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## Pain and Emotion: New Research Directions



**Francis J. Keefe**

*Duke University Medical Center*



**Mark Lumley**

*Wayne State University*



**Timothy Anderson**

*Ohio University*



**Thomas Lynch**

*Duke University Medical Center*



**Kimi L. Carson**

*Ohio University*

Recently, there has been growing interest in the relation between pain and emotion. Numerous recent studies have been conducted in this area. This article provides an introduction to this interesting area by highlighting selected research topics including studies on: stress and pain, negative emotional states and pain, catastrophizing and pain, the fear of pain, emotional regulation processes and pain, the effects of enhancing emotional regulation on pain, and the relation of emotional distress to treatment seeking in persons having pain. The article concludes with a discussion of important directions for future research in this area. © 2001 John Wiley & Sons, Inc. *J Clin Psychol* 57: 587-607, 2001.

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Correspondence concerning this article should be addressed to: Francis J. Keefe, Ph.D., Pain Prevention and Treatment Research, Box 3159, Duke University Medical Center, Durham, NC 27710; e-mail: keefe003@mc.duke.edu.

In the early decades of psychosomatic medicine, chronic pain problems were often ascribed to various emotional conflicts, or to defenses against emotional experiences (Engel, 1959; Szasz, 1957). These early theoretical models generally lacked empirical support, and they failed to stimulate effective and widely implemented interventions. From the 1960s through the 1980s, psychosomatic models of chronic pain problems were largely displaced by psychosocial models that were based first on behavioral learning principles, and subsequently on cognitive-behavioral theories (Keefe, Dunsmore, & Burnett, 1992). These cognitive and behavioral models of pain have been supported by a wealth of studies demonstrating the important roles played by environmental factors, conditioning and other learning processes, and beliefs and expectations (Affleck, Tenen, Urrows, & Higgins, 1992; Jensen, Turner, Romano, & Karoly, 1991; Keefe & Lefebvre, 2000; Reich & Zautra, 1995; Smith & Wallston, 1992; Zautra & Manne, 1992). Numerous clinical trials have demonstrated the efficacy of treatments based on cognitive and behavioral models to reduce pain and disability, and these treatments are becoming widely used (Compas, Haaga, Keefe, Leitenberg, & Williams, 1998; Keefe, 2000; Morley, Eccleston, & Williams, 1999).

Yet, the past decade has witnessed an explosion of interest in basic and applied aspects of emotion research within the broader field of psychology, and the literature on emotional processes is burgeoning. Recently, this interest in emotion has reinvigorated research and theory on pain, and with the heightened emphasis on the association between emotion and pain has come renewed interest in the role that emotional regulation can play in pain control. Much of the interest in pain and emotion has been fueled by a growing number of new and innovative research studies. The literature on this topic is large, however, and a comprehensive review is beyond the scope of this article. Instead, we seek to provide an introduction to new research on pain and emotion by highlighting selected research topics and studies.

Our review focuses on studies examining the following topics: stress and pain, negative emotional states and pain, catastrophizing and pain, the fear of pain, emotional regulation processes and pain, the effects of enhancing emotional regulation on pain, and the relation of emotional distress to treatment seeking in persons having pain. Finally, we discuss some important directions for future research.

### Stress and Pain

We begin our examination of the recent research on emotion and pain by addressing a topic that has a relatively long history—the role of stress in pain problems. Although stress has been studied over many decades, contemporary views of psychological stress clearly link it to negative emotional states that result from specific appraisals of environmental experiences (Lazarus, 1991).

Clinicians have long observed that stressful or conflictual experiences are common in the lives of many patients with chronic pain, and these observations contributed to early psychosomatic theories (Engel, 1959.) The systematic study of stressful events and chronic pain began in the 1960s when investigators examined whether stressful events were more common in patients having pain than controls, and whether stress preceded pain onset. These studies typically were retrospective, and they often found relationships between reports of increased stressful life events and clinical pain problems such as rheumatoid arthritis (Baker, 1982; Moos & Solomon, 1965a, 1965b), chronic pelvic pain (Rapkin, Kames, Darke, Stampfer, & Naliboff, 1990; Toomey, Hernandez, Gittelman, & Hulka, 1993), and head pain (Passchier & Andrasik, 1993). Critical reviews of this literature have raised a number of concerns about these studies, including the use of inappro-

priate comparison groups, problems determining the temporal order of stressful events and pain onset, and doubts about memory and reporting biases for events occurring years earlier (Roy, 1985).

In more recent years, researchers interested in stress and pain have begun using more rigorous study designs and data analytic methods such as prospective studies, daily diaries, and studies integrating environmental, psychological, and biological measures. For example, Affleck, Urrows, Tennen, and Higgins (1997) used daily diary methodology to assess daily stress and joint pain in patients with rheumatoid arthritis. Using pooled within-person analyses, they found that increases in daily stressors were consistently related to increased joint pain during the subsequent week. Zautra et al. (1998) conducted a prospective study of interpersonal stress, immune markers, disease activity, and pain in women with rheumatoid arthritis. They found that interpersonal stress was associated with immune changes, which were followed by increased inflammation and pain within a week. Interestingly, increased pain resulting from inflammation was followed by lower interpersonal stress the next week, suggesting that patients may have responded to pain by avoiding stressful social interactions, or that the social network responded to the pain by decreasing negative exchanges.

Several studies have demonstrated that the relationship between stress and pain is not straightforward. For example, in a study of rheumatoid arthritis patients having persistent pain conducted by Zautra, Burseson, Matt, Roth, and Burrows (1994), interpersonal stress was found to be related to depression and physiological changes, but stress of a noninterpersonal nature was not. Interestingly, this study also showed that interpersonal stress had a much stronger influence on depression and physiological changes in rheumatoid arthritis patients than in osteoarthritis patients. In another study, Potter and Zautra (1997) found that whereas minor life stressors were followed by increased disease activity, major life stressors were followed by a reduction in disease activity. Finally, in a prospective study of migraine headache sufferers, Holm, Lokken, and Myers (1997) found that prediction of migraines was dependent not just on "stress" but also on participant's appraisals of events as threatening and whether resources were available to cope with the stress. Overall, these findings suggest that the simple examination of "stress" is insufficient, and that we need to understand the emotional processes associated with stressful events, as well as the person's responses to these emotions. We turn now to a more focussed examination of the role of negative emotions in chronic pain.

### Negative Emotional States and Pain

Another group of recent studies has focussed on specific affective states such as depression and anxiety, rather than on measures of stress. Dworkin, Hartstein, Rosner, Walther, Sweeney, and Brand (1992), for example, examined the degree to which depression and anxiety assessed at the time patients were diagnosed with acute herpes zoster was related to the later emergence of postherpetic neuralgia. Univariate correlations suggested there was a relationship between high initial levels of depression and anxiety and the development of postherpetic neuralgia pain. Patients who were more depressed and anxious at the time of diagnosis of acute herpes zoster were much more likely to develop postherpetic neuralgia. A major limitation of this study was the very small sample size ( $n = 19$ .)

A subsequent investigation of rheumatoid arthritis patients by Affleck and colleagues (1992) evaluated how depression assessed prior to diary recording related to subsequent daily diary pain reports over a 75-day period. This study found that patients who were more depressed prior to diary recording were much more likely to have high levels of pain over the 75-day diary period.

Breslau, Chilcoat, and Andreski (1996) conducted a five-year prospective study of a large cohort of young adults in a health maintenance organization. At baseline, they assessed neuroticism, which is the disposition to experience and report aversive emotions, and they assessed the new onset of migraine headaches over the five-year period. These researchers found that the risk of developing migraine increased in direct proportion to baseline neuroticism scores, at least among women. (There were too few men with migraines to determine whether the relationship held for men.) Taken together, the results of these studies suggests that negative emotional states are not just correlated with pain problems, but serve as risk factors to increase the likelihood of pain on set or exacerbation.

A person's emotional state may also influence memory for pain. Kent (1985) studied the relationship of dental anxiety to memory for acute dental pain among 58 people having dental procedures. Prior to their dental procedure, participants rated their dental anxiety, and subsequently rated their dental pain immediately following their dental procedure. Memory for pain was assessed three months later by asking participants to recall the amount of pain they had experienced during the dental procedure. The vast majority of the participants recalled their pain as more severe than it had actually been. Interestingly, those participants having the highest initial levels of anxiety were much more likely to overestimate their pain.

Finally, in contrast to the correlational studies noted above, there have been some experimental studies, which have directly tested the causal effects of negative emotions on pain. Zelman, Howland, Nichols, and Cleeland (1991) evaluated the effects of mood induction on pain tolerance in 65 pain-free participants. The participants were assigned to one of three mood induction conditions: positive, negative, or neutral. The mood induction procedure consisted of participants reading either depressive, elative, or neutral mood statements, and then attempting to experience the mood described by the statements. Results indicated that participants who received the negative mood induction showed a significant decrease in cold-pressor pain tolerance, while those receiving the positive mood induction showed a significant increase in cold-pressor pain tolerance. The results of this study using experimental pain support other laboratory studies showing that inducing negative moods leads to an increase in people's reports of general aches, pains, and other symptoms (Salovey & Birnbaum, 1989).

### Catastrophizing and Pain

Recently, there has been considerable interest in catastrophizing and its relationship to pain (Sullivan et al., in press). Catastrophizing is an emotionally focused response to pain that has been defined as "an individual's tendency to focus on and exaggerate the threat value of painful stimuli and negatively evaluate one's own ability to deal with pain" (Rosensteil & Keefe, 1983). The relationship between catastrophizing and pain was first described in studies conducted in the late 1970s and early 1980s (Chaves & Brown, 1987; Rosensteil & Keefe, 1983; Spanos, Radtke-Bodorik, Ferguson, & Jones, 1979). These studies found that during painful stimulation individuals who tended to catastrophize reported significantly higher levels of pain and emotional distress.

Recent studies have confirmed that catastrophizing is associated with a number of pain-related outcomes including higher self-reported pain (Sullivan, Stanish, Waite, Sullivan, & Tripp, 1998), higher levels of overt pain behaviors (Nicassio, Schoenfeld-Smith, Radojevic, & Schuman, 1995), increased levels of pain-related disability (Sullivan et al., 1998), increased use of pain medications (Jacobsen & Butler, 1996), and greater overall health care utilization (Gil, Thompson, Keith, Tota-Faucette, Noll, & Kinney, 1993). The relationship between catastrophizing and pain outcomes has now been demonstrated across

numerous pain populations including low back pain (Rosensteil & Keefe, 1983; Turner & Clancy, 1986), dental pain (Sullivan & Neish, 1998), fibromyalgia (Martin et al., Nicasio et al., 1995), postsurgical pain (Jacobsen & Butler, 1996), rheumatoid arthritis (Beckham, Keefe, Caldwell, & Roodman, 1991), and sickle-cell disease pain (Gil, Abrams, Phillips, & Keefe, 1989). Catastrophizing has also been shown to related to pain outcomes in different age groups ranging from children with juvenile arthritis (Schanberg, Keefe, Lefebvre, Kredich, & Gil, 1996; Schanberg, Lefebvre, Keefe, Kredich, & Gil, 1997; Thastum, Zachariae, Scholer, Bjerring & Herlin, 1997) or sickle cell disease pain (Gil et al., 1993) to older adults suffering from osteoarthritic knee pain (Keefe et al., 1987.) Finally, research has shown that catastrophizing not only relates to clinical pain but also to the experience of experimentally-induced pain (Sullivan, Bishop, & Pivik, 1995; Sullivan, Rouse, Bishop, & Johnston, 1997).

Although multivariate statistics support a unique and independent relationship between catastrophizing and pain, this research area has drawn criticism regarding the reliance on cross-sectional designs. To address this question, investigators have begun conducting prospective studies. One such study conducted by Keefe, Brown, Wallston, and Caldwell (1989) found that catastrophizing predicted pain intensity ratings as well as functional impairment six months later among a sample of over 200 rheumatoid arthritis patients even after controlling for baseline pain and disability as well as other demographic and pain-related variables.

Having demonstrated that catastrophizing can indeed predict future pain ratings, pain behavior, and disability, researchers have begun to examine whether cognitive-behavioral interventions designed to reduce catastrophizing can reduce pain and disability. Research has shown that patients having persistent pain show less catastrophic thinking following interventions that include cognitive-behavioral therapy components (e.g., ter Kuile, Spinhoven, Linssen, & van Houwelingen, 1995; Bennett, Burckhardt, Clark, O'Reilly, Wiens, & Campbell, 1996; Vlaeyen, Haazen, Schuerman, Kole-Snijders, & Van Eck, 1995). In a study conducted in our lab (Keefe et al., 1990), osteoarthritis patients who showed the largest improvements in Pain Control and Rational Thinking (a composite coping factor that includes a measure of catastrophizing) following a pain coping skills training group therapy protocol had the best long- and short-term treatment outcomes.

Researchers also have begun exploring whether catastrophizing helps explain observed gender differences in the report of pain (Sullivan, Tripp, & Santor, 2000b). Several investigators have identified interesting gender differences in measures used to assess catastrophizing. Sullivan and colleagues (in press) hypothesized that the tendency to catastrophize about the pain experience may explain gender differences in reported pain levels and two recent studies have supported this hypothesis. Results reported by Sullivan et al. (2000b) showed that there were substantial gender differences in pain severity and pain behavior among a sample of healthy young adults exposed to a cold pressor task with women reporting higher levels of pain and showing more pain behavior. Interestingly, these gender differences were eliminated when catastrophizing was statistically controlled. More recently, Keefe, Lefebvre, Egert, Affleck, Sullivan, and Caldwell (in press) conducted a study of gender differences in the pain experience of osteoarthritis patients in which they examined the role of catastrophizing as a mediator. They found that women had significantly higher levels of pain and physical disability, and exhibited more pain behavior during an observation session than men. An additional analyses showed that catastrophizing mediated the relationship between gender and pain-related outcomes such that once catastrophizing was entered into the analyses, previously significant effects of gender were no longer evident. An interesting aspect of this study was that catastrophizing still mediated the gender-pain relationship even after controlling for depression. Taken together, the findings of Sullivan

et al. (2000b) and Keefe et al. (in press) highlight the importance of both pain catastrophizing and gender in understanding the pain experience.

A lingering concern about research addressing the link between catastrophizing and pain link has been the lack of theoretical model of pain catastrophizing (e.g., Geisser, Robinson, & Riley, 1999a, 1999b; Haythornthwaite, & Heinberg, 1999; Keefe, Lefebvre, & Smith, 1999; Thorn, Rich, & Boothby, 1999). Although several theoretical models have been advanced, a coping perspective offers an appealing framework for understanding the relationship. Sullivan and colleagues (in press) have suggested that persons who engage in catastrophizing may be seeking emotional support in coping with pain. Thus, catastrophizing may serve a primary communicative function, recruiting and eliciting the help of others and subsequently reducing future behavioral expectations placed on the individual in pain.

Much of the research on pain and catastrophizing has employed a trait-like conceptualization of the construct (e.g., Sullivan et al., 1995). However, catastrophizing may vary considerably from one situation to the next. Newly developed ecological momentary assessment methods (EMA; Stone & Shiffman, 1994) may provide a better understanding of the link between catastrophizing and pain. This methodology asks patients to make entries at multiple time points across the day. Research using the EMA approach has shown promise in developing a better understanding of the immediate impact of coping strategies on pain (Affleck et al., 1992; Lefebvre, Keefe, Affleck, Raezer, Starr, Caldwell, & Tennen, 1999) and this method could provide new insights into the relationship of catastrophizing to pain.

### Fear of Pain

The fear and avoidance of pain is another research topic that is attracting increased attention from investigators interested in emotion and pain (Asmundson, Norton, & Norton, 1999). It is increasingly recognized that there are people who especially fear the experience of pain and frequently avoid stimuli that they think might trigger pain, including movement, social interactions, and situations. McNeil and Rainwater (1998) developed and recently revised a fear of pain scale, and their research has supported the validity of this scale. For example, people with high fear of pain had greater escape or avoidance responses to a pain-relevant behavioral avoidance test. Crombez, Eccleston, Baeyens, van Houdenhove, and van den Broeck (1999) tested whether the fear of pain moderates pain's interference with attention and cognitive processing. They studied 40 chronic pain patients and tested attentional interference using a numerical interference test. They found that interference with attention was predicted best by a combination of high pain-related fear and pain severity, suggesting that pain-related fear creates a hypervigilance to pain. Schmidt and Cook (1999) examined whether anxiety sensitivity, which is conceptually related to the fear of pain (Asmundson et al., 1999) predicts the experience of pain during a laboratory pain induction task. Twenty-two patients with panic disorder and 22 age and sex matched nonclinical controls were assessed for anxiety sensitivity and exposed to a 2-minute cold-pressor task. Pain ratings were higher among panic than control patients, and greater anxiety sensitivity accounted for this relationship. Thus, the fear and avoidance of pain appears to worsen the experience of pain and create greater disability and cognitive interference.

Another study sought to test whether the induction of fear or anxiety could increase pain. Rhudy and Meagher (2000) studied 60 adults who were randomly assigned to one of three emotion induction conditions: a fear condition, in which they were exposed to three brief shocks; an anxiety condition, in which they experienced the threat of shock; and a neutral, no induction condition. Pain thresholds to radiant heat pain were tested

before and after the inductions. Results revealed that anxiety and fear had different effects on pain threshold. Whereas anxiety led to increased pain reactivity (as assessed by finger withdrawal), fear resulted in decreased pain reactivity. The authors note that these results agree with animal research studies in which fear has been found to inhibit pain, whereas anxiety has been found to enhance it. This study suggests that the cognitive underpinnings of fear and anxiety may be different and should be explored further for their influence on pain.

### Emotion Regulation Processes and Pain

There is a growing literature examining processes that people use to identify, understand, and express or otherwise manage their emotions (Kennedy-Moore & Watson, 1999; Mayer & Salovey, 1997; Stanton, Danoff-Burg, Cameron, & Ellis, 1994; Taylor, Bagby, & Parker, 1997). Contemporary stress theory and research indicate that the effects of stressful experiences and inner conflicts are mediated by one's emotional processing of the event (Borkovec, Roemer, & Kinyon, 1995; Greenberg & Safran, 1989; Lang, 1985; Pennebaker, 1993). In this section, we will examine two emotion regulation processes identified by Kennedy-Moore and Watson (1999) that are likely to be important in the experience of pain. The first is the ability to be aware of, identify, and understand one's emotions; and the second is whether emotions are expressed or inhibited.

Working from the perspective of stress and coping theory, Stanton et al. (1994) demonstrated that there is a type of emotion-focussed coping that appears to be adaptive rather than maladaptive. They labeled this "emotional approach coping," which denotes attempts to recognize, acknowledge, and express one's feelings. These authors demonstrated that many emotion-focussed items in standard pain coping scales, such as those labeled catastrophizing, have content that is widely considered to be a sign or manifestation of psychological disturbance or distress, rather than a cause of distress. They argued that this confounding could lead to the items' association with maladaptive adjustment. These authors created a scale that focuses on emotional abilities such as identifying, understanding, and appropriately expressing emotions, and demonstrated that greater use of emotional approach coping was related to better health, at least among women. Although this scale has not been widely used in studies of pain populations, it could provide new insights into more positive emotion regulation strategies used by many persons experiencing persistent pain.

Alexithymia (literally, "no words for feelings"), is a term coined in the 1970s by clinicians to describe a pattern seen in many patients having chronic pain, substance abuse, and other stress-related disorders (Sifneos, Apfel-Savitz, & Frankel, 1977). Although attempting insight-oriented therapy, clinicians found that many of these patients had great difficulty identifying their own feelings, differentiating feelings from other physical sensations, and communicating feelings to others. These patients also tended to think about external, concrete matters rather than introspect, and seemed to lack imagination or fantasy. Research during subsequent decades has supported these clinical observations, and alexithymia has been found to be a reliable correlate—and possibly a risk factor—for a host of medical and psychiatric disorders (Taylor et al., 1997). Alexithymia is thought to be a deficit in the ability to use cognitive mechanisms to understand and hence regulate emotions, with the result that the person experiences chronic dysphoria, impaired social relations, physiological sensations of emotions that are misinterpreted as physical symptoms, and sympathetic, hormonal, and immune changes that may predispose to illness. The primary instrument employed to assess alexithymia is the Toronto Alexithymia Scale or its 20-item revision (Bagby, Parker, & Taylor, 1994).

A number of studies have found that patients with chronic pain problems show higher levels of alexithymia than healthy controls. For example, Porcelli, Zaka, Leoci, Centonze, and Taylor (1995) assessed alexithymia in 112 patients with inflammatory bowel disease (IBD) and in a comparison group of healthy people matched for age, gender, and education. These authors found that 35.7% of the IBD patients, but only 4.5% of the control patients were alexithymic. Fernandez, Srira, Rajkumar, and Chandrasekar (1989) assessed alexithymia in rheumatoid arthritis patients and matched healthy controls using both the TAS and an interview-based measure. They found significantly greater alexithymia in the patients than controls. Other studies have found significantly greater alexithymia in patients diagnosed as having psychogenic pain disorder than controls (Sriram, Chaturvedi, Gopinath, & Shanmugam, 1987). Research has also shown that approximately one-third of patients with various chronic pain problems were classified as alexithymic, which is substantially above levels found in nonpatient groups (Lumley, Asselin, & Norman, 1997; Millard & Kinsler, 1992). It is conceivable that alexithymia contributes to chronic pain because patients are unable to differentiate emotion arousal from sensations stemming from injury, or because of the direct contributions of physiological arousal to tissue changes, or because of patient's limited ability to use cognitive strategies to diminish pain.

A second emotional regulation strategy that has been gaining substantial attention is whether or not one experiences and expresses emotions, especially negative emotions stemming from stressful experiences (Kennedy-Moore & Watson, 1999). The normal response to a stressful experience is a process of intermittent cognitive avoidance of emotional memories alternating with memory recollection—a process that usually eventuates in cognitive integration and resolution of the stressful experience. Although temporary avoidance of the emotions stemming from major stressful experiences may be adaptive (Suls & Fletcher, 1985), excessive or prolonged avoidance or rigid inhibition of negative emotional memories prevents their reappraisal, integration, and resolution (Borkovec et al., 1995; Horowitz, 1986; Pennebaker, 1993). Emotional inhibition leads to anxious rumination and/or unwanted thought intrusion, depression, physical symptoms, interference with adaptive behavior, and increased physiological arousal, which may cause autonomic and immune dysfunction and possibly disease (Borkovec et al., 1995; Pennebaker, 1993; Pennebaker & O'Heeron, 1984). Personality research indicates that people who habitually inhibit various emotions are more likely to suffer from a range of health problems (Emmons & King, 1988). Laboratory studies demonstrate that emotional inhibition leads to autonomic arousal (Buck, 1984; Pennebaker, Hughes, & O'Heeron, 1987), keeping secrets impairs and cognitive processing (Lane & Wegner, 1995; Larson & Chastain, 1990), and suppressing emotional expressions during sad or amusing films increases physiological arousal (Gross & Levenson, 1997). Studies have found that suppressing negative thoughts related to stressful experiences decreases circulating T lymphocytes (Petrie, Booth, & Pennebaker, 1998), and active suppression of emotional thoughts increases psychophysiological responses even after suppression is discontinued (Wegner & Gold, 1995).

People with chronic pain may avoid certain thoughts or emotions (e.g., Philips & Jahanshahi, 1986; Pilowsky & Spence, 1977), and such avoidance is usually associated with poorer adaptation. Emotional inhibition and avoidance of conflict is an important distinguishing characteristic among patients with chronic back pain (Coen & Sarno, 1989). Patients with intractable pain who have not responded to conventional treatment report significantly higher anger inhibition compared to patients reporting pain as a prominent symptom of a current medical illness (Pilowsky & Spence, 1977). Pilowsky and Bassett (1982) reported that denial of both affective disturbance and life problems clearly differentiated 114 patients having pain from 53 psychiatric inpatients with depression. Finally,

among males with chronic pain, the suppression of anger has been found to predict poorer outcome following multidisciplinary pain management (as assessed by measures of depression and activity) (Burns, Johnson, Devine, Mahoney, & Pawl, 1998).

Several studies have used experimental designs to directly test whether mental suppression can exacerbate pain. For example, Cioffi and Holloway (1993) had students engage in a cold-pressor task after being instructed either to intentionally suppress thoughts about their painful sensations, or to monitor their pain. Those assigned to suppress pain thoughts reported the slowest recovery from the cold pain stimulus and later reported that an innocuous vibration was more unpleasant than did participants who were instructed to monitor their pain experience. In addition, when asked later in the semester to recall the degree of pain they experienced during the cold-pressor task, those who had been told to suppress pain recall having significantly greater pain than subjects who had been asked to monitor their pain. In a study using similar methods, Sullivan, Rouse, Bishop, and Johnston (1997) experimentally examined the effects of thought suppression of an upcoming painful ice water immersion. Those instructed to suppress pain thoughts reported significantly more thought intrusions regarding the upcoming procedure (e.g., "I wonder if it will hurt") and reported more severe pain during the actual ice water immersion compared to participants who were asked not to suppress (Sullivan et al., 1997).

Taken together, the studies reviewed in this section suggest that problems in regulating and expressing emotions are linked to increased pain and distress.

#### Enhancing Emotional Regulation: Potential Effects on Pain

Although alexithymia and limited emotional awareness are associated with increased pain and other health problems, few studies have examined whether interventions designed to increase emotional awareness can improve health and reduce pain. Only one controlled study has attempted to improve alexithymia and examined the health consequences, and this was not conducted in persons with coronary artery disease who did not have persistent pain (Beresnevaite, 2000.) In this study, a group therapy intervention that included wide variety of treatment techniques was found to significantly reduce alexithymia when compared to a control group. Importantly, reductions in alexithymia were associated with a significant reduction in subsequent coronary events among those patients receiving the group therapy intervention.

Clinical observations suggest that traditional insight-oriented therapy is of little benefit to patients who lack emotional awareness. It has been suggested that by modifying insight-oriented therapy or providing other emotional reeducation experiences one might have more of an impact on alexithymia. Krystal & Krystal (1988), for example, suggests a modified psychodynamic approach in which the therapist is much more active in teaching patients to tolerate affective states, to attend to bodily states, to identify and label their emotions, and to note and try to understand their dreams. Levant (1998) has described a therapy protocol in which alexithymic, physically symptomatic clients are asked to engage in homework assignments to increase their awareness of their emotions and the relationship of emotions to their symptoms. Levant reports that, following treatment, most of these clients show improved emotional awareness and a reduction in symptoms, such as less temporomandibular joint pain or low back pain. Unfortunately, to our knowledge the interesting clinical techniques for alexithymia described by Krystal & Krystal (1988) and Levant (1998) have not been tested in controlled treatment outcome studies.

Although research into the health effects of improving emotional awareness or decreasing alexithymia is limited, there is a substantial body of research on the benefits of emotional disclosure and expression. Correlational research indicates that verbal disclo-

sure about surviving traumas such as the Holocaust (Pennebaker, Barger, & Tiebout, 1989) or losing a spouse (Pennebaker & O'Heeron, 1984) is associated with better health and reduced sympathetic arousal. Evidence from psychotherapy studies also shows that various clinical problems (e.g., anxiety disorders) can be improved by repeated exposure to negative emotional stimuli (objects, memories, physiological arousal, emotions themselves) (Daldrup, Engle, Holiman, & Beutler, 1994; Foa & Kozak, 1986; Greenberg, Rice, & Elliott, 1993; Lang, 1985; Rachman, 1980). These studies have revealed that exposure leads to cognitive changes and the eventual alteration of the negative emotions.

What about the effects of emotional disclosure in patients with chronic pain and other stress-related physical disorders? Some authors have proposed that emotion-provoking psychotherapy may exacerbate symptoms in patients who have alexithymia or problems processing emotions, including patients with pain conditions (Sifneos, Apfel-Savitz, & Frankel, 1977). Indeed, several studies with chronic pain patients have attempted to implement a form of psychotherapy, in which intense emotions, especially anger, are elicited using exercises and Gestalt techniques. Beutler et al. (1987), for example, randomly assigned 18 patients diagnosed as having psychogenic pain to either focused expressive therapy or a control, didactic therapy. Although there was some evidence of improvement in depression for the therapy patients, there was no improvement in pain. In a study of six patients with rheumatoid arthritis, Beutler and Norcross (1987) found that focused expressive therapy produced some improvement in depression and an activation of the beta-endorphin system. Although pain levels were lower after each session, the average pain level of patients increased during the weeks of treatment. At follow-up, two patients had slightly higher pain than when they started, and only one improved. These studies suggest that although intensive emotion-provoking therapy might improve the depression of persons having pain, it may not help pain. However, it is possible that a less intensive and more supportive interpersonal therapy, designed to help people express inhibited emotions in an interpersonal context, may be more effective for emotional regulation and pain control.

Another intervention that appears to be helpful in enhancing emotional regulation involves encouraging people to disclose their emotional difficulties privately, and thereby process or work through their emotional experience at their own pace. This approach has been tested in experimental studies originated by Pennebaker in the 1980s (Pennebaker, 1985). The basic design of these studies involves assigning participants to one of two groups: (1) writing privately for about 20 minutes per day for several days in a row about their most difficult or stressful experiences, or (2) writing about emotionally neutral, control topics. The two groups are then compared to examine changes in participants' health status from baseline to follow-up. This emotional disclosure paradigm is self-directed and self-paced, which may limit the potential for symptom exacerbation, because people are able to control the rate and extent that they disclose and process experiences and emotions.

Most of the initial studies of this emotional disclosure paradigm were conducted with college or medical students, or with healthy people in the community. These studies indicated that expressing stressful emotional experiences via private writing (or even privately talking into a tape recorder) is beneficial to health. Written or verbal emotional disclosure, for example, has been found to lead to health benefits, such as decreased health center visits (Greenberg & Stone, 1992; Greenberg, Wortman, & Stone, 1996; Pennebaker & Francis, 1996), enhanced immunological response (Petrie, Booth, Pennebaker, Davidson, & Thomas, 1995), improved grade point average, and faster reemployment after being laid off (Spera, Buhrfeind, & Pennebaker, 1994).

Studies of emotional disclosure in populations experiencing pain or other medical conditions have only recently been conducted. Kelley, Lumley, and Leisen (1997) examined 72 adults with rheumatoid arthritis who were randomized to talk privately into a tape recorder at home for four days about either their most stressful experiences (disclosure group) or about neutral topics (descriptions of pictures, control group). A range of health measures was assessed at baseline and again at two weeks and three months following the intervention. A review of audiotapes showed that the arthritis patients disclosed a wide range of stressful topics, including the effects of their disease and a host of other nondisease-related topics, particularly about troubled relationships. The groups did not differ in health status at two weeks, but at three months, the disclosure group reported significantly less affective disturbance and a better ability to conduct activities of daily living than the controls. In addition, those disclosure patients who experienced the greatest increase in negative mood after verbal disclosure each day had the most improvement in their joint condition at three months, as determined by physical tests and a joint examination by a physician.

Smyth, Stone, Hurewitz, and Kaell (1999) recently reported that private emotional disclosure led to objective health improvements in 51 rheumatoid arthritis patients. In this study patients were randomized to two conditions: (a) writing for three days in the laboratory about stressful experiences (disclosure condition), or (b) writing for three days about how they had managed their time in recent days (control condition). Patients were assessed by a rheumatologist for their global level of impairment at baseline and at several follow-up periods. Although no differences in outcome were noted at two weeks or two months follow-up, at four months follow-up the disclosure patients had significant reductions in global impairment scores compared to controls. A noteworthy feature of this study was that health status was assessed objectively, i.e., by physicians who were blind to group status of patients. Although this study did not report separate outcomes for pain, the physicians' global ratings of impairment took into account a number of aspects of the disease, such as joint condition, disability, movement, mood, as well as pain.

Another recent study used a variation of the disclosure paradigm in a sample of people undergoing a potentially painful dental procedure (Sullivan & Neish, 1999). This study also examined whether pain catastrophizing served as a moderator of the effects of written disclosure. Prior to the dental procedure, these researchers asked 80 college students to complete a measure of pain catastrophizing. These students were then randomly assigned to write privately for 5 minutes either about their thoughts and feelings associated with the upsetting aspects of dental treatment (disclosure group) or about daily activities (control group). Pain and negative mood were assessed immediately after treatment. Data analysis showed that there was a significant interaction between treatment group and catastrophizing for both pain and mood outcomes. Students were scored high on catastrophizing, and those who were in the disclosure condition reported significantly less pain and better mood than students who scored high on catastrophizing and were in the control condition. That is, written disclosure reduced pain and improved mood primarily among those people who were particularly vulnerable to having a negative experience during dental treatment.

Both psychological and physiological explanations for the health benefits of disclosure have been proposed, and research suggests that two processes, disinhibition of negative affect and cognitive processing, may underlie health benefits. Yet, it currently is unclear whether emotional disclosure decreases pain through psychological coping mechanisms, or if emotional disclosure directly alters physiological mechanisms that contribute to pain. The available studies also indicate that disclosing negative emotions is distressing in the short term, with increased negative mood immediately following dis-

closure (Kelley et al., 1997; Pennebaker & Beall, 1986), and the benefits usually take several months to occur. These observations agree with those found in research on the treatment of anxiety disorders, which indicates that successful emotional processing necessitates temporary activation of the emotional memory (schema or network), which is experienced as increased negative emotion. This activation permits the emotional schema to be cognitively reconfigured over time with affect-disconfirming information of less threat or harm (Foa & Kozak, 1986; Lang, 1985). Pure emotional experience or catharsis without cognitive change is not helpful (Pennebaker & Beall, 1986), and it appears that a person's understanding about their emotional experience must increase over the course of repeated or prolonged exposure for benefits to accrue.

#### Emotional Distress, Pain Complaints, and Treatment Seeking

Numerous studies have documented that patients with various chronic pain problems have higher levels of emotional problems, such as depression and anxiety than do normal controls. Yet, people with pain problems come to the attention of clinicians by virtue of their behavior—they complain of pain and seek treatment for it. Recent research suggests that emotional distress contributes substantially to treatment seeking. Research studies in this area have taken advantage of a unique group against which to compare patients with pain who are seeking treatment—people who have the same pain condition, but who are not treatment-seeking patients.

Drossman (1988) compared 72 patients who sought treatment for painful, irritable bowel syndrome (IBS) with 82 people with IBS who had not sought treatment, and 84 healthy people. Reported pain and diarrhea were highest in the patient group, but after statistically controlling for these severity of these symptoms, the IBS patients continued to show higher levels of emotional distress on various Minnesota Multiphasic Personality Inventory (MMPI) scales, to have greater health worries, and to have experienced fewer positive life events than the people who had IBS but were not seeking treatment, who did not differ from the healthy participants. Whitehead, Bosmajian, Zonderman, Costa, and Schuster (1988) also studied IBS patients seeking treatment and nontreatment seeking persons with IBS as well as healthy people, and found results that paralleled those of Drossman (1988). Both sets of authors concluded that emotional distress was associated with seeking treatment for IBS, rather than with having IBS symptoms per se.

Aaron et al. (1996) compared 64 patients who had fibromyalgia syndrome (FMS) with 28 people with the symptoms of FMS recruited from the community but who were not seeking treatment, and 23 healthy people. They administered structured psychiatric interviews, and found that the FMS patients had a greater number of lifetime diagnoses, compared with FMS nonpatients, who did not differ from healthy controls. Anxiety and mood disorders were the most commonly diagnosed conditions that differentiated FMS patients and nonpatients.

Using a similar methodology, Ziegler and Paolo (1995) compared headache symptoms as well as psychological profiles based on the MMPI-2 of clinical patients with headaches, as well as a group of people with headaches, but who had not sought treatment in the past two years. Clinic patients had higher scores than nonpatients on a number of scales of the MMPI-2, including scales assessing depression, anxiety, as well as the hypochondriasis, hysteria, and social introversion scales. This difference held even after statistically controlling for differences in the severity of the respondent's most intense headaches.

Finally, Lumley and colleagues (Torosian, Lumley, Pickard, & Ketterer, 1997) have used another approach to address this issue. This group examined chest pain or angina

during myocardial ischemia (a lack of oxygen to the heart muscle) among people referred for treadmill exercise and radiographic imaging of the heart. Among a cohort of 276 patients, 106 of these patients were found to have ischemia during exercise that was caused by heart disease. Of these patients 67 did not report chest pain during ischemia ("silent ischemia"), whereas 39 patients did (symptomatic ischemia). This study found that silent ischemia patients had greater control over anger, and were more externally focussed than patients with chest pain. In addition, it showed that the silent ischemic patients had used primary care and emergency services less often than symptomatic ischemic patients, even after controlling for various health risk and demographic factors (Lumley, Rowland, Torosian, Bank, Ketterer, & Pickard, 2000). In this study, there also was a smaller group of patients who complained about chest pain during exercise in the absence of cardiac ischemia, who reported the highest levels of psychological distress and had higher levels of stress and abuse in their histories. These findings are consistent with other studies (e.g., Freedland, Carney, Krone, & Smith, 1991; Mayou et al., 1989) indicating that elevated negative emotional states are associated with complaining about chest pain, even in the absence of underlying cardiac disease, whereas not complaining about chest pain in the presence of disease is associated with tendencies to deny emotional distress and not seek treatment.

In summary, these studies suggest that emotional distress has a strong influence on the treatment seeking behaviors of persons with pain symptoms. Emotional distress may lead people to interpret sensations as symptoms of pain, or it may simply prompt treatment seeking. Both researchers and clinicians need to remember this bias to counteract the tendency toward developing illusory correlations.

#### Future Research Directions

There are a number of important directions for future research on emotion and pain. In this section, we highlight some directions in which we are moving, as well as directions we would suggest that others pursue.

Research on the relationship between affect and pain has only recently begun to benefit from advances in the past 15 years in psychometric work geared toward identifying the dimensions of self-reported affect. Pain researchers have begun to shift from using older, content-derived measures, like the POMS (McNair, Lorr, & Droppleman, 1984) or MAACL (Zuckerman, Lubin, & Rinck, 1983), and toward the factorially derived PANAS (Watson, Clark, & Tellegen, 1999) that assesses positive affect (PA) and negative affect (NA.). However, it is unclear if the parsimonious two-factor model of affect can provide as salient a conceptual model for understanding pain as it has for understanding depression and anxiety. Many pain researchers are interested in both depression (or other emotions) and pain, and thus may be most interested in how pain and depression (or other emotions) independently interact with each other. A more complex model, like the circumplex, may prove useful in identifying more direct linkages between affect and pain. Circumplex theorists assume dependence among affect states and have chosen to empirically map affect on a two dimensional space, which essentially places PA and NA along the x-axis, or a *single* valiance continuum. The y-axis has received different labels but can be thought of as an *activation* continuum. The circumplex and factorial models are similar in content, at least for the x-axis, though the factorial proponents (e.g., Watson & Clark, 1997) would argue that activation is not an affect state but an intensifier.

We believe that this recent work in measuring and conceptualizing self-reported affect, if coupled with studies that examine empirical-conceptual links to specific disor-

ders may lead to new models for pain. The resulting affect–pain models may appear very different from the affect–anxiety and affect–depression models developed by Clark and Watson (1991). There is good reason to believe that pain involves a unique set of experiences that may not be captured by the above affect models. For example, when individuals were asked to rate a comprehensive set of affect words in terms of the extent to which the words truly identified emotional experiences, an empirically derived class of “Physical and Bodily States” emerged as separate from three other types of affect categories (Clare, Ortony, & Foss, 1987). It seems plausible that an empirical analysis of pain experiences might yield separate dimensions that could be especially useful in pain research. Further, a dimensional model of self-reported pain might help us understand if flaring joint pain is described in a way that is empirically distinct (other than by bodily location) from intense migraine pain.

In general, the relationship between negative affect and pain has been found to be stronger than the relationship between positive affect and pain. Watson (2000) argues that positive affect only emerges as a significant predictor of health complaints when analyzing within-subject fluctuations in affect across time. If Watson is correct, advances in research on pain and affect may depend on the ability of researchers to track individual changes within individuals. Detecting such findings may require the use of more robust statistical models such as time series analysis or hierarchical linear modeling that are just beginning to be used by pain researchers (e.g., Affleck et al., 1992.)

With respect to emotional expression, research should investigate the construct of ambivalence over emotional expression (King & Emmons, 1990). Not expressing an emotion does not necessarily mean that the individual is suppressing or inhibiting expression. Instead, a desire to express an emotion coupled with a desire to inhibit the emotion may exacerbate obsessive thoughts about the inhibited emotion or action, increase chronic autonomic arousal, and make psychosomatic disease more likely (King & Emmons, 1990; Pennebaker, 1985). Ambivalence regarding emotional expression rather than expressiveness itself has been found to be significantly associated with negative mental health outcomes (King & Emmons, 1990). Emotional ambivalence has also been shown to be a significant mediator between dependency and eating disorder pathology, as well as between chronic negative emotion intensity and acute psychological distress (Krause, Robins, & Lynch, *in press*; Lynch, Robins, Morse, & Krause, 2000). Future studies need to explore whether emotional ambivalence is an important variable influencing the experience of pain.

With respect to emotional interventions for chronic pain patients, the negative or neutral effects of intensive emotion-provoking psychotherapy (Beutler et al., 1987) need to be reconciled with the apparent benefits written or verbal emotional disclosure interventions (Kelley et al., 1997; Smyth et al., 1999). It needs to be determined exactly what processes of emotional disclosure and processing are necessary and helpful, and whether having a supportive, less emotion-provoking therapist can facilitate the process. It is likely that therapist responses will be crucial, at least for certain types of patients, and research needs to understand for whom therapist-facilitated versus patient-directed disclosure and processing is more beneficial. We are currently conducting a study that compares self-directed emotional disclosure with a supportive nurse-facilitated disclosure intervention in rheumatoid arthritis patients. Additionally, it may be possible to alter Pennebaker’s basic paradigm by adding additional guidance or didactic instructions to help patients who are alexithymic or who are not familiar with emotional exploration. Also, research needs to test the various clinically derived approaches to helping alexithymic patients develop emotional awareness (e.g., Levant, 1998), and to determine whether this increased awareness leads to pain reduction.

Finally, it is recommended that cognitive-behavioral treatments for pain need to be systematically compared with emotional disclosure interventions. These two approaches seem diametrically opposed—CBT usually seeks to decrease emotional arousal and avoid negative emotion through techniques such as relaxation, distraction, and positive imagery, whereas emotional disclosure techniques encourage the experience of negative emotions. How these two approaches compare in efficacy and how they might be integrated is an important and interesting direction for future research on pain and emotion.

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