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Pain and Emotion: A Biopsychosocial Review of Recent Research

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Abstract

Objective and Method—Research on emotion and pain has burgeoned. We review the last decade's literature, focusing on links between emotional processes and persistent pain.

Results—Neurobiological research documents the neural processes that distinguish affective from sensory pain dimensions, link emotion and pain, and generate central nervous system pain sensitization. Psychological research demonstrates that greater pain is related to emotional stress and limited emotional awareness, expression, and processing. Social research shows the potential importance of emotional communication, empathy, attachment, and rejection.

Conclusions—Emotions are integral to the conceptualization, assessment, and treatment of persistent pain. Research should clarify when to eliminate or attenuate negative emotions, and when to access, experience, and express them. Theory and practice should integrate emotion into cognitive-behavioral models of persistent pain.

Keywords

Persistent pain; chronic pain; emotion; biopsychosocial model

Pain is the most common symptom reported to health care providers, is a driving force of health care utilization and lost productivity, and exacts a substantial toll on the afflicted, their loved ones, and society in general. Pain is a prevalent symptom not only in primary medical care and specialty pain clinics, but also in mental health and substance dependence treatment settings. Thus, it is vital that psychologists remain abreast of recent theory and research that informs and directs case conceptualization, assessment, and intervention among patients experiencing pain.

Definitions, Controversies, and Clarifications

The International Association for the Study of Pain defines pain as "an unpleasant sensory and emotional experience associated with actual or potential damage, or described in terms of such damage" (Mersky & Bogduk, 1994). Thus, pain is partially an emotional experience, and the correspondence between pain and bodily damage is variable. Although these two points are widely acknowledged by experts in pain research and practice, they are still not

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fully appreciated in more general practice settings, where pain is often treated as a purely sensory experience reflecting underlying tissue damage. As a result, there remain major gaps between our understanding of persistent pain and the ways that many patients are assessed and treated. In this section, we address several controversial issues and delineate the boundaries of our review.

Types of Pain

Pain is traditionally dichotomized into acute and chronic. As an indicator of potential tissue damage, acute pain can be viewed as an adaptive alarm, alerting the person to attend to the cause of the pain and motivating action to prevent tissue damage, protect the affected body part, and avoid similar future encounters. Although of interest to some psychologists, such as those working with painful medical procedures, acute pain typically is conceptualized, evaluated, and treated biomedically.

Chronic pain or persistent pain, which is defined as lasting at least 3 months, is more complicated than acute pain. In particular, learning occurs—neurobiological, psychological, and social changes that can maintain the pain. The adaptive alarm of acute pain loses some efficiency, because pain no longer is a reliable indicator of tissue damage, and behavioral changes to reduce pain may be maladaptive (Nesse & Ellsworth, 2009). Consequently, people with persistent pain are much more likely than those with acute pain to come to the attention of psychologists, either at a pain clinic for treatment of pain itself, or via traditional therapy to address maladaptive correlates of pain, such as mood problems, substance abuse, or relationship difficulties.

The nosology of persistent pain is inconsistent and evolving. Some types of pain are tied to disease processes in specific tissues, including joint degeneration (osteoarthritis), inflammation (e.g., rheumatoid arthritis, inflammatory bowel disease), tumor growth (cancer pain), damaged nerves (neuropathic pain), or tissue anoxia (sickle cell disease). There also is a heterogeneous group of pain problems that have traditionally been classified according to location, such as low back, neck, head, abdomen, pelvis, and chest. The latter includes pain presentations that are part of broader multi-symptom syndromes, such as fibromyalgia or irritable bowel syndrome.

These latter syndromes and types of pain are particularly vexing because clinical and laboratory studies do not reliably indicate abnormalities in the peripheral tissue sites where patients experience pain. Furthermore, many of these apparently dissimilar pain conditions co-occur; for example, it is quite common to find a patient reporting headaches, abdominal pain, and fibromyalgia. A recently developed conceptual framework substantially advances our understanding of these problems. The concept of “central sensitivity syndrome” describes “an overlapping and similar group of syndromes without structural pathology and are bound by the common mechanism of central sensitization (CS) that involves hyperexcitement of the central neurons through various synaptic and neurotransmitter/ neurochemical activities” (Yunus, 2007; p. 339). Central sensitization implies that the brain and spinal cord are more important in generating the persistent pain experience than peripheral tissues. Central or peripheral pain is not a simple dichotomy, however, because both central and peripheral mechanisms are involved in pain. For example, central sensitization develops after peripheral damage such as osteoarthritis (Arendt-Nielsen et al., 2010) or neck injuries (Banic et al., 2004).

Mind-body Dualism and Somatization

Two related and controversial concepts are mind-body dualism and somatization. Dualism views pain as caused by either biological factors or psychological factors, and these

processes are distinct. Such dualistic thought remains common among both lay people and professionals, and is seen, for example, when patients seek organic validation that their pain "is real" lest they be viewed as having psychological or "functional" problems and be accused that the pain is "all in their heads." Fortunately, dualism is increasingly being challenged by more sophisticated models of pain that recognize that psychosocial and biological processes are tightly integrated if not isomorphic—the brain is the basis of mental processes. In our view, pain is undoubtedly real—regardless of whether peripheral or central abnormalities are found—in part because the brain is the organ where pain is experienced and modulated, and in part, simply because people experience and report pain.

As originally defined by Lipowski (1988), somatization is the tendency to experience, communicate, and seek care for somatic symptoms that are disproportionate to pathological findings. A key tenet is that patients who somatize are reluctant or unable to acknowledge psychological or emotional problems, and their somatizing represents an alternative pathway to communicating their difficulties. Somatization is a concept used by both lay people and professionals to explain symptoms that seem disproportionate or excessive. The concept, however, is quite controversial, and some have argued that it should be eliminated (Merskey, 2009), particularly because little sound research supports the construct, and most studies purporting to demonstrate somatization measure only symptoms but fail to test the assumed mechanisms (Crombez, Beirens, Van Damme, Eccleston, & Fontaine, 2009). We believe, however, that a lack of good research does not fully invalidate a concept. Rather, both conceptual refinement and rigorous empirical testing are needed to determine whether and how emotions influence pain, so that more powerful assessment and intervention approaches can be developed.

Emotional States and Emotional Processes

The burgeoning research on pain and emotions has been spurred by a shift in theory. Traditionally, emotion has often been viewed as less mature than reason, and negative emotion as pathological and needing rational control. More recently, affective science has espoused a functional or evolutionary model, which views emotion as having the potential to facilitate awareness and guide and motivate adaptive behavior (Nesse & Ellsworth, 2009). Such a view underlies our review of the literature.

We have articulated a view of emotions as subsuming two distinguishable types—emotional states and emotional processes (Lumley, 2010). Emotional states include transitory moods, longer duration affects, and various emotional disorders (e.g., mood or anxiety disorders). Emotional processes, in contrast, refer to the mechanisms by which emotions and generated, experienced, and used; and include emotional awareness, labeling, expression, processing, and integration. Emotional processes strongly influence emotional states as well as mental, behavioral, and physical health more generally. Research on such emotional processes is more recent than that on emotional states, and its application to pain is newer yet.

Our conceptualization of emotional processes is broader than "emotion regulation"—a label that typically refers only to the attenuation or reduction of emotional experience or expression (Gross, 2002), rather than the fuller range of processes, including enhancing awareness, experiencing, and expression. Emotional processes also are broader than emotion-focused coping, which refers to volitional strategies or actions targeting emotions. Studies routinely suggest that emotion-focused coping strategies are maladaptive; yet, items in most of these coping scales are contaminated with distress or negative affect, resulting in a biased relationship between emotion-focused coping and pain or dysfunction (Stanton, Danoff-Burg, Cameron, & Ellis, 1994).

The Boundaries and Goals of this Review

The literatures on both emotion and pain are substantial, necessitating that we draw boundaries to focus this review. With respect to pain, our interest is persistent pain, due to its greater relevance than acute pain to psychologists. Yet, we include research on experimentally-induced acute pain when such studies are informative. With respect to emotion, we recognize the importance of emotional states, particularly depression, anxiety, and anger as responses to pain. But the literature on these states and disorders is rather substantial, and a recent comprehensive review of persistent pain discussed them (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). In contrast, we lack reviews on pain and emotional processes, emotional modulation of pain, and the role played by emotional states in neurobiological and social research on pain.

Persistent pain is complex, and emotions are only one contributing factor. We recognize that other factors are important, but they cannot be covered in this review. These includes genetic factors, environmental contingencies and models, overt behavior (e.g., exercise, deconditioning), the placebo effect, and cognitions. We recognize that the distinction between cognition and emotion is tenuous; most emotional experience involves cognitions, such as core beliefs, assumptions, perceptions, and expectancies. Furthermore, constructs typically considered cognitive, such as self-efficacy and controllability have great implications for pain but have been reviewed elsewhere (Gatchel et al., 2007). There are also many topics that have implications for emotions and are linked to pain, such as psychopathology or personality traits; however, we focus primarily on studies of emotions or closely related processes.

A decade ago, several of us published a review of the available literature on pain and emotion (Keefe et al., 2001), and research has accelerated since then. In this article, we review research on pain and emotion published in the last decade. Following the biopsychosocial model, we start with research on the neurobiology of pain and emotions, because doing so underscores our view that the brain generates and organizes psychological and social experience and is the organ most relevant to persistent pain. We next examine research on the psycho-emotional processes and pain, followed by socio-emotional processes and pain. Within each domain, we examine several topics and present illustrative studies. Due to the volume of research, this review is not exhaustive; rather, we provide a representative sampling of the literature. At the end of the article, we highlight several limitations and future research directions, and then summarize and offer clinical implications.

Neurobiology, Emotions, and Pain

This decade has witnessed substantial growth in neurobiological research on pain and emotion. There has been continued development of earlier paradigms such as lesion, stimulation, and pharmacologic intervention of the central nervous system (CNS) of animals, and newer approaches using brain imaging. In this section, we examine findings on the processes underlying the sensory and the affective components of pain, central sensitization of pain pathways, brain processes linking pain and emotions, and imaging research that elucidates the neural basis of emotions and pain.

Sensory and Affective Dimensions of Pain

It has been argued that the human pain experience is composed of three dimensions (Melzack & Casey, 1968). The sensory-discriminative dimension identifies the location, timing, and physical characteristics (e.g., mechanical, chemical, heat) of the noxious stimulus, and prompts withdrawal reflexes to prevent or limit tissue damage. The affective-motivational dimension, which is the one most closely linked with emotion, underlies the

unpleasantness associated with exposure to a noxious stimulus and activates defensive behaviors such as escape and recuperation, which enable the individual to cope with the noxious stimulus. Finally, the cognitive-evaluative dimension influences the appraisal of the meanings and consequences of an injury or pain.

The first two dimensions are supported by separate but parallel neural systems. The lateral pain system, which supports the sensory-discriminative dimension of pain, has axons that ascend laterally within the spinothalamic tract of the spinal cord, synapse within lateral nuclei of the thalamus, and ultimately project to the primary somatosensory cortex. The medial pain system, which supports the affective-motivational dimension of pain, arises from neurons whose axons project medially within the spinothalamic tract in the cord and brainstem and synapse within medial thalamic nuclei before projecting to a number of regions, including the cingulate cortex and limbic system. This medial pain system underlies pain affect (Vogt & Sikes, 2000), and provides “emotional coloration” to painful stimuli (Rome & Rome, 2000).

Both experimental and clinical studies support the distinction between medial and lateral pain systems. For instance, Kulkarni et al. (2005) found that directing people to pay attention to the location of a noxious stimulus activated the primary somatosensory cortex, whereas attention to the unpleasantness of the noxious stimulus activated the medial pain system. Damage to the lateral pain system makes it difficult to localize or describe the physical characteristics of pain; however, an unpleasant experience persists (Ploner, Freund, & Schnitzler, 1999). People whose pain is treated by destroying part of the medial pain system (e.g., the cingulate cortex or medial thalamus) report alleviation of the affective component of pain, but no loss of sensory-discrimination. Such observations have influenced clinical practice. For example, destroying the cortical and thalamic components of the medial pain system as a treatment for intractable pain has regained some popularity (Romanelli, Eposito, & Adler, 2004).

Central Sensitization and Pain Affect

Persistent pain appears to be mediated by neural plasticity or sensitization, particularly in the medial pain system. The medial thalamus is the principal relay of nociceptive input to the anterior cingulate cortex, and persistent stimulation of this pathway by pain in peripheral tissues changes neurons in the cingulate cortex (Shyu & Vogt, 2009; Zhuo, 2007). Thus, persistent pain, particularly when initiated by peripheral injury or stimulation, is associated with long-term changes in the morphology, neurochemistry, and gene expression in the anterior cingulate cortex, which contribute to the maintenance and exacerbation of pain (Cao et al., 2009). Such central sensitization is characterized by an enhanced pain response to normally painful stimuli (hyperalgesia), a decrease in pain threshold to normally non-painful stimuli (allydonia), and an increase in spontaneous activity (spontaneous pain).

The medial pain system also projects to a number of subcortical sites that are key to emotions, including the amygdala, hypothalamus, and periaqueductal gray. For example, the amygdala is activated during persistent arthritic pain, but not acute experimental pain (Kulkarni et al., 2007). In rats, pain-induced sensitization of neurons within the amygdala contributes to persistent pain affect associated with arthritis (Neugebauer, Galhardo, Maione,& Mackey, 2009). As expected, lesioning or injecting morphine into the amygdala suppresses rats’ emotional responses to a painful stimulus (Nandigama & Borszcz, 2003).

Peripheral pain also induces changes in neurons projecting from the basolateral amygdala to the medial prefrontal cortex. These projections are implicated in cognitive and emotional processes such as value-based decision-making; for example, avoiding risky choices in favor of adaptive, goal-directed behavior (Kouneiher, Charron, & Koechlin, 2009). Sensitizing

these projections in a rat model of arthritis deactivated the medial prefrontal cortex and impaired decision-making (Ji et al., 2010). Studies of people with complex regional pain syndrome or back pain found impaired performance on emotional decision-making tasks that resemble that of patients with lesions in their prefrontal cortex (Apkarian et al., 2004).

Changes in the neurons projecting from the basolateral amygdala to the ventromedial hypothalamus also contribute to long-term increases in pain. The dorsomedial division of the ventromedial hypothalamus organizes innate defensive behaviors to threats, including pain (Braz, Nassar, Wood, & Basbaum, 2005). Stimulating this structure in rats elicits pain-like emotional behaviors, and manipulating inhibitory neurotransmitters within this structure alters rats' emotional response to a painful shock (Borszcz, 2006). Furthermore, partial kindling of the basolateral amygdala in rats generates long-term sensitization of neurons in this structure, which correlates with increases in their affective response to painful shocks (Borszcz & Spuz, 2009). This form of central sensitization may contribute to the persistent pain of fibromyalgia, for example, because people with this condition have augmented defensive reactions to threatening stimuli (Bartley, Rhudy, & Williams, 2009).

Neurobiological Links Between Pain and Emotional States

The brain supports a complex cyclical interaction between pain and specific emotional states. There is a subcortical circuit that governs defensive responses, and this circuit involves the nonconscious processing of stimuli that underlie emotional states associated with persistent pain. When dysregulated, this subcortical defensive circuit interacts with the cerebral cortex and yields the conscious experience of fear and anxiety as well as evaluation and rumination about the consequences of pain or injury, including fear of pain (Johnson, Nolen-Hoeksema, Mitchell, & Levin, 2009). Thus, sustained activation of these cortical sites by the dysregulated subcortical defense circuit may contribute to secondary emotional reactions associated with pain, which then can contribute to further suffering and disability (Ericsson et al., 2002). Furthermore, reciprocal projections of the cortex to the subcortical defense circuit can either exacerbate or inhibit dysregulation, suggesting that conscious processes modulate fear, suffering, and disability (Shin & Liberzon, 2010).

Both fear and anxiety influence pain, but these two emotional states do so quite differently. Fear is elicited by a present or imminent threat and motivates defensive responses such as escape. In contrast, anxiety stems from anticipation of threat and is characterized by hypervigilance and passive defensive responses. Research shows that fear of an external stimulus can inhibit pain in both humans and animals through activation of endogenous opioids, whereas anxiety increases pain (Rhudy & Meagher, 2000). Yet, repeated fear experiences can elicit anticipatory anxiety, thereby contributing to persistent pain. For example, rats exposed to the stress of swimming, social defeat, novel environments, cold, loud noise, or restraint exhibit long-term increases in pain sensitivity (Andre et al., 2005; Khasar, Green, & Levine, 2005; Suarez-Roca, Leal, Silva, Pinerua-Shuhaibar, & Quintero, 2008), but pre-treating these animals with diazepam to reduce anxiety prevents this stress-induced increase in pain (Andre et al., 2005). In people, increased pain sensitivity is seen when people expect pain, and this is accompanied by increased neural activity in the anterior cingulate cortex (Benedetti, Lanotte, Lopiano, & Colloca, 2007). As with animals, giving people diazepam reduces both anticipatory anxiety and pain sensitization (Benedetti, Amanzio, Vighetti & Asteggiano, 2006).

In contrast to the effects of anxiety in augmenting pain, positive emotional states generally reduce pain. The neural substrates that underlie reinforcement contribute to pain suppression, presumably by reducing the distress that accompanies pain—a phenomenon referred to as "affective analgesia" (Franklin, 1998). Activation of certain dopamine neurons underlies the reinforcement produced by food, water, sexual interaction, and drugs of abuse.

Activation of this system suppresses emotional reactions to pain (Kender, Harte, Munn, & Borszcz, 2008), and positively correlates with induction of placebo analgesia and positive mood ratings during placebo (Zubieta & Stohler, 2009). Additionally, activation of the brain reward circuitry contributes to the positive emotional state created by pleasant music, which reduces pain through mechanisms that may involve inhibition in the amygdala (Blood & Zatorre, 2001). The reduction of pain associated with viewing pictures of a romantic partner or with orgasm is also associated with activation of the pain reward circuit (Bianchi-Demicheli & Ortigue, 2007; Younger, Aron, Parke, Chatterjee, & Mackey, 2010). Fields (2007) has proposed that reward or positive emotions are linked to pain analgesia, in part, through opioids acting on a dopaminergic mesostriatal circuit. Opioids suppress responses to various noxious stimuli, including pain, in the presence of a conflicting motivation, such as hunger or sex. Research supports this model by demonstrating that the amount of relief experienced when an acute painful stimulus ends is correlated positively with the degree of activation of this brain reward circuit (Baliki, Geha, Fields, & Apkarian, 2010).

Brain Imaging, Emotional Processes, and Pain

A final category of neurobiological research on pain and emotion uses imaging studies to demonstrate brain pathways that link pain and emotion-relevant processes. Among patients with irritable bowel syndrome, inducing pain by distending the rectum activates the anterior cingulate cortex, and the degree of activation correlates positively with anxiety, stressful life events, and a history of abuse, suggesting the importance of early emotional experiences in pain responding (Ringel et al., 2008). As discussed later, social rejection (exclusion from a virtual ball-tossing game) leads to activation of the anterior cingulate cortex, which is similar to what occurs when a person is given painful stimulation (Eisenberger, Lieberman, & Williams, 2003). Experiencing pain and observing another's painful injuries activates the anterior cingulate cortex and anterior insula (Ochsner et al., 2008), and these empathic responses are correlated with the intensity of pain (Saarela et al., 2007; Singer et al., 2004). Pain catastrophizing (discussed below) is linked to abnormal brain processing of painful stimuli (Seminowicz & Davis, 2006), and there is an overlap of neural circuits that contribute to both pain and the regulation of anger (Bruehl, Burns, Chung, & Chont, 2009).

Studies have begun to examine emotional processes that attenuate the activity of the brain's pain circuits. For example, long-term practitioners of transcendental meditation showed 40–50% less activity in the thalamus and total brain in response to experimental pain than did matched controls (Orme-Johnson, Schneider, Son, Nidich, & Cho, 2006). Patients with irritable bowel syndrome treated with psychological therapy had not only reduced pain and anxiety but also reduced activity of the cingulate cortex and parahippocampal gyrus (Lackner et al., 2006). Overall, then, an increasing body of imaging studies illuminate the neurobiology that supports the role of psychological and social emotional factors in pain, to which we turn next.

Psychology, Emotions, and Pain

Research on the psychology of emotion and pain has proliferated on many fronts, and we propose the following framework to help organize these topics. Emotions, particularly negative emotions, stem from many sources including stressful life events and the experience of pain itself. We conceptualize four psychological processes, which, although overlapping and not linearly related, are particularly relevant for these emotions. They include emotional *awareness* (attention, differentiation, and labeling of emotion), *expression* (avoidance or suppression vs. expression of emotion), and *experiencing* (accessing, experiencing, and reflecting on one's emotions to enhance adaptation). In addition, emotions *modulate* the pain experience by influencing cognitions and behaviors. In this section, we

first present research on stress and pain, followed by a review of pain-related research on these four emotional processes.

Stressful Life Events, Trauma, and Pain

There is increasing evidence that psychological stress or trauma is associated with persistent pain, and likely predisposes to it. Reports of childhood adversities (e.g., divorce, family conflict, sexual abuse, physical abuse) and adulthood conflict and victimization are elevated in people with various pain conditions, including migraine headaches (Sumanen, Rantala, Sillanmäki,& Mattila, 2007), interstitial cystitis or painful bladder (Latthe, Mignini, Gray, Hills, & Khan, 2006), pelvic pain (Meltzer-Brody et al., 2007), irritable bowel syndrome (Mayer, Naliboff, Chang, & Coutinho, 2001), and fibromyalgia (Imbierowicz & Egle, 2003). However, these studies do not confirm that stress or psychological trauma causes or even predisposes to pain. Persistent pain can increase exposure to stressful events, such as job losses, marital disruption, and medical procedures and surgeries. Also, most studies of stressful life events suffer not only from the bias of retrospective recall but also of patient selection; treatment-seeking patients have higher stress levels than non-treatment seeking people with pain. One meta-analysis, however, found that childhood abuse and neglect reliably predicted pain in adulthood, and this relationship held when patients with pain were compared to both healthy controls and community non-patients with persistent pain, and even when non-patients with pain were compared with non-patients without pain (Davis, Luecken, & Zautra, 2005). A prospective study found a 4-fold increase in new onset fibromyalgia among workers exposed to workplace bullying, and a 2-fold increase among those with high work demand and low decision latitude (Kivimaki et al., 2004). Thus, it appears that stressors before pain can trigger or exacerbate pain.

Another approach to clarifying the direction of the stress-pain relationship has been to examine pain in PTSD. Persistent pain has been found to be quite common in people with PTSD, such as combat veterans and civilians in vehicle accidents (Asmundson, Coons, Taylor, & Katz, 2002). Many of these studies are problematic, however, in that they do not control for pre-existing pain or examine the time course of PTSD and pain. These limitations were addressed by a recent study of middle-aged adults who had experienced childhood abuse or neglect as documented by court records, and non-abused, matched controls. Having only current PTSD or childhood abuse/neglect alone conferred only a small increased risk for pain 30 years later, but the combination of both childhood abuse and current PTSD substantially increased the risk of later pain (Raphael & Widom, 2011). This suggests that a series of unresolved stressors over the life course may be most relevant to persistent pain.

Emotional Awareness and Pain

A fundamental emotional process involves being aware of, differentiating, and labeling one's feelings. Two areas of research on emotional awareness and pain are the correlates of alexithymia and the links among negative affect, positive affect, and pain.

Alexithymia, which literally means "no words for feelings," refers to a deficit in one's ability to identify feelings, differentiate among them, and label or describe them, along with a preference for externally-oriented thought rather than introspection. Alexithymia is elevated in a range of disorders, particularly in central sensitization conditions such as low back pain, fibromyalgia, and temporomandibular disorder (Ak, Sayar, & Yontem, 2004; Celikel & Saatcioglu, 2006; Sayar, Gulec, & Topbas, 2004). Also, alexithymia is often positively correlated with pain severity (Lumley et al., 2005; van Middendorp et al., 2008), not only when pain is reported retrospectively, but also prospectively using experience sampling (Glaros & Lumley, 2005).

What mechanisms link poor emotional awareness, differentiation, and labeling to pain? There is some evidence that alexithymia is associated with physiological hyperarousal, which can lead to pain-inducing changes such as prolonged muscle tension. Emotional awareness deficits also are related to somatosensory amplification—increased attention to and concern about one's body—which may prompt an increase in physical sensations, including pain. Clinical observations suggest that people with limited emotional awareness and verbalization ability may describe the physiological aspects of emotions in somatic terms, such as "my muscles are tight" or "my stomach hurts" (Lumley, Neely, & Burger, 2007). Lane et al. (2009) have proposed a neuroscience model of alexithymia that suggests that difficulty differentiating between emotions and physical sensations and processing emotions at the conscious level (i.e., alexithymia) may result in reports of pain that are suffused with emotion.

The construct of alexithymia has critics, however. Some view alexithymia as a pejorative term that denigrates people, including those from lower socioeconomic circumstances or other cultures, who do not communicate in the same, insight-oriented fashion as mental health professionals. Alexithymia typically is assessed by self-report, which raises questions of validity for people who are poor at introspection. Also, the most commonly used alexithymia scale is substantially correlated with negative affect, which might account for its relationship to pain.

Although most researchers view alexithymia as a risk factor for pain, the opposite direction may occur—the experience of stressors, including pain, may reduce the ability to identify and differentiate emotions. This is suggested by the dynamic model of affect (Davis, Zautra, & Smith, 2004). This model proposes a framework for understanding how pain influences the relationship between negative affect (NA) and positive affect (PA)—two constructs that are theoretically independent, but whose inter-correlation can vary from orthogonal or fully distinct ($r = 0$), to inversely related—opposite poles of a single dimension ($r = -1.0$). The model predicts that under conditions of low stress or threat—including low pain—people can differentiate affects, and PA will be relatively independent of NA. Yet, under elevated stress or pain, emotional complexity and differentiation are reduced, resulting in inversely correlated NA and PA. Zautra and colleagues have conducted prospective diary studies in people with rheumatoid arthritis, osteoarthritis, or fibromyalgia to understand relationships among NA, PA, and pain. They have found that when people experience increased pain, they are less able to distinguish PA from NA, and when they experience increased PA, their NA is less related to their pain (Strand et al., 2006; Zautra, Smith, Affleck & Tennen, 2001). Other researchers using daily diary methods also have found that increases in positive affect and decreases in negative affect predict pain reductions (Connelly et al. 2007; Paquet, Kergoat, & Dubé, 2005).

Theory and research on alexithymia and the dynamic model of affect suggest that the ability to differentiate and accurately label one's feelings is adaptive. These prospective studies suggest a causal pathway, but the findings are correlational nonetheless, and experimental testing is needed. Also, it will be interesting to see whether the model holds not only for the general dimensions of NA and PA, but also for specific emotions such anger, fear, sadness, and so on. Yet, researchers' interest in PA is a welcome shift from the usual focus on negative emotions, because PA is seen as a motivator to engage in reward seeking, growth, interpersonal connections, and creativity, which may lead to new perspectives on self-regulation and motivation among people with persistent pain (Frederickson, 2004; Hamilton, Karoly, & Kitzman, 2004).

Emotional Expression vs. Suppression and Pain

People vary in the degree that they verbally and non-verbally inhibit or suppress rather than express their emotions. Two examples of recent research on this process pertain to ambivalence over emotional expression and the suppression of anger.

The construct of ambivalence over emotional expression refers to the desire to express one's emotions, yet fear of the consequences of doing so. Several studies indicate that such ambivalence is associated with greater pain and maladjustment. Porter, Keefe, Lipkus, and Hurwitz (2005) found that patients with gastrointestinal cancer who were high in ambivalence reported higher pain behavior and poorer quality of life than patients low in ambivalence. Carson et al. (2007) found that greater ambivalence over emotional expression was related to higher evaluative and affective pain in patients with persistent low back pain. Other suppression-related constructs have also been examined. van Middendorp and colleagues (2010) found that anger inhibition predicted higher pain ratings at the end of the day, whereas anger expression predicted lower pain ratings, among women with fibromyalgia.

Such correlational studies do not clarify causality, unlike controlled experimental research in which emotional processes are manipulated and pain outcomes are assessed. An elegant set of experiments by Burns and colleagues has tested the role of anger suppression on pain. In these studies, anger is elicited in the lab, typically by the harassing actions of a confederate. Participants are randomized to either suppress their anger—they are instructed to refrain from verbal or nonverbal expression of anger during harassment—or to a control condition, in which there are no restrictions on anger expression. In studies of healthy young adults, anger suppression led to lower pain tolerance and higher pain ratings during the cold pressor test (Burns, Quartana, & Bruehl, 2007; Quartana, Yoon, & Burns, 2007). Among people with low back pain, anger suppression led to increased pain behavior during a functional task. Furthermore, anger suppression increased muscle activity in the area of the pain (the lower paraspinal muscles), but not muscles distant from the pain (the trapezii)—suggesting that anger suppression creates symptom-specific physiological changes that exacerbate pain. Furthermore, the effects of anger inhibition on the paraspinal muscles were most pronounced for high trait anger-out patients—those who typically express their anger, suggesting that anger suppression is particularly pain-inducing when it counters one's usual anger regulation tendency (Burns, Holly, et al., 2008; Burns, Quartana et al., 2008).

These experimental studies on anger suppression suggest strongly that the failure to adaptively express anger exacerbates pain. Yet, we do not know the relevance of these laboratory findings to the onset and course of pain during daily life. We also do not know whether reversing suppression by encouraging the expression of anger will reduce pain, and this is vital, because anger expression can have maladaptive interpersonal consequences, such as when anger that is expressed in a non-skilled manner alienates family members or health care providers who might otherwise provide help. Finally, it is interesting to consider how much the inhibition of emotions actually stems from negative reactions patients have received when expressing their emotions to health care providers who would rather focus on biomedical issues.

Emotional Experiencing and Pain

A third process is emotional experiencing, which we view as volitionally accessing, experiencing, and using one's emotions in an effort to promote better health and functioning. Various emotional experiencing interventions have been tested for their effects on pain, including mindfulness and acceptance therapies, emotional disclosure, and emotional exposure-based interventions.

There is growing interest in interventions that enhance one's awareness, acceptance, and mindfulness of internal experiences such as emotions, thoughts, and physical sensations. Mindfulness is one of the most widely studied emotional experiencing interventions and involves bringing non-judgmental awareness to thoughts, emotions, and sensations as they arise.

Training in mindfulness skills leads to greater tolerance of experimental pain than learning guided imagery (Kingston, Chadwick, Meron, & Skinner, 2007). Clinically, mindfulness is often taught in the mindfulness-based stress reduction (MBSR) program, but results of this intervention for chronic pain problems have been mixed. Among adults with low back pain, MBSR has been show to have benefits (Morone, Greco, & Weiner, 2007), as has a loving-kindness meditation program, a form of meditation used in the Buddhist tradition to develop love and transform anger into compassion (Carson et al. 2005). Results of studies of MBSR for fibromyalgia have been more negative, with an initial study suggesting improved quality of life, but not reduced pain, (Grossman, Tiefenthaler-Gilmer, Raysz, & Kesper, 2007), but a larger and better controlled study by the same research team found no benefits (Schmidt et al., 2011), as did another study of MBSR with fibromyalgia (Astin et al., 2003).

Mindfulness is a key component of Acceptance and Commitment Therapy (ACT), which has been tested in several recent studies. The benefits of ACT for adolescents with musculoskeletal pain has been shown in an uncontrolled study (Wicksell, Melin, & Olsson, 2007), and subsequently in a controlled trial, compared with multidisciplinary treatment (Wicksell, Melin, Lekander, & Olsson, 2009). The effectiveness of ACT has been suggested in two uncontrolled studies on adults with pain, which also found that increases in acceptance were correlated with improvement (McCracken, Vowles, & Eccleston, 2005; Vowles & McCracken, 2008). A randomized clinical trial found that ACT was more effective than a wait-list control condition among adults with chronic pain and whiplash-associated disorders (Wicksell, Ahlqvist, Bring, Melin, & Ollsson, 2008).

Research on interventions to enhance mindfulness is growing rapidly, and this technique holds promise for people with persistent pain, although more and better controlled studies are needed. It should be noted that most studies that report positive results find these for only a few outcome measures, and sometimes not for pain. Also, the mechanisms of action of mindfulness therapies are still being explored, and it is not clear whether such training improves emotional awareness as the mediator. ACT appears to be efficacious for chronic pain, but it is a complex intervention with many components including behavioral exercises and broader examination of values. Thus, the efficacy of individual components, including emotional experiencing, is not known.

A second experiential intervention is emotional disclosure, which involves privately writing or talking about stressful experiences and emotions for several sessions. Early studies were conducted on healthy people, but recent studies have been done on clinical populations, including people with persistent pain. Two controlled studies found that written emotional disclosure led to benefits in pain and other symptoms after several months for people with fibromyalgia (Broderick, Junghaenel, & Schwartz, 2005; Gillis, Lumley, Mosley-Williams, & Roehrs, 2006). Studies of emotional disclosure in rheumatoid arthritis, however, show less consistent benefits. Although Smyth, Stone, Hurewitz, and Kaell (1999) found that writing about stress led to better physician ratings of disease than control writing, other studies using written or verbal emotional disclosure have shown limited benefits or null results (Broderick, Stone, Smyth, & Kaell, 2004; Danoff-Burg, Agee, Romanoff, Kremer, & Strosberg, 2006; Keefe et al., 2008; Lumley et al., 2011; van Middendorp, Gennen, Sorbi, van Doornen, & Bijlsma, 2009; Wetherell et al., 2005). Written disclosure about stressful aspects of pelvic pain led to benefits on only one minor measure, but none of the primary

outcomes (Norman, Lumley, Dooley, & Diamond, 2004), and disclosure had no effect on migraine or tension headaches (D'Souza, Lumley, Kraft, & Dooley, 2008).

Meta-analyses indicate that the effects of emotional disclosure are small and probably depend on other factors (Frattaroli, 2006; Frisina, Borod, & Lepore, 2004). For example, disclosure is likely to be of most benefit to those with unresolved emotional stress. Thus, it may be more helpful for people with fibromyalgia than with RA because life stress is higher in fibromyalgia than RA (Walker et al., 1997). Individual differences in emotional processes may also be relevant. Norman et al. (2004) found that disclosure's benefits occurred for women with pelvic pain who were ambivalent about expressing their feelings, engaged in catastrophizing, and had higher baseline negative affect. Kraft, Lumley, D'Souza, and Dooley (2008) found benefits for people who reported a preference for emotional understanding and processing but low self-efficacy to manage migraine headaches. It should be noted, however, that research on moderating variables is relatively new and typically exploratory.

Unfortunately, researchers have usually recruited patients for disclosure studies based on the presence of pain and patients' willingness to be in research, not the presence of unresolved stress and patients' motivation to engage in emotional disclosure. Many patients do not participate in disclosure studies, and the disclosures of those who do participate are often less than optimal, lacking personal disclosure and processing of unresolved stressors and emotions (Lumley et al., 2011). Thus, one option may be to target emotional disclosure to patients who have unresolved stressful experiences and are motivated to engage in the sometimes challenging work of disclosure (Lumley, 2004). Research might explore whether one can increase the interest and engagement of more patients by using techniques such as motivational interviewing or at least providing a clearer rationale before emotional disclosure. Research might also explore ways to modify the instructions or provide guidance to assist people in disclosing, processing, and resolving emotional struggles. A more intensive emotional awareness intervention might prove useful. A recent randomized trial demonstrated that an affect awareness program involving intensive writing exercises, mindfulness exercises, and learning about links between stress and pain was highly beneficial for patients with fibromyalgia, compared with a wait-list control (Hsu et al., 2010).

Patients with persistent pain and histories of psychosocial trauma might benefit from clinician-provided emotional exposure and processing techniques, which have been shown efficacious for PTSD (Leserman, 2005). Although this has not been tested in controlled studies, Lumley et al. (2008) conducted an uncontrolled study of emotional exposure therapy for women with fibromyalgia and unresolved trauma (most commonly, childhood abuse). Therapy identified the stimuli and experiences that each patient avoided and used exposure-based techniques (e.g., written disclosure, imaginal desensitization, empty chair techniques, assertiveness training, in vivo exposure) to help patients confront and process avoided emotional experiences. There were small to moderate improvements on pain and disability, and moderate to large improvements in stress and emotional symptoms. These results suggest that a PTSD treatment model may be helpful for patients with persistent pain and psychological trauma, but larger, controlled studies are needed.

Emotional Modulation of Pain

Research also has examined how emotions may modulate the experience and duration of pain. In this section, we examine research on the valence-arousal interaction model, catastrophizing, pain-related anxiety, and fear of pain-related activity.

Emotional valence refers to the positive-negative quality of an emotion, or its pleasantness-unpleasantness. Arousal describes the intensity or activation of the emotion. Although it is commonly thought that negative emotions in general augment pain, and positive emotions inhibit pain, laboratory research indicates that an emotion's valence interacts with its arousal level to determine its pain effects. Only under relatively high arousal levels will an unpleasant emotional state exacerbate pain, or a pleasant emotional state inhibit pain; low arousal negative or positive emotions do not influence pain sensitivity (Rhudy, Bartley, & Williams, 2010). For example, Rhudy, Williams, McCabe, Russell, and Maynard (2008) found that experimental pain was augmented by arousing, unpleasant pictures of threatening scenes, and inhibited by arousing pleasant pictures of erotica, but that low arousal pictures such as food (positive) or grief (negative) did not modulate pain. These interesting findings with experimental pain need to be tested on clinical pain.

Pain catastrophizing refers to the tendency to ruminate upon pain sensations and feel helpless about pain, and its importance has been demonstrated in numerous studies (Quartana, Campbell, & Edwards, 2009). Pain catastrophizing is associated with greater pain and maladjustment in acute pain, such as surgery and childbirth (Pavlin, Sullivan, Freund, & Roesen, 2005; van den Bussche, Crombez, Eccleston, & Sullivan, 2007), as well as persistent pain conditions such as temporomandibular disorder, headache, rheumatic diseases, chronic prostatitis, and pelvic pain (Drahovzal, Stewart, & Sullivan, 2006; Edwards, Bingham, Bathon, & Haythornthwaite, 2006; Tripp et al., 2006). Evidence for its causal role comes from the demonstration that reductions in catastrophizing mediate the benefits of behavioral interventions for persistent pain (Smeets, Vlaeyen, Kester, & Knottnerus, 2006). Catastrophizing probably exerts its negative effects through several pathways, including the creation of an aroused, negative emotional state that exacerbates pain, the generation of helplessness that decreases adaptive pain responding, and the direct alteration of neural processes related to attention and responses to pain. People with trauma histories are more likely to catastrophize (Casey, Greenberg, Nicassio, Harpin, & Hubbard, 2008), suggesting that catastrophizing may mediate the effects of earlier psychological stress on pain.

Pain-related anxiety is broader than pain catastrophizing and is conceptualized as having four components: fearful thoughts about pain or its consequences, cognitive anxiety symptoms, somatic anxiety symptoms, and escape/avoidance from pain. Kinesiophobia, an excessive and irrational fear of movement and injury or re-injury, is one aspect of pain anxiety (Vlaeyen & Linton, 2000). Several studies have examined these constructs. Patients reporting high pain-related anxiety showed poorer physical performance on behavioral tasks such as reaching for objects (Thomas & France, 2007). Patients with acute low back pain who scored high a scale of kinesiophobia had elevated pain and physical disability (Swinkels-Meewisse, Roelofs, Verbeek, Oostendorp, & Vlaeyen, 2003), and baseline kinesiophobia scores were the strongest predictor of future functional disability—even stronger than baseline pain severity (Swinkels-Meewisse et al., 2006). Activating pain-related fear implicitly led to increased pain perception, suggesting that the validity of this construct is not dependent on self-report (Kirwilliam & Derbyshire, 2008). It should be noted, however, that patients rarely describe themselves as fearful or phobic, and there are debates about the factor structure of some of the scales used to measure these constructs.

It appears that people with elevated pain-related anxiety and fear avoid activities that may be important to recovering from acute pain (George, Fritz, & McNeil, 2006; Thomas & France, 2007). Such avoidance is associated with disuse and disability, and it increases the risk of persistent pain (Vlaeyen & Linton, 2000). Interestingly, these measures do not necessarily predict physical fitness or deconditioning, at least in back pain patients (Bousema, Verbunt, Seelen, Vlaeyen, & Knottnerus, 2007), and it is likely that the adverse behavioral outcomes

of high pain-related fear occur only for some people, such as those who also overpredict the consequences of movement (Huijnen, Verbunt, Peters, & Seelen, 2010). Pain-related fear and anxiety may also exert pain-inducing effects by other non-behavioral mechanisms, such as increasing somatosensory amplification.

Social Factors, Emotion, and Pain

Early models of persistent pain focused on the role that others play in reinforcing or punishing pain behaviors. Recent research has shifted from this operant model to a broader perspective that acknowledges emotional processes in social contexts (Cano & Williams, 2010). In this section, we examine pain research involving emotional communication between patients and their caregivers, the role of interpersonal empathy, and attachment and social rejection.

Emotional Communication Between Patients with Pain and Their Caregivers

Pain-related communication between patients and their caregivers, including family members such as spouses as well as health care providers, is complicated. Patients may view their pain as a simple sensory event, but their pain-related emotions can lead to behaviors that they might not realize are being communicated to others. Such interpersonal communication not only influences relationships but also may affect pain. The sociocommunications model of pain (Hadjistavropoulos & Craig, 2002) suggests the importance of attending to both the sender of information (i.e., the patient) and the receiver (i.e., caregivers). Although this model focuses on the communication of pain, it recognizes that emotion often is intermixed with the pain that is communicated.

Patients with persistent pain (information senders) may express their emotions in a variety of ways, including behaviors that reflect pain catastrophizing. The communal coping model of catastrophizing (Thorn, Ward, Sullivan, & Boothby, 2003) postulates that pain catastrophizing may lead to behaviors that communicate the patient's need for support in dealing with the both the pain and the emotional distress that accompanies pain. Sullivan and colleagues have shown that participants who exhibited greater catastrophizing during a cold pressor task also exhibited more pain behaviors (e.g., facial expressions of pain), especially when in the presence of another person (Sullivan, Adams, & Sullivan, 2004; Sullivan, Martel, Tripp, Savard, & Crombez, 2006). The effect of pain catastrophizing in increasing pain-related behaviors (i.e., facial expressions and verbalizations) has also been demonstrated in adolescents with persistent pain (Vervoort et al., 2009). Unfortunately, over the long term, catastrophizing may undermine patients' support needs. Researchers have found that catastrophizing is associated with losses of support over time in married patients (Buenaver, Edwards, & Haythornthwaite, 2007), and high catastrophizing patients may express their needs for support in aversive ways, which are then met with negativity from family members (Cano, Leong, Heller, & Lutz, 2009). Overall, the communal coping model helps to explain dyadic patterns of emotion and behavior in people with pain, but it remains somewhat speculative, and more research is needed on how catastrophizing affects a variety of behaviors that serve to communicate pain.

Information receivers or observers, including family caregivers, face many challenges with regard to pain communication. First, they may experience stress from seeing their loved ones suffer from pain, and these caregivers must differentiate their sense of the patient's pain from their own personal affective response to this distress (Goubert et al., 2005). Second, observers often have difficulty estimating pain in patients and may over- or underestimate it (Cano, Johansen, & Franz, 2005), leading to either unsupportive responses and distress (Martire et al., 2006) or psychological distress in the patients (Cremeans-Smith et al., 2003). However, observers' own negative thoughts and beliefs about patients' pain

influence how the observers evaluate and respond to this pain (Leonard & Cano, 2006). Several studies have shown that observers, including strangers and parents, are more accurate in estimating pain when they themselves report higher levels of catastrophizing (Goubert, Vervoort, Cano, & Crombez, 2009; Martel, Thibault, Roy, Catchlove, & Sullivan, 2008). Goubert, Vervoort, Sullivan, Verhoeven, and Crombez (2008) suggest that parents' catastrophizing may affect how threatening they perceive their children's pain to be, thus resulting in the higher pain ratings. It is possible that these higher pain estimations result in parental behaviors aimed at alleviating pain or distress in the patient.

Finally, despite their best intentions, caregivers may respond to pain communication in ways that patients perceive as unhelpful. When family members or health care providers are overprotective (overly solicitous), or overly critical and punishing, patients experience increased pain and distress, and report higher levels of physical disability (Romano, Jensen, Turner, Good, & Hops, 2000).

Fear that one's expressions of pain will upset or burden family members may lead to increased guilt or worry in patients as well as attempts to conceal pain from others (Druley, Stephens, Martire, Ennis, & Wojno, 2003). Yet, attempts to conceal pain are rarely entirely successful, because non-verbal expressions of pain are less subject to voluntary control than verbal expressions and tend to convey pain to others regardless of what patients say about their pain (Hadjistavropoulos & Craig, 2002). Also, patients may be concerned that their expressions of pain will lead to unhelpful responses from others (Druley et al., 2003). Patients may worry about whether their caregivers can be relied upon to provide attention and validation when patients are upset because of pain-related concerns (Reich, Olmsted, & van Puymbroeck, 2006).

In addition, research has shown that couples who lack confidence in their abilities to communicate about pain and who hold back from discussing pain and related concerns are likely to experience problems in adjustment. In a study of pain communication among patients with osteoarthritis and their partners (Porter, Keefe, Wellington, & Williams, 2008), patients with low self-efficacy for pain communication reported higher levels of pain, physical disability, and psychological distress. Also, patients who reported holding back from discussing pain and arthritis-related concerns reported higher psychological disability and pain ratings. Spouses who held back from disclosing their concerns reported higher caregiver strain and more negative affect. Thus, self-efficacy for communication and the tendency to hold back on talking about pain and related problems are factors that warrant exploration if we are to understand how dyads mutually manage emotion in people having persistent pain.

Empathy and Pain

There has been increased interest in pain empathy as a process through which caregivers and others attempt to understand and respond to the emotions of the person with pain. Green, Tripp, Sullivan, and Davidson (2009) found that observers with higher self-reported empathy rated individuals being administered experimental pain as experiencing much higher levels of pain. Empathy also has been manipulated experimentally. Loggia, Mogil, and Bushnell (2008) generated empathy by having healthy participants watch high or low empathy interviews with an actor, and then measured sensitivity to heat while the participants viewed the actor receiving similar stimulation. Participants in the high-empathy condition rated pain applied to themselves as more intense and unpleasant than did people in the low empathy group. Another study found that observers experienced empathic distress—as manifest in the tightening of muscles around the eyes, which is part of the prototypic facial pain expression—only when instructed to imagine themselves experiencing the procedure, but not when imagining the patient's feelings (Lamm, Porges, Cacioppo, &

Decety, 2008). There also is an animal model for the empathic experience of pain. Rodents both recognize and have their own emotional reactions to the pain of other rodents (Callahan, Gil, Levesque, & Mogil, 2008; Mogil, 2009). For example, Langford et al. (2006) found that mice exposed to cage-mates in pain also displayed pain behaviors themselves, but this reaction did not occur when exposed to non cage-mates in pain.

Empathy has been investigated in the context of marriages. For example, Gauthier, Thibault, and Sullivan (2008) conducted a study in which spouses estimated the pain of their partner from a videotape of the partner engaging in a task. Interestingly, spouse "empathic accuracy" (less discrepancy between spouse and patient pain ratings) was associated with greater patient pain, catastrophizing, fear of pain, and disability. Martire et al. (2006) used a similar approach but found that spouses with higher empathic accuracy responded less negatively and provided more emotional support that was more satisfying to patients. Spouses who were more accurate reported less stress from their caregiving. Empathic accuracy for pain estimations may be only one manifestation of empathy in relationships. Research suggests that empathic understanding may be just as important as responding to emotion distress in patient adjustment (Cano, Barterian, & Heller, 2008).

Empathy has also been explored in health care providers. Tait, Chibnall, Luebbert, and Sutter (2005) found that surgeons high in self-reported empathy were less likely than nonempathic surgeons to blame patients for failed back surgery. Using an experimental paradigm, Finset and colleagues have tested the effects of different forms of physician communication on patients with fibromyalgia. They found that a medical interview that including questions about psychosocial issues and empathic communication created negative affect and increases in a stress hormone (cortisol), but only among alexithymic patients (Finset, Graugaard, & Holgerson, 2006). However, in another study, this team found that alexithymic patients responded positively—with greater satisfaction—to empathic communication from the physician (Graugaard, Holgersen, & Finset, 2004). Linton, McCracken, and Vlaeyen (2008) reported that health care providers often express reassurance to patients about their condition, but this may reduce patients' worry only in the short-term, but not long-term. These authors advise providers to express empathy to improve the patient's engagement in treatment, but it remains unclear how to do so in a way that is reliably helpful.

Research on empathy in the context of pain is relatively recent. One of the limitations of this research is that the term "empathy" is variously used to explain the understanding of another's pain, the accurate estimate of another's pain, one's felt concern or sympathy, or the responses that are generated by these cognitive and emotional processes. Thus, it is important for researchers to define precisely what they mean by empathy, to ground their work in established models of empathy, and to use methods that are appropriate to their constructs and models.

Attachment, Social Rejection, and Pain

Another social or interpersonal construct that has been studied in relation to pain is attachment (Porter, Davis, & Keefe, 2007). A substantial body of research demonstrates that being insecurely attached to parents or providers, such as having a fearful, avoidant, or disorganized attachment, is a risk factor for maladaptive outcomes, and recent studies suggest that this includes pain. For example, children's reactions to separation from caregivers have been found to mirror their reactions to pain, suggesting a common diathesis underlying reactions to both separation and pain (Walsh, Symons, & McGrath, 2004). Meredith and colleagues have studied attachment in adults and proposed the attachment-diathesis model of chronic pain, which views pain as a stressor that triggers attachment-related cognitive, behavioral, and emotional processes, which subsequently influence pain

(Meredith, Ownsworth, & Strong, 2008). For example, being less securely attached predicted more catastrophizing in response to experimental pain (Meredith, Strong, & Feeney, 2006), whereas patients with persistent pain who were securely attached reported less pain and catastrophizing and viewed pain as a challenge rather than threat (Meredith, Strong, & Feeney, 2005).

Eisenberger and Lieberman (2004) have argued that the colloquial use of pain language to describe social estrangement, separation, or loss (e.g., “broken heart,” or something “hurts”) has a neural basis. That is, mammals’ social-attachment neural system has “borrowed” or is overlapping with the phylogenetically older neural pain system, which now serves the role of minimizing dangers associated with social separation as well as avoiding physically painful stimuli. These authors experimentally manipulated social rejection or exclusion using a virtual ball-tossing game, allegedly with other individuals who either included or excluded the participant. In a sample of healthy volunteers, the authors found that greater distress in response to induced social rejection was predicted by greater baseline sensitivity to pain, and rejection subsequently predicted greater reports of pain unpleasantness to applied heat pain (Eisenberger, Jarcho, Lieberman, & Naliboff, 2006). Such innovative research suggests that social estrangement increases the experience of both distress and pain.

We expect to see more research on the social-interpersonal context of pain, and the role played by emotions in that context. We also anticipate that more interpersonal treatment studies will be conducted, including work that might improve disclosure, communication, or empathy. For example, Porter, Keefe, Baucom, Hurwitz, and Moser (2009) found that a novel partner-assisted emotional disclosure protocol for patients with gastrointestinal cancer and their partners facilitated patients’ cancer-related disclosures to the partner. The intervention led to greater improvements in relationship quality and intimacy for couples that usually inhibited discussing cancer-related concerns, than did a randomly assigned control condition (a couple’s cancer education/support group). This type of intervention could be adapted for couples in which one person is experiencing persistent pain.

Limitations and Future Directions

Theoretical and empirical research on emotions and pain is rather new, and there are many limitations of the available literature and the conclusions that can be drawn from it. Here, we highlight limitations pertaining to interpretations of causality, individual and population differences in pain and emotion, and the assessment of stress and emotional processes. These limitations also point to many directions for future research.

Most studies noted in this review have detected only correlations or associations between emotions and pain, and the vast majority of these are cross-sectional. Such associations have multiple interpretations, and temporal and causal links remain unclear. Other than the research on anger suppression and on brain lesions or stimulation, few studies use well-controlled experimental designs in which the causal role of emotions can be specified. Even the randomized trials of mindfulness or emotional disclosure interventions typically involve multi-faceted interventions and relatively weak control conditions, hindering conclusions about the specific role of emotional processes. Much of the neuroscience research demonstrates convincingly that the brain links psychosocial processes and pain, but one should remember that the brain also is a dependent variable—responding to earlier experiences and learning. It will be of great interest to see how emotional experiences contribute to central sensitization, perhaps by studying people at high risk for both psychological trauma and pain (e.g., soldiers) both before and after the trauma occurs and pain develops. Research also should examine whether corrective emotional experiences—

such as exposure-based therapies—are able to reverse or eliminate such sensitization and pain.

Another limitation of the research is that the populations studied varied widely. Some studies were of healthy people experiencing experimental pain, and others were of patients with persistent pain. The meaning of pain, particularly its controllability and extent of tissue damage, is quite different in these populations, and one should be cautious about generalizing findings from one group to the other. Also, there is variation among the many conditions, syndromes, and diseases that generate persistent pain, and the degree to which emotional factors and central sensitization contribute to each may vary. Participants in the available studies also vary. Many of the studies were conducted with patients in specialty pain centers, and patients in these settings typically have long pain histories and multiple failures to respond to traditional treatments. They also report elevated life stress and emotional problems, which inflates associations between emotions and pain. More research is needed in people with pain who are seen in primary care settings or the community and who are likely to experience less life interference due to their pain and have fewer life stressors or emotional difficulties. Also, emotions play varying roles in the pain of any given patient, and it will be important to target emotional interventions only to those who need it. For example, only those patients with unresolved trauma, emotional inhibition, and sufficient motivation may benefit from an emotional expression intervention. Others may benefit more from behavioral techniques or psychotropic medications. Research should distinguish among patients and test whether matching treatments to patients optimizes outcomes.

Regarding assessment methods, the almost exclusive reliance on self-report measures of emotions and pain has limitations. Many self-report measures, including those used to assess ambivalence over emotional expression, anger expression or inhibition, alexithymia, pain catastrophizing, and pain anxiety and fear, tap not only the construct of interest but also the respondent's distress, psychopathology, or self-critical response style. This confounding inflates their associations with measures of pain. Fortunately, researchers are increasingly sensitive to this concern and may statistically covary negative affect when testing relationships between emotion and pain. When done with catastrophizing and alexithymia, the unique relationship between the emotion and pain measures is typically attenuated, but remains present. Newer constructs such as ambivalence over emotional awareness and pain-related anxiety also need to demonstrate their unique contribution to pain. Another concern is that measures of pain catastrophizing, fear, and anxiety may predict outcomes, in part, because they contain item content that overlaps with pain measures. When pain is assessed in ways other than retrospective self-reports, such as during daily life using experience sampling (Friedberg & Quick, 2007), or behaviorally, as in new disability claims (Mehling & Krause, 2007), relationships between emotion and pain are often attenuated or absent. Finally, it is likely that self-report measures of emotional states and processes lack validity for patients with limited introspection ability, such as those who are alexithymic repressed. Research should determine unique, non-confounded relationships by using a range of pain and emotion assessment methods.

The assessment of the emotional processes involved with stress is particularly challenging. Studies have focused on external or observable stressful events, but this approach fails to consider stress that stems from internal cognitive and affective conflicts. For example, a common stressor for people with persistent pain is the conflict between independence and dependence on others, which may manifest in a cycle of demonstrating their independence and engaging in excessive activity, which is then followed by increased pain and dependency on those around them for help (Cignac, 1998). There also can be conflicts between emotions with different action tendencies. For example, "stress" is often reported

when a person simultaneously experiences anger and fear, sexual desire and guilt, or attachment feelings and shame. We have observed that some people with persistent pain do not report external stressors or trauma, but are nonetheless troubled by such emotional conflicts. Such people often have difficulty recognizing or reporting these conflicts. Although it may prove challenging, methods to reliably and validly assess such important emotional processes need to be developed.

Summary and Clinical Implications

For several decades, research has shown that negative emotional states and emotion-focused pain coping are associated with greater pain. This has led to the widely-held view that negative emotions in people with persistent pain are maladaptive and need to be down-regulated or eliminated. Our review of recent research on emotions and pain only partially supports this view, however.

On the one hand, pain catastrophizing, pain anxiety, pain-related fear, social rejection, attachment insecurity, and high arousal negative emotions are related to greater pain and poorer adjustment, and these emotional factors occur not only in response to pain but also trigger, maintain, or exacerbate pain. Neuroscience research supports the view that negative emotions contribute to a pain-affect experience, or provide the emotional coloring to pain, particularly via the medial pain system and its projections to the anterior cingulate cortex, amygdala, and medial prefrontal cortex. Thus, clinical interventions should reduce these negative emotional states or pain modulators and increase positive emotions. On the other hand, our review also supports a seemingly contradictory perspective—that the *lack of* awareness, expression, and experiencing of negative emotions is associated with—and likely contributes to—greater pain and dysfunction. This alternative view is buttressed by a functional or adaptive model of emotion, which posits that people should be aware of, informed by, and motivated to adaptive action by their emotions.

How can we reconcile these two different perspectives? Should patients with persistent pain attempt to avoid, reduce, or minimize the experience of negative emotions such as fear, sadness, and anger? Or should they attempt to elicit, experience, and be guided by them? Contemporary emotion theory provides some guidance by distinguishing primary and secondary emotions and delineating how emotional processes contribute to emotional states (Greenberg & Paivio, 1997). Primary adaptive emotions are fundamental to our evolutionary development, are elicited by prototypic situations and motivate prototypic behavior, and have survival value. For example, primary anger is elicited when something of value is taken or threatened to be taken and motivates either defense or attack. Emotional difficulties arise when people do not recognize, understand, and express these primary emotions, but instead ignore or suppress them, which typically results from socialization (e.g., cultural rules, gender roles, punitive social environments). The suppression of primary anger appears to be a common occurrence, and research suggests that this contributes to pain, although primary sadness, fear, or even joy is ignored or suppressed by some people or at some times. As a result of the lack of awareness, expression, and processing, people commonly experience “secondary emotions” such as depression, anxiety, guilt, and irritability, which do not have evolutionarily-based adaptive value, but are frequently reported by people with persistent pain. Also, maladaptive emotional associations or learning also occur, particularly the association of fear with persistent rather than acute pain. Primary fear in response to acute pain is adaptive, motivating escape from the potentially dangerous source of the pain; however, fear of persistent pain is typically maladaptive because there is no longer is a genuine threat to the body (although this is not fully true for some conditions, such as rheumatoid arthritis or sickle cell disease).

This contemporary model of emotion helps to reconcile the apparent paradox of the role and value of emotion for people with persistent pain. Emotional awareness helps to distinguish those secondary emotions that should be reduced from those primary emotions that should be elicited and used to motivate and direct action. Experiencing and expressing secondary emotions is not fundamentally helpful and may simply exacerbate pain, and techniques to reduce such emotions are indicated. In contrast, one should be aware of those emotions related to primary, biologically based situations in patients' lives, such as violations (anger), loss (sadness), true threats (fear), and even accomplishments or victories (joy), and they should be accessed and experienced for their informative and motivational properties. Notably, relief is often experienced when awareness occurs and these primary emotions are accessed and expressed, and this relief may be accompanied by a reduction in pain. Mindfulness interventions, written or verbal disclosure exercises, and perhaps emotional exposure and processing techniques borrowed from experiential therapy or the treatment of PTSD may be helpful in this regard. Techniques to encourage behavioral or activity "exposure," including exercise, among people with persistent pain, despite their fear or anxiety regarding the pain or its consequences, also appears to be of value (Boersma et al., 2004; de Jong et al., 2005).

Overall, the conceptualization, assessment, and treatment of persistent pain should include a sophisticated understanding of emotional states and emotional processes. The burgeoning neuroscience research indicates that pain pathways in the CNS are tightly linked with and influenced by emotions, and such pathways are sensitized both by early painful or traumatic experiences (Goldenberg, 2010) as well as later painful stimulation from peripheral tissues. Psychological research indicates that emotional awareness, expression, and experiencing as well as pain-related emotion modulation play keys roles in the pain experience. Social research indicates that interpersonal factors contribute to the modulation of negative emotions through processes such as interpersonal disclosure and empathy, and these processes also influence pain. These converging lines of research should arm clinicians to educate patients about the important ways that emotions, stemming from relationships and experiences over one's life and modulated by psychological factors, both influence and are influenced by neural processes that shape the experience of pain.

Clinicians might be guided by a growing number of models that attempt to explain persistent pain by integrating variables such as life stress, neurobiology, cognitions, emotions, and behavior. These models eschew simple cause-effect thinking in favor of more complex interactive or recursive processes. For example, Sharp and Harvey's (2001) mutual maintenance model posits that emotional stress and various factors (e.g., somatic attentional bias, anxiety sensitivity, emotional avoidance, and limited cognitive reserve) exacerbate distress and disability, thereby maintaining or prolonging initial pain. Asmundson and Hadjistavropolous (2006) proposed a model of shared vulnerability, in which individual differences (e.g., genetic factors, personality) predispose people to anxiety sensitivity, making them more likely to respond to physical injury with intense emotional reactions, such as fear. Such emotional reactions may result in pain-related avoidance and disability following injury. Van Houdenhove, Egle, and Luyten (2005) proposed that "stress intolerance and pain hypersensitivity syndromes" result from a chronically overburdened stress response system that shifts from hyper to hypoactive, causing reduced effort tolerance, altered inflammatory activity, and increased sensitization. Finally, McLean, Clauw, Abelson, and Liberzon (2005) proposed that the pain that develops after injuries such as whiplash involves interactions among such factors as past experience, acute stress responses to trauma, post-injury behavior, and cognitive/psychosocial processes. These interactions, in turn, alter activitywithin brain regions that process pain.

At a minimum, we encourage clinicians working with patients who have persistent pain to at least inquire about—if not explore at length—a number of issues. These include patients' emotional development including stressful life events, accidents, injuries, and abuse in both childhood and adulthood; currently experienced emotions; how significant others respond to the patient's pain and emotions; and how much patients experience or avoid various emotions, actions, and relationships. Clinicians and patients should work jointly to determine how these factors link to the onset, exacerbation, and attenuation of the current pain problem. Clinicians should state clearly that an examination of emotional states and processes does not imply the pain is in any way not real, or that it represents a moral or psychological failure of the patient. Rather, they should stress the reality and legitimacy of the pain and emphasize that emotions and emotional processes, along with beliefs and actions, are vital parts of human pain experience.

In conclusion, we welcome the past decade's increased emphasis on emotional states and processes as major factors in the pain experience. Ongoing theoretical development and empirical study of biological, psychological, and social aspects of emotion hold the promise to improve not only our understanding of pain, but to shift the zeitgeist to recognizing the potential value of emotional processes and primary emotions, which we believe will result in a broader range of assessment and intervention approaches to help the millions of people who suffer with pain. We anticipate further development of a true biopsychosocial model, in which the three domains are more tightly integrated than we have presented them. We also look forward to the development of a comprehensive cognitive-affective-behavioral model, which recognizes that, although these three domains might be discussed separately, they are strongly linked and reciprocally influence each other.

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