101:206–7.
69. Geisser ME, Robinson ME, Riley JL. Pain beliefs. Coping, and adjustment to chronic pain. *Pain Forum* 1999;8:161–8.
70. Geisser ME, Robinson ME, Keefe FJ, et al. Catastrophizing, depression and the sensory, affective and evaluative aspects of chronic pain. *Pain* 1995;59:79–83.

71. Walsh TM, Smith CP, McGrath PJ. Pain correlates of depressed mood in young adults. *Pain Res Manage* 1998;3:135–43.
72. Affleck G, Tennen H, Urrowns S, et al. Neuroticism and the pain mood relation in rheumatoid arthritis: insights from a perspective daily study. *J Consult Clin Psychol* 1992;60:119–26.
73. Keefe FJ, Lefebvre JC, Smith SJ. Catastrophizing research: avoiding conceptual errors and maintaining a balanced perspective. *Pain Forum* 1999;8:176–80.
74. Haythornthwaite JA, Heinberg LJ. Coping with pain: what works, under what circumstances, and in what ways? *Pain Forum* 1999; 8: 172–5.
75. Thorn BE, Rich MA, Boothby, JL. Pain beliefs and coping attempts: conceptual model building. *Pain Forum* 1999;8:169–71.
76. Beck AT. *Cognitive therapy and the emotional disorders*. New York: International Universities Press, 1976.
77. Beck AT, Rush AJ, Shaw BF, et al. *Cognitive therapy for depression*. New York: Guilford, 1979.
78. Lazarus RS, Folkman S. *Stress, appraisal, and coping*. New York: Springer Publication Company, 1984.
79. Haythornthwaite JA, Menefee LA, Heinberg LJ, et al. Pain coping strategies predict perceived control over pain. *Pain* 1998;77: 33–9.
80. Crisson JE, Keefe FJ. The relationship of locus of control to path coping strategies and psychological distress in chronic pain patients. *Pain* 1988;35:147–54.
81. Stroud MW, Thorn BE, Jensen MP, et al. The relation between pain beliefs, negative thoughts, and psychosocial functioning in chronic pain patients. *Pain* 2000;84:347–52.
82. Cioffi D, Holloway J. Delayed costs of suppressed pain. *J Pers Soc Psychol* 1993;64:274–82.
83. MacLeod C, Mathews A, Tata C. Attentional bias in emotional disorders. *J Abnorm Psychol* 1986;95:15–20.
84. Mathews A, May J, Mogg K, et al. Attentional bias in anxiety: selective search or defective filtering. *J Abnorm Psychol* 1991; 99:163–73.
85. Crombez G, Eccleston C, Baeyens F, et al. When somatic information threatens, catastrophic thinking enhances attentional interference. *Pain* 1997;74:230–7.
86. Gil KM, Williams DA, Keefe FJ, et al. The relationship of negative thoughts to pain and psychological distress. *Behav Ther* 1990;21:349–62.
87. Heyneman NE, Fremouw WJ, Gano D, et al. Individual differences and the effectiveness of different coping strategies for pain. *Cog Ther Res* 1990;14:63–77.
88. Eccleston C, Crombez G, Aldrich S, et al. Attention and somatic awareness in chronic pain. *Pain* 1997; 72: 209–16.
89. Eccleston C, Crombez G. Pain demands attention: a cognitive-affective model of the interruptive function of pain. *Psychol Bull* 1999;125:356–66.
90. Derry PA, Kuiper NA. Schematic processing and self-reference in clinical depression. *J Abnorm Psychol* 1981;90:286–97.
91. Kirsch I. Response expectancy as a determinant of experience and behavior. *Am Psychol* 1985;40;1189–202.
92. Pratto F, John OP. Automatic vigilance: the attention grabbing power of negative social information. *J Pers Soc Psychol* 1991; 61:380–91.
93. Riemann, BC, McNally, RJ. Cognitive processing of personally relevant information. *Cognition and Emotion* 1995;9:325–40.
94. Leventhal H, Nerenz Dr. A model for stress research with some implications for the control of stress disorders. In: Meichenbaum D, Jaremko ME, eds. *Stress reduction and prevention* New York: Plenum Press, 1983.
95. Wells A, Matthews G. *Attention and emotion: a clinical perspective*. Hove, United Kingdom: Lawrence Erlbaum, 1994.
96. Pearlin, LI, Schooler, C. The structure of coping. *J Health Soc Behav* 1978;19:2–21.
97. Turner JA, Jensen, MP, Romano JM. Do beliefs, coping, and catastrophizing independently predict functioning in patients with chronic pain? *Pain* 2000;85:115–25.
98. Lawson KC, Reesor KA, Keefe FJ, et al. Dimensions of pain-related cognitive coping: cross-validation of the factor structure of the Coping Strategy Questionnaire. *Pain* 1990;43:195–204.
99. Lyons R, Sullivan MJL, Ritvo P, et al. *Relationships in chronic illness and disability.* Thousand Oaks, CA: Sage, 1995.
100. Coyne JC, Gottlieb B. The mismeasure of coping by checklist. *J Pers* 1996;64:959–91.
101. Coyne JC, Racioppo MW. 'Never the twain shall meet': closing the gap between coping research and clinical intervention research. *Am Psychol* 2000;55:655–64.
102. Mickelson KD, Lyons RF, Sullivan MJL, et al. Yours, mine, ours: the relational context of communal coping. In: Sarason BR, Duck S, eds. *Clinical and community psychology: personal relationships*. Chichester, United Kingdom: Wiley, 1999:181–200.
103. Coyne JC, Smith DA. Couples coping with myocardial infarction: a contextual perspective on wifes' distress. *J Pers Soc Psychol* 1991;61:404–12.
104. Hobfoll SE. *Stress, culture, and community: the psychology and philosophy of stress*. New York: Plenum, 1998.
105. Lyons R, Sullivan MJL. Curbing loss in illness and disability: a relationship perspective. In: Harvey JH, ed. *Perspectives on personal and interpersonal loss.* New York: Taylor & Francis, 1998: 579–605.
106. Coyne JC, Fiske V. Couples coping with chronic illness. In: Akamatsu TJ, Crowther JC, Hobfoll SC, et al., eds. *Family health psychology.* Washington, DC: Hemisphere, 1992.
107. Craig KD, Prkachin KM. Social modeling influences on sensory decision theory and psychophysiological indices of pain. *J Pers Soc Psychol* 1978;36:805–13.
108. Rubin Z, Hill CT, Peplau LA, et al. Self-disclosure in dating couples: gender roles and the ethic of openness. *J Marriage Fam* 1980;42:305–17.
109. Wood W, Karten SJ. Gender differences in interaction style as a product of perceived gender differences in competence. *J Person Soc Psychol* 1986;50:341–7.
110. Endler NS, Parker JDA. Assessment of multidimensional coping: task, emotional, and avoidance strategies. *Psych Assess* 1994;6: 50–60.
111. Keefe FJ, Kashikar-Zuck S, Robinson E, et al. Pain coping strategies that predict patient's and spouses rating of patients self-efficacy. *Pain* 1997;73:191–9.
112. Melzack R, Wall PD. *The challenge of pain*. Harmondsworth, United Kingdom: Penguin Books, 1973.
113. Bandura A, O'Leary A, Taylor CB, Gauthier J, et al. Perceived self-efficacy and pain control: opioid and nonopioid mechanisms. *J Pers Soc Psychol* 1987;53:563–71.
114. Ploghaus A, Tracey I, Gati JS, et al. Dissociating pain from its anticipation in the human brain. *Science* 1999;284:1979–81.
115. Woolf CJ, Wall PD. Morphine-sensitive and morphine-insensitive actions of C-fibre input in the rat spinal cord. *Neurosci Lett* 1986;64:221–5.
116. Dickenson AH, Sullivan AF. Subcutaneous formalin-induced activity of dorsal horn neurones in the rat: differential response to an intrathecal opiate administered pre- or post-formalin pain. *Pain* 1987;30:349–60.
117. Coderre TJ, Vaccarino AL, Melzack R. Central nervous system plasticity in the tonic pain response to subcutaneous formalin injection. *Brain Res* 1990;535:155–8.
118. Katz J. George Washington Crile, Anoci-Association, and pre-emptive analgesia. *Pain* 1993;53:243–5.
119. Katz J, Clairoux M, Kavanagh BP, et al. Pre-emptive lumbar epidural anaesthesia reduces post-operative pain and patient-controlled morphine consumption after lower abdominal surgery. *Pain* 1994;59:395–403.
120. Melzack R. Phantom limbs and the concept of a neuromatrix. *Trends Neurosci* 1990;13:88–92.
121. Melzack R. Pain: past, present and future. *Can J Exp Psychol* 1993;47:615–29.
122. Melzack R. From the gate to the neuromatrix. *Pain Supp* 1999; 6:S121–6.