Amending the Fear-Avoidance Model of Chronic Pain: What Is the Role of Physiological Arousal?

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This paper outlines an amendment to current fear-avoidance models of chronic pain. These models propose that fear and avoidance behavior contribute significantly to the development and maintenance of chronic pain and related functional limitations. Fear is typically expressed in three response domains, including cognitive, behavioral, and physiological. To date, however, fear-avoidance models of chronic pain have focused primarily on the role of the cognitive and behavioral responses to the relative exclusion of the physiological response domain. This paper describes how states of physiological arousal resulting from autonomic nervous system activation (e.g., increased heart rate, increased blood flow to voluntary muscles, decreased blood flow to the gastrointestinal tract and to the skin, decreased motility, decreased cerebral blood flow) may influence fear of pain and avoidance behavior and thereby contribute to the maintenance of chronic pain. Clinical implications and future research directions are discussed.

A significant increase in published research examining psychological factors implicated in chronic pain syndromes has been observed over the past 10 years (Norton, Asmundson, Norton, & Craig, 1999). Among the promising lines of psychological investigation are those examining the roles of pain-related anxiety, pain-related fear, and avoidance behavior as they relate to chronic musculoskeletal pain (for recent reviews, see Asmundson, Norton, & Norton, 1999; Vlaeyen & Linton, 2000). The fear-avoidance models have their roots in early observations of significant anxiety in the pathology of pain (e.g., Paulett, 1947; Rowbotham, 1946) as well as operant conditioning theory (Foydyc, 1976).

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Fear-Avoidance Models

The recent popularity of fear-avoidance models of chronic pain has led to an improved understanding of the development and maintenance of chronic pain conditions. In particular, these models have tendered a theoretical background for numerous empirical investigations of chronic musculoskeletal pain. Over the years, several models have been proposed. These include the Fear Avoidance Model of Exaggerated Pain Perception (Lethem, Slade, Troup, & Bentley, 1983), a model of fear of movement/reinjury (Vlaeyen, Kole-Snijders, Boeren, & van Eek, 1995), and cognitive models of chronic pain proposed by Philips (1987) and Sharp (2001). Although there are subtle differences among these models, they share the basic premise that fearful reactions to the anticipation of pain, the consequences of pain (e.g., injury), or pain itself promote escape from or avoidance of potentially pain-producing activities.

A true appreciation of the impact of fear and anxiety on chronic pain requires a distinction between these two constructs. Fear, which is the primary focus of these models, is an emotional state characterized by autonomic nervous system activation, including physiological changes characteristic of increased sympathetic outflow and parasympathetic withdrawal, to prepare the organism for action. Fear is typically elicited in response to an actual or perceived threat, is relatively immediate in onset, and motivates escape or active avoidance behavior. Anxiety, on the other hand, is a more gradual building mood state, involves more cognitive processing, less autonomic arousal or even autonomic suppression, and passive avoidance behavior (for comprehensive reviews, see Barlow, 2002, and Craske, 1999). Although these two constructs are distinct, they are very much intertwined in that heightened anxiety may increase the likelihood of a fearful response to a perceived danger, while fearful experiences may promote anxiety regarding similar future experiences.

Given the burgeoning popularity of the fear-avoidance models in applied and research settings, Vlaeyen and Linton (2000) composed a state-of-the-art review of the fear-avoidance literature, which culminates in a comprehensive fear-avoidance model of chronic musculoskeletal pain (see nonboldface text in Figure 1). This model can be summarized as follows. Following a pain-provoking injury, one of two competing responses may occur. The pain experience may be appraised as nonthreatening. In this case, pain is dealt with in an adaptive manner (i.e., initial rest of the affected area followed by gradual increases in activity) that allows the person to proceed toward recovery. On the other hand, the pain may be appraised as threatening (i.e., the person may view the pain as dangerous and may catastrophize about its harmful consequences). In this case, the person may become mired in a vicious cycle that promotes increased fear of pain, avoidance of activities that are expected to produce pain, hypervigilance for pain, and, as a consequence, disuse, depression, and disability.
The aforementioned fear-avoidance models appear to hold great utility in understanding the development and maintenance of chronic pain. Indeed, the mounting empirical support for fear-avoidance models is impressive. To illustrate, Asmundson and Taylor (1996), employing structural modeling techniques, revealed support for a model wherein fear of pain was a direct positive causal influence on self-reported escape/avoidance behavior in a sample of chronic musculoskeletal pain patients. Others (e.g., Crombez, Vlaeyen, Heuts, & Lysens, 1999; McCracken, Zayfert, & Gross, 1992; Waddell, Newton, Henderson, Sommerville, & Main, 1993) have found converging evidence demonstrating that pain-related fears are more strongly related to functional disability in chronic pain patients than are indices of pain severity. Further, Vlaeyen et al. (1995) noted that fear of movement/reinjury (i.e., kinesiophobia) was significantly related to avoidance as assessed using a controlled pain-inducing behavioral task. Indeed, in reviewing the pertinent literature, Vlaeyen and Linton (2000) concluded that “pain-related fear and avoidance appears to be an essential feature of the development of a chronic problem for a substantial number of patients with musculoskeletal pain” (p. 317).

In a similar yet cognitively focused model, Sharp (2001) emphasized the impact of pain-related anxiety on beliefs and appraisals of pain rather than on
disuse. Both models posit that negative beliefs about pain, or pain catastrophizing, promote an affective response and, therefore, cognitive, behavioral, and physiological responses. In Sharp’s model, however, the cognitive and physiological responses are seen as confirming and strengthening an individual’s negative beliefs about pain. In addition, pain-related avoidance behavior or other safety behaviors prevent the individual from acquiring schema-incongruent information about the meaning of pain.

Although the Vlaeyen and Linton (2000) and Sharp (2001) models differ in the specific variables that are emphasized in perpetuating chronicity, the two models are not mutually exclusive. Indeed, there is support for both the disuse formulation (e.g., Wagenmakers, Coakley, & Edwards, 1988) and the cognitive formulation (e.g., Crombez, Vervaet, Lysens, Baejens, & Eelen, 1998). Accordingly, our amended model (presented below) assumes that both mechanisms of action may be simultaneously involved.

Parallels Between Models of Fear and Chronic Pain

As noted in previous works (Asmundson, Norton, & Norton, 1999; Rachman & Arntz, 1991), parallels can be drawn between the fear-avoidance models of chronic pain and models of chronic fear and anxiety. Both describe interplay between fear, anxiety, and escape/avoidance behavior, wherein such behavior serves as a strategy employed by the person to cope with the feared stimuli. In panic, for example, avoidance of certain situations (e.g., driving alone) occurs in an attempt to reduce the probability of experiencing paroxysmal somatic sensations (e.g., palpitations, dyspnea) or thoughts (e.g., losing control) that are believed to be potentially harmful. Similarly, in the person with chronic pain, avoidance of situations thought to be potentially pain provoking serves to reduce the anxiety associated with the expectation of experiencing pain. Both models also hold that avoidance is a maladaptive strategy that offers only transient and situational relief from feared stimuli and, of particular importance, creates functional difficulties (e.g., social withdrawal, occupational limitations).

Compelling evidence exists to suggest that the fear associated with anxiety disorders, chronic pain, and other chronic health conditions (e.g., asthma, vertigo) may stem from anxiety sensitivity (see Asmundson, Norton, & Norton, 1999; Asmundson, Wright, & Hadjistavropoulos, 2000). Anxiety sensitivity is a predispositional tendency to respond with fear to sensations that one associates with anxiety (Reiss, 1991; Reiss & McNally, 1985). Elevated anxiety sensitivity has been frequently noted across the anxiety disorders (see Taylor, Koch, Woody, & McLean, 1996). Anxiety sensitivity also appears to be related to fear of pain in patients with chronic back pain and headache (Asmundson & Norton, 1995; Asmundson, Norton, & Veloso, 1999). As well, anxiety sensitivity may influence the expression of various other health conditions (for a recent review, see Asmundson et al., 2000).

Thus, models of general and pain-specific fear are similar in many ways. Both postulate chronic conditions that are maintained by the reinforcing
effects of anxiety reduction that results from avoidance of feared stimuli. It appears that anxiety sensitivity, or the predisposition to respond with fear to sensations of anxiety, may play a key underlying role in these conditions. Some (e.g., Taylor, 1995; Taylor et al., 1996) have posited that anxiety sensitivity, in fact, embodies three dimensions: (a) fear of somatic sensations, (b) fear of cognitive dyscontrol, and (c) fear of publicly observable symptoms. Not only is there extensive empirical support for the existence of these factors, but also the factors map closely to Lang’s (1968) three-response (physiological, cognitive, and behavioral, respectively) model of anxiety expression.

Three-Response Model of Fear

Fear is typically conceptualized as a three-response system, comprising cognitive, behavioral, and physiological components (Lang, 1968). The cognitive component reflects the beliefs, perceptions, and attentional processes involved in the anxious state. The behavioral component reflects motivation mechanisms designed to promote avoidance of, or escape from, the perceived or anticipated threat. The physiological component reflects autonomic nervous system responses (i.e., circulatory, neuromuscular, and somatic changes) that prepare an organism to protect against the perceived or anticipated threat. Although all three components appear to be involved in the experience of fear, the intensity of each component appears to vary across individuals and situations. Indeed, even within each component, a great deal of variability exists (see Wilhelm & Roth, 2001). Some individuals, for example, may tend to respond physiologically with increased heart rate but minimal increases in muscular tension, while others may respond in a different or the opposite manner, depending both on individual differences as well as situational differences (Myrtek, 1984).

The three-response model of fear has helped advance our understanding of pathological fear, and many current empirically supported treatment protocols for phobias and other anxiety disorders specifically target all three components (e.g., Craske, Barlow, & Meadows, 2000). In the anxiety disorders, these three components are seen as mutually reinforcing and exacerbating (Beck, Emery, & Greenberg, 1985). In panic disorder, for example, somatic sensations are perceived and misinterpreted as potentially dangerous. This process often leads to a state of intense anxiety, which culminates in panic. The intense anxiety introduces a series of cognitive, physiological, and behavioral symptoms. These can include the sensations that initially provoked the anxiety as well as additional sensations that may be catastrophically misinterpreted. As such, the symptoms perpetuate and exacerbate the level of anxiety, again promoting stronger cognitive, physiological, and behavioral responses. This cyclical relationship can quickly intensify, with anxiety magnifying symptoms and the symptoms causing greater anxiety, quickly spiraling into a panic attack.
To illustrate, consider the case of a man who walks home from a morning visit with friends at a local coffeehouse. During the walk home, he notices that his heart is beating rapidly. Although the rapid beating of his heart could be the result of caffeine or the brisk walk home, the man becomes fearful that he may be experiencing a heart attack. As he becomes anxious about this possibility, autonomic nervous system activation produces several physiological changes, including an elevated heart rate, shortness of breath, and tingling sensations in his hands and fingers. The amplification of his heart rate combined with the other symptoms strengthens his belief that he is experiencing a heart attack, leading to greater anxiety and continued intensification of the physiological symptoms. Because he is alone, he runs toward a telephone to call for help, further increasing his heart rate and respiration.

As is evident in this example, physiological sensations were interpreted as a threat to physical safety, producing fear. The anxiety resulted in physiological symptoms and a behavioral response that further compounded the catastrophic misinterpretation, served to exacerbate the anxiety symptoms, and quickly escalated to panic. Other anxiety disorders show similar self-reinforcing effects. In social anxiety, for example, individuals may become anxious due to fears that they will be negatively evaluated because of their (perceived) inability to adequately perform in social situations. The anxious state may produce physiological and cognitive symptoms that either interfere with performance (e.g., a “lump” in the throat interfering with a speech) or are believed to be publicly noticeable and embarrassing (e.g., excessive perspiration). These symptoms can then amplify the social fears as they may seemingly provide the individual with evidence of social incompetence (Hope, Heimberg, Turk, & Juster, 2000; McEwan & Devins, 1983).

The three-response model has provided a solid framework from which understandings of the nature of anxiety disorders have grown. Furthermore, Lang’s (1968) model has had an impact on the understanding of fear and avoidance in chronic pain. Indeed, Vlaeyen and Linton (2000), Sharp (2001), and others (e.g., McCracken, Zayfert, & Gross, 1993) draw reference to each of the behavioral, cognitive, and physiological components of anxiety in their discussions of fear and avoidance in chronic pain.

**Three-Response System in Fear of Pain**

**Behavioral Component of Pain-Related Fear**

The behavioral component of anxiety, particularly escape and avoidance behavior, has been the subject of considerable empirical investigation. Indicators of fear-related behaviors, such as submaximal performance and guarded movements during physical tasks, have been shown to significantly relate to self-report measures of pain-related fear (e.g., Crombez et al., 1998; Crombez, Vlaeyen, et al., 1999; McCracken et al., 1992; Vlaeyen et al., 1995; Watson, Booker, & Main, 1997). When this avoidance behavior persists beyond the normal healing time, it may become maladaptive and lead to physical
deconditioning characterized by loss of ligamentous flexibility, muscular atrophy, and degeneration (Bortz, 1984; Fordyce, 1976). Consequently, the deconditioned anatomical structures (e.g., joints, musculature) become less capable of initiating or sustaining mechanical or supportive actions and, therefore, activities involving these structures are more likely to produce pain. Despite these negative consequences, avoidance behavior becomes reinforced by the short-term reduction of pain-related fear (Fordyce, 1976; Fordyce, Shelton, & Dundore, 1982; McCracken, Zayfert, et al., 1993).

**Cognitive Component of Pain-Related Fear**

Similarly, the cognitive component of pain-related fear has been the subject of much empirical investigation (for a review, see Sharp, 2001). Several studies have found strong relationships between fear of pain and catastrophic interpretations or negative appraisals of painful or potentially painful stimuli (e.g., McCracken, Gross, Sorg, & Edmands, 1993; Vlaeyen et al., 1995) as well as initial overprediction of pain intensity during physical tasks (Crombez, Vervaeke, Lysens, Eelen, & Baeyens, 1996; McCracken, Gross, et al., 1993). Furthermore, McCracken (1997) and Crombez, Eccleston, Baeyens, van Houdenhove, and van den Broeck (1999) both provide evidence that elevated fear of pain is related to attentional bias to pain. Specifically, McCracken noted a significant positive relationship between fear of pain and a self-report measure of attention to pain, the Pain Vigilance and Awareness Questionnaire (McCracken, 1997). Crombez, Eccleston, et al. (1999), employing a numerical interference task, found that attentional interference due to pain was best predicted by both self-reported pain-related fear and measures of pain severity. Recent converging evidence from modified Stroop testing confirms these findings (Snider, Asmundson, & Wiese, 2000).

**Physiological Component of Pain-Related Fear**

Despite the detailed investigation of the behavioral and cognitive components of pain-related fear, the physiological component has received only scant attention. Although Vlaeyen and Linton (2000) and Sharp (2001) note preliminary evidence supporting a role for the physiological component, physiological variables receive limited attention in their models of chronic pain.

Fear, regardless of the triggering stimuli, can produce physiological arousal through the autonomic nervous system. This activation is characterized by symptoms including, but not limited to, accelerated heart rate, elevated blood pressure, increased respiration, gastrointestinal activity, increased muscular tension, and increased circulation to skeletal muscles as well as dermal and cerebral vasoconstriction (Guyton & Hall, 1996; Hoehn-Saric & McLeod, 1993). These symptoms, if prolonged, stress the body and may have direct bearing on the physiological processes and anatomical structures implicated in specific chronic pain syndromes. Some studies (Aritz, Merckelbach, Peters, & Schmidt, 1991; Flor, Birbaumer, Schuep, & Lutzenberger, 1992), but not all (Collins, Cohen, Nowiboff, & Schandler, 1982; Flor, Turk, &
Birbaumer, 1985), have shown lower heart rate reactivity in patients with chronic pain compared to healthy controls when exposed to challenge procedures that induce stress. The mixed results may be a product of method variance, with reactivity evidenced only under more prolonged or intense stress induction. Notwithstanding, the evidence of lower heart rate reactivity in chronic pain patients suggests an absence in sympathetic outflow and, perhaps, parasympathetic activation, whereas the pattern in healthy controls suggests the expected sympathetic activation and parasympathetic withdrawal.

There is also evidence to suggest that arousal-induced muscular tension plays a significant role in chronic musculoskeletal pain (Merskey, 1980, 1993; Turk, 1996a, 1996b). Although the exact method by which muscular tension exacerbates musculoskeletal pain has not been elicited, hypothesized mechanisms include inefficient or contraindicated biomechanics during movement (Watson et al., 1997), direct aggravation of deconditioned or damaged tissues, or muscular fatigue as a result of protracted contraction. Additionally, elevated blood flow to skeletal muscles may affect the muscular oxygen supply and lipid use during contraction, thereby accelerating muscular fatigue. Flor et al. (1985, 1992), using anxiety or personally relevant stress-induction techniques with healthy controls and individuals with chronic pain conditions (including low back pain, temporomandibular pain, and tension-type headache), found significantly increased activity in the musculature specific to the person’s pain complaints when compared to healthy controls. Elevated muscular activity was not noted at sites distal to the primary pain location (Flor et al., 1992).

Despite the evidence implicating physiological reactivity in response to pain-related fear activation, Merskey (1980, 1993) and Turk (1996a, 1996b) suggest that there is insufficient empirical and clinical evidence to firmly establish stress- or anxiety-induced arousal as causes of chronic pain. Indeed, physiological arousal alone does not appear to be the primary pain-producing agent. However, we propose here that the physiological component of pain-related fear plays an interactive role in perpetuating the cycle of chronic pain and disability.

The Amended Fear-Avoidance Model

Although the exact mechanism through which compromised autonomic nervous system function and muscular tension exacerbate musculoskeletal pain has not been elucidated, hypothesized mechanisms include autonomic nervous system dysregulation, increased blood flow to skeletal muscles, inefficient or contraindicated biomechanics during movement, direct aggravation of deconditioned or damaged tissues, or muscular fatigue as a result of protracted contraction. In the context of fear-avoidance models it may be the contributions of the physiological effects of autonomic dysregulation and muscular tension to the overall condition of anxious anticipation that is of particular significance. While others (Sharp, 2001; Vlaeyen & Linton, 2000) allude to the role of physiological arousal in fear avoidance, none give it more than passing mention.
Based on the contemporary models, as well as scant but generally supportive preliminary empirical evidence, we propose an amendment to the fear-avoidance model that describes the role of physiological arousal in chronic pain and related disability. Specifically, we propose that the physiological component interacts with the cognitive and behavioral components in a positive symptom feedback loop, influencing anxious apprehension (i.e., continued appraisal of pain as negative, perceived stress), catastrophizing, inability to cope effectively, and dysfunctional mood and performance. The amendments to the model are presented in boldface type in Figure 1. Associated predictions are outlined below.

1. Physiological symptoms, particularly increased heart rate, increased muscular tension, and increased blood flow to voluntary muscles, influence avoidance behavior by aggravating damaged or weakened tissues and producing pain. These pain sensations confirm expectations that movement will produce pain. This, in turn, directly reinforces the pain-related fears and the beliefs that activities or actions will be painful. Accordingly, as fears and negative expectations are validated, the adoption of avoidance behavior is strengthened.

2. Physiological arousal interacts with the cognitive component of the pain-related fear. While physiological arousal could potentially produce pain, as discussed above, the symptoms of this arousal may be more likely to produce bodily sensations such as muscular tension that elicit misinterpretations of the nature and meaning of the sensations. These misinterpretations are believed to be the product of individual differences in the propensity to respond with fear to sensations that are anxiety provoking (e.g., anxiety sensitivity, negative affect; Asmundson, Norton, & Norton, 1999). Indeed, the individual who is hypervigilant for pain may interpret the bodily sensations as evidence of pain (see Al Absi & Rokke, 1991; Nibbett & Schachter, 1966; Weisenberg, Aviram, Wolf, & Raphaeli, 1984). Likewise, Amdudson and Taylor (1996) suggest that the physiological sensations of anxiety, provoked by anticipation of, or exposure to, a pain-provoking situation, are catastrophically misinterpreted as evidence of impending harm by those with high anxiety sensitivity. In short, physiological responses may lead directly to pain and subsequent avoidance, or they may be catastrophically misinterpreted and, as a consequence, associated situations avoided.

To illustrate, consider a woman rehabilitating from a low back injury. During a therapy session she begins to lift an object from the ground. Anticipating that the movement will produce pain, she becomes anxious. In becoming anxious, a state of arousal is produced, including a general increase in muscular tension in the lumbar region. The woman's attention is directed toward the lumbar region and the sensations are perceived as evidence of pain. The perceived pain amplifies the pain-related fear, which, in turn, promotes
continued hypervigilance and physiological symptoms. Consequently, the woman discontinues the behavior, leading to a reduction in pain-related fear and associated physiological sensations. Ultimately, the avoidance behavior is reinforced as a result of the reduction in the negative stimuli and confirms the negative expectations of a painful outcome from the activity.

Therefore, induction of an anxious state, whether by sensation or anticipation of pain, can produce cognitive and behavioral responses, as well as a physiological response characterized by activation of symptom-specific muscular activity, increased heart rate, increased blood flow to voluntary muscles, decreased blood flow to the gastrointestinal tract and to the skin, and decreased cerebral blood flow. This physiological component of pain-related fear interacts with the aforementioned cognitive and behavioral responses, strengthening the associated fear. The physiological arousal may then perpetuate the cycle of chronicity by triggering the experience of pain (Proposition 1 above; pathway from Physiological Arousal to Pain Experience), strengthening negative beliefs about the nature and meaning of pain (Proposition 2 above; pathway from Physiological Arousal to Pain Catastrophizing), or both.

Finally, although somewhat beyond the focus of this paper, there is evidence that fear-related cognitions and avoidance behavior could also directly influence pain catastrophizing (see Sharp, 2001) and, possibly, pain experience. Given the focus here on physiological arousal, these potential pathways have been withheld from the amended model for the sake of clarity.

Implications for Assessment and Treatment

The amended fear-avoidance model holds several potential implications for assessment and treatment, depending on whether one or both of the main postulates of this addition to the model are upheld. If physiological arousal leads directly to muscular pain, and initiates the cycle, then assessment of physiological arousal patterns and treatments that assist the patient in reducing physiological arousal (e.g., relaxation techniques, active coping strategies) may be most effective. If physiological arousal induced by exposure to a potential pain-provoking situation leads to catastrophic misinterpretation of those sensations (i.e., the fear is of the anxiety), then intervention effective for clinical anxiety may be best. These interventions would, of course, be preceded by an assessment focused on delineating the specific degree and nature of the anxiety (e.g., anxiety sensitivity; pain-related fear, kinesiophobia; Asmundson, Norton, & Norton, 1999; Vlaeyen & Linton, 2000). These assessments also are of potential use in the evaluation of treatment outcome. Indeed, reductions in pain-related fear have been obtained in studies of such treatment programs (e.g., Vlaeyen, de Jong, Geilen, Heuts, & Van Breukelen, 2001). It is likely that both scenarios hold at different times in different individuals. However, in addressing the physiological symptoms of anxiety that may be interacting with cognitive and behavioral components in a positive symptom feedback loop, additional targets for intervention are
presented. Specifically, interoceptive exposure techniques, which serve to break fearful reactions to symptoms in panic disorder (see Craske et al., 2000), could help break the component interactions proposed here. Clearly, however, this area requires continued investigation.

Conclusions and Future Directions

Obviously, this amendment to the fear-avoidance models remains speculative and awaits empirical investigation. Although tools exist to quantify the physiological components of pain-related fear (i.e., Pain Anxiety Symptoms Scale; McCracken, Zayfert, et al., 1993; Fear of Pain Questionnaire–III; McNeil & Rainwater, 1998), it appears that the physiological component of fear of pain has been largely overlooked in theoretical and empirical investigations of chronic pain. To catalyze investigations in this area, three initial lines of exploration are proposed.

1. Research must confirm that increases in state pain-related fear indeed produce the arousal and physiological symptoms noted in anxiety stemming from other stimuli. In particular, increased blood flow to the musculature and increased muscular activity in the anatomical areas in which the chronic pain is focused is a necessary first step.

2. Investigations that compare chronic musculoskeletal pain patients to health control participants (and, ideally, patient control participants such as people with acute pain) on challenges that increase muscle tension (e.g., static exercises), induce discomfort and pain (i.e., cold pressor), and increase general somatic sensations and discomfort are needed. Careful selection of tasks would allow researchers to evoke different patterns of responsivity in the autonomic nervous system—including sympathetic discharge and augmented vagal tone—allowing evaluation of the ability of the autonomic nervous system to respond in a bidirectional manner to stressors. It is through this line of investigation that the integrity of the autonomic nervous system in patients with chronic musculoskeletal pain, or homogeneous subsamples thereof, will become clear.

3. It must be demonstrated that relevant physiological arousal exacerbates pain-related fear in the cognitive and behavioral domains. Indeed, the influence of anxiety sensitivity, particularly sensitivity to physiological or somatic sensations, may be particularly fruitful. However, fine-tuned analyses may be required to elucidate whether measures of anxiety sensitivity are capturing fear-of-anxiety sensations provoked by situations expected to provoke pain, or whether such measures reflect fear of sensations of pain. While this distinction is subtle, it may hold implications for targeting treatment approaches. Anxiety-induction techniques may prove useful in this regard.

Although this discussion has focused on the physiological component of fear as an additional consideration in fear-avoidance models of chronic
musculoskeletal pain, this model may have implications for the maintenance of other chronic pain syndromes, including chronic headache syndromes. It is indeed possible that pain of different origins may prompt anxious responses, resulting in physiological symptoms that may exacerbate and perpetuate the positive symptom feedback and thereby promoting the negative cycle postulated in the fear-avoidance model of chronic pain. Regardless, the evidence reviewed here suggests that physiological activity occurring as a function of pain-related fear is an important variable deserving attention in the fear-avoidance models and in assessment and treatment packages for various chronic pain conditions.

References


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