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ORIGINAL ARTICLE

The Impact of Goal Cognition and Pain Severity on Disability and Depression in Adults with Chronic Pain: An Examination of Direct Effects and Mediated Effects via Pain-Induced Fear

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Abstract A group of 100 adults with chronic low back pain (CLBP), drawn from a larger national sample, completed a questionnaire battery that assessed (among other things) goal conflict and goal self-efficacy, pain severity, pain-induced fear, and 3 months later, two important clinical outcomes: physical disability and depression. Consistent with emerging motivation-centered models of adaptation (e.g., Ford, *Humans as self-constructing living systems: A developmental perspective on behavior and personality*. Erlbaum, 1987; Karoly *Review of General Psychology, 3*, 264–291, 1999) and cognitive-behavioral accounts of pain-specific fears (e.g., Asmundson et al. *Clinical Psychology Review, 19*, 97–119, 1999), structural equation analyses revealed that (a) goal self-efficacy, goal conflict, and pain severity independently predicted pain-induced fear, (b) pain-induced fear fully mediated the effects of goal conflict and goal self-efficacy on physical disability and depression, and (c) pain-induced fear partially mediated the effects of severity on disability and depression. Results suggest that clinical pain specialists should treat pain-induced fear as a means of forestalling disability and depression, and that they should also seek to modify how CLBP patients think about and organize their life goals.

Keywords Chronic low back pain · Goal conflict · Self-efficacy · Pain-induced fear · Structural equation modeling

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Introduction

Investigators of chronic pain are currently utilizing several conceptual models to guide their research. Cognitive modelers have, for example, established the importance of an array of information-processing biases, including those centering on attention, schematic understanding, beliefs, and memory, as key factors in the etiology and maintenance of maladaptive pain coping and related psychological dysfunctions (e.g., McKellar et al. 2003; Pincus and Morley 2001; Rode et al. 2001; Turner et al. 2000). Behavioral or learning-based researchers have, for their part, emphasized the role of classical conditioning as well as the secondary attentional gains that accrue to the overt expression of pain behaviors (e.g., Fordyce 1976; Philips 1987; Turk et al. 1983). And, because pain avoidance is now widely viewed as a presumptive force underlying the persistence of dysfunctional pain behavior, the study of the emotional, phobic, or ''fear driven'' aspects of pain reactivity has received renewed research attention in recent years (e.g., Asmundson et al. 1999; Crombez et al. 1999; DeRaedt et al. 2002; Vlaeyen and Linton 2000).

The purpose of the present research is to graft a motivational perspective onto the multi-component cognitive-behavioral study of both pain and fear, taking chronic low back pain (CLBP) as its clinical target. We focused on CLBP not only because it is a common and disabling pain condition for which biomedical factors are believed to explain only about half the disability variance (Waddell et al. 1984) but also because of recent efforts to establish the power of fear in the transition from acute to chronic status among low back pain sufferers (Vlaeyen and Linton 2000; Vlaeyen and Morley 2004).

Although several formulations of fear and its consequences for chronic pain have been put forward over the years (Lethem et al. 1983; Philips 1987; Vlaeyen et al. 1995), most tend to converge in their emphasis upon the de-motivating and illness-maintaining role of unpleasant or painful sensory signals that possess the potential to become the focus of anticipatory fear reactions. Moreover, a sizeable empirical literature has accumulated to support the broad utility of models that demonstrate a link between negative outcomes (such as disability and depression) and various psychological manifestations of fear (Asmundson and Taylor 1996; Burns et al. 2000; Crombez et al. 1999). In the present research, we seek to demonstrate that fear (specifically, pain-induced fear [see below]) directly impacts both depression and perceived disability, and, furthermore, mediates the effects of goal-centered motivational constructs (to be discussed) on depression and disability. The rationale for bringing a motivational conception to bear on how pain leads to disability and negative affect is briefly presented next.

A goal-centered motivational perspective on pain, fear, and adjustment

Contemporary motivational conceptions built around the organizing power of goals and self-regulatory processes may expand our understanding of maladaptive pain behaviors, including the vicious cycle of pain $\rightarrow$ fear $\rightarrow$ psychological dysfunction. For example, a broad-based social-cognitive perspective has strongly emphasized the importance of viewing the pain sufferer as an active processor of information whose patterns of coping and long-term adjustment are shaped by mental representations, attitudes, and beliefs (Bandura 1997; Pincus and Morley 2001; Jensen et al. 1994; Karoly and Jensen 1987; Turk and Okifuji 2002; Walsh and Radcliffe 2002). Over and above the

social-cognitive emphasis on active thought processes, a motivational model adds the higher-order element of long- and short-term *trajectory guidance* (the process of self-regulation) by assuming that pain is temporally regulated by a feedback-sensitive system (cf., Karoly 1985). This regulatory feedback system choreographs the individual's behaviors, thoughts, and feelings, permitting them to align with pre-set (but not invariant) internal standards, reference levels, or, more colloquially, *goals*. In other words, people are *goal-guided self-regulators* who strive for an array of long-term objectives (social, spiritual, work-related, health-related, etc.). Moreover, the pursuit of goals invariably involves the modulation of psychological states, including chronic pain and fear (cf., Carver and Scheier 1998; Karoly 1985, 1993, 1999); and the immediate and long-term effects of self-regulated emotions are invariably fed back to the higher-order goal system. Such a transactional view opposes the implicit biomedical assumption that the sole or preeminent determinant of action and thought in a chronic sufferer's life is the aversive pain experience itself. Instead of viewing pain perception as the unilateral driving force behind compensatory behaviors, negative affect, and biased information processing, a self-regulatory conception casts pain as a key player in a larger reciprocally deterministic system (cf., Bandura 1997; Jensen et al. 2003; Mischel and Ayduk 2004; Mischel and Shoda 1995).

Goal cognition and pain

Within the broad domain occupied by contemporary goal theories, it has also been suggested that people formulate schematic models of their goal pursuit processes that serve to propositionally specify, evaluate, organize, and store their goal-relevant experiences (e.g., Ford 1987; Karoly 1999). It has further been hypothesized that individuals who live with persistent pain for extended periods of time are motivated to create schematic working models not only of their pain, but of the relationship between their larger life pursuits (i.e., goals) and their nociceptive experiences (cf., Edwards and Pearce 1994; Hamilton et al. 2005; Karoly and Jensen 1987; Leventhal and Everhart 1979; Price and Harkins 1992). Presumably, then, cognitive models and schematic representations eventually come to play a formative role in the day-to-day experience of pain and in the associational links between pain and fear (see Power and Dalgleish 1997).

Recent research on the connection between pain, goal cognition, and goal pursuit has tended to confirm the view that everyday goal-directed behavior and thought can serve as potentially valuable analytic tools for apprehending the unfolding pain adjustment process. For example, Affleck et al. (2001) examined whether self-rated goal value and goal-relevant self-efficacy were linked to the experience of pain and to the ongoing pursuit of a health- and a social goal in a group of women with fibromyalgia. For 30 days, the women responded to palm-top computers that ''interviewed'' them about their pain and fatigue levels three times a day. The women also provided nightly reports about their effort and their progress on their social and health goals, and about how pain and fatigue interfered with the pursuit of these goals. In addition, one week prior to the onset of daily self-monitoring, the women rated their two goals on the *Value* and *Self-Efficacy* scales of the Goal Systems Assessment Battery (GSAB; Karoly and Ruehlman 1995), an instrument expressly designed to gauge propositional aspects of goal cognition. The results of Affleck et al.'s (2001) mixed, nomothetic–idiographic analysis of goal cognition and pain provided support for the proposed cognitive-motivational framework. For example, women assigning higher goal values to their

health and social pursuits reported expending more daily effort to attain their two goals and greater progress toward actually achieving them.

Pain adaptation and fear

The majority of individuals with persistent pain do *not* develop self-defeating habits such as fear avoidance or psychological disorders such as depression. In order to forge a comprehensive account of the role of fear in self-regulated pain adaptation, we must not only identify the *antecedents* of pain-linked fear, but we must offer a motivationally relevant definition of fear.

Therefore, in keeping with a number of conceptions of emotion (see Ekman and Davidson 1994; Lazarus 1991; Power and Dalgleish 1997), fear can be seen as a multidimensional emotional state. The literature linking pain and fear is currently focusing on its manifestation at higher levels, including the role of relatively complex schematic representations, such as fear of re-injury (Vlaeyen et al. 1995), catastrophic misinterpretation of bodily events (Sullivan et al. 2001), and beliefs about how avoidance of certain activities will prevent future pain (Fritz et al. 2001). In contrast, we elected, in the present study, to examine fear at its simplest or most basic level, i.e., as a motivated reaction to pain. Our measure, simply called *pain-induced fear*, assesses the degree to which an individual is *fearful of the pain experience per se.*

Specific hypotheses

In light of the goals research briefly reviewed above and the central role played by goals in multilevel models of emotion (including pain), we hypothesized that two dimensions of goal cognition might serve as distal determinants of disability and of depression through their direct connection to pain-induced fear.

First, we sought to examine the role of *goal conflict* as a potential contributor to pain-specific fear and to depression. As noted by Emmons (1999), intrapsychic conflict, a topic of interest that unites the Greek philosophers, Freudian psychoanalysts, and early behaviorists, has proven particularly useful in motivational studies of physical health and illness. With respect to conflict between goals (inter-goal conflict), research has demonstrated that such motivational incompatibility can be linked to physiological symptoms (Emmons and King 1988), to low levels of life satisfaction (Palys and Little 1983), and to a tendency to relapse after cigarette smoking cessation (McKeeman and Karoly 1991). In addition, Karoly and Ruehlman (1996) found, in a sample of persons working in managerial positions, that people with chronic pain reported higher levels of goal conflict than did individuals with no pain or with episodic pain. Therefore, in the present study, because inter-goal conflict has been shown to act as a source of ongoing frustration and negative arousal (Emmons 1999), we hypothesized that such conflict would be positively related to pain-linked fear and could indirectly precipitate depression and disability in low back pain sufferers.

We also sought to investigate the contribution of *goal-relevant self-efficacy* to fear activation. Self-efficacy, the belief that one has the ability to manage situations so as to achieve one's objectives, has proven to be an important psychological component in chronic pain adjustment and a key ingredient of chronic pain treatment regimens (e.g., Arnstein et al. 1999; Jensen et al. 1991; Lefebvre et al. 1999; Lorig and Holman 1993). Self-efficacy is also central to effective long-term self-regulation (Bandura 1997; Karoly

1993). Therefore, we hypothesized that back pain sufferers with low goal-centered self-efficacy would be predisposed to fearful reactions that could then trigger both disability and depressive affect.

The role of *pain severity* as an antecedent of fear, disability, and depression must also be considered. Having asserted that physiological arousal processes are not likely to serve as the only determinant of fear, we nonetheless acknowledge the powerful role of repetitive nociceptive stimulation and the influence of limbic structures in the total pain experience (see Chapman 2004). As many of the studies cited have shown, the aversive quality of pain tends to precipitate behavioral avoidance, emotional distress, and potentially dysfunctional patterns of cognitive appraisal. Therefore, we assume that pain severity will, in concert with selected goal measures, influence pain-induced fear. Moreover, because of its hardwired neural and noradrenergic links to nociceptive processes (Chapman 2004), pain severity was also hypothesized to have direct effects on both clinical outcomes: depression and disability. Finally, we note that unlike a great many studies of psychological factors in CLBP that make use of patients recruited from medical settings, the present research was conducted as part of a national survey of pain and its psychosocial characteristics in which random-digit dialing procedures were employed to obtain a representative sample of adults who were interviewed by phone and, as was the case with the sub-sample reported here, also asked to complete a paper-and-pencil battery of pain-relevant questionnaires on two occasions. We provide a psychometric rationale (see Sect. ''Method'' below) for those scales employed in the present analysis. Additional information regarding sampling procedures and scale development can be found in Ruehlman et al. (2005a, b).

Method

Participants

Participants included 100 individuals with CLBP. The sample is a subgroup of a national sample (N = 2,407) of respondents with a wide variety of types of chronic pain. Brief descriptions of the national sample and the low back pain sub-sample are provided below.

National sample

The national sample of 2,407 was obtained through random digit dialing (RDD)[1] and was stratified by gender and according to three age groupings (25–44, 45–64, and 65–80). Each of the six Age × Gender groups consisted of approximately 400 subjects with chronic pain. Respondents were screened via telephone interview using the Profile of Chronic Pain: Screen (PCP: S; Ruehlman et al. 2005a). Participants were required to score at least one standard deviation above the mean on one or more of the three scales of the PCP: S to be eligible for participation and were required to speak English. Of a

[1] The RDD procedure involves the generation (by a survey sampling company) of a list of phone numbers, randomly drawn from across the United States to be representative of population distribution patterns. Non-working, cellular, and non-residential numbers are excluded from this original list. Calls were made throughout the day and evening on all days of the week. A working number was called a minimum of six times. Calling patterns were regulated in keeping with the time zone being called, so as to neither call too early in the morning nor too late in the evening.

total of 9,759 individuals who were screened for chronic pain, 3,050 subjects were judged eligible. Of those who were eligible, 2,407 chose to participate, yielding a good telephone survey response rate of 79%[2]. Those who were eligible for participation on the basis of their scores on the PCP: S, and who agreed to participate, were then administered the Profile of Chronic Pain: Extended Assessment battery (PCP: EA; see Ruehlman et al. 2005b) via telephone interview. The average duration of the interview was 34 min. Respondents were paid $30 in exchange for their participation.

Low back pain subgroup

The low back pain group was drawn from a sub-sample of 261 (127 females, 134 males) of the original subjects who volunteered to take part in a psychometric validation study of the PCP: EA. The group of 261 represented a sub-sample of the original 2,407 that was willing to complete additional questionnaires twice. All participants were paid $100.00 to complete two paper-and-pencil surveys, separated by a 3-month interval. To be considered for the present analyses, respondents were required to score a 3, 4, or 5 when asked to rate the average intensity of lower back pain over the past 6 months, using a 0 (''it hurt a little'') to 5 (''it was extremely painful'') point scale. Thus, from the 261 who participated in the additional assessment, a total of 100 individuals with lower back pain of moderate intensity (at least a three on a six point scale) were included in the present analyses. The majority of the participants were female (56%). The sample was primarily Caucasian (80%), but also included African-Americans (15%), Hispanics (2%), and those who described themselves as ''mixed'' or ''other'' (3%). Twenty-six percent of the participants were between the ages of 25–44 years old, 41% were 45–64 years old, and 33% were 65–80 years old. Seventeen percent of the sample was employed full-time, 9% part time, 26% were disabled, 25% retired, 7% were homemakers, 14% were unemployed, and 2% failed to provide employment data**.** All but one respondent reported currently receiving treatment for their pain from a health care professional. Over half (58%) of the sample was married, 7% living together as married, 13% single, 13% divorced, and 9% widowed. Fifteen percent of the sample reported having less than a high school degree, 31% had a high school diploma, 32% had some college training, 11% had a bachelor's degree, and 11% had an advanced degree.

Measures

Although participants completed an extensive battery of instruments, only those that are relevant to the present study are herein described.

Goal measures

Two goal-related constructs were included in the present study: goal conflict and goal self-efficacy. As part of the first paper-and-pencil survey, participants were asked to list

[2] To examine possible selection biases, the 643 unwilling participants were compared to the 2,407 willing subjects. A Multivariate Analysis of Variance was conducted in which willingness to participate was the between subjects factor and scores on the PCP: S were the dependent measures. The groups differed significantly (Wilks' Lambda = .97; F (3, 3046) = 31.85, $p < .001$). However, the partial η^2 was .03, indicating that group differences were quite small.

their ''current, medium range goals—goals toward which you will be working for a minimum of several weeks up to a maximum of a year''. From this list, subjects selected their three most important goals and ranked these in order of importance. Next, participants were asked to rate how much each of their three most important goals tended to conflict (or interfere) with each other goal (0 = no conflict, 5 = A great deal of conflict), yielding three conflict ratings. A total *goal conflict* score was constructed by summing these three ratings, yielding a possible score range of 0–15. The mean and standard deviation for this scale were 4.78 and 4.99, respectively. Conflict assessed in this manner has proven to be quite effective in predicting various psychological and physical outcomes (see Emmons and King 1988). Internal consistency reliability as estimated by coefficient α was.86.

Next, subjects completed the 36-item Goal Systems Assessment Battery (GSAB; Karoly and Ruehlman 1995) for their most important goal. As noted above, the GSAB provides information concerning nine important goal-based self-regulatory skills. Individual items employ a 0–4 point rating scale. The GSAB possesses acceptable psychometric properties including good retest reliability, low social desirability response bias, and ample evidence of predictive validity. In addition, the factor structure of the instrument has been confirmed across multiple samples. Only the 4-item Self-Efficacy scale of the GSAB was used in the present analyses. The mean and standard deviation for this scale were 11.74 and 3.56, respectively. Internal consistency reliability as estimated by coefficient α was.86.

Pain severity

The pain severity sub-scale of the PCP: S (Ruehlman et al. 2005a) served as our index of pain's aversive qualities. The 5-item scale included items that assessed average pain level, frequency of pain, typical pain level, frequency of at least 1 h of severe pain, and the greatest amount of pain experienced in the past 6 months. The severity scale yielded a coefficient α of .89 in the national sample and a retest reliability of .77.

In the present sample, the coefficient α was .67. The mean and standard deviation for this scale were 23.91 and 3.84, respectively.

Pain-induced fear

Respondents completed the PCP: EA (Extended Assessment) battery (Ruehlman et al. 2005b). Of particular interest in the present analysis was the measurement of pain-induced fear via the 5-item PCP: EA Fear of Pain Scale (When I am in pain, I: (1) feel scared; (2) am afraid the pain will get too intense; (3) don't feel safe; (4) feel afraid; and (5) feel a sense of panic). The Pain-Induced Fear Scale has been found to be internally consistent and temporally reliable. Coefficient α, based on the 2,407 respondents in the national sample, was .85. Retest reliability, calculated from data gathered on those individuals who completed both a PCP: EA telephone interview and paper-and-pencil version of the scale, was.73 ($n = 171$; $p < .01$). Construct validity of the Pain-Induced Fear Scale was examined based on individuals who completed a paper-and-pencil validity battery that included the Zung Self-Rating Anxiety Scale (Zung and Cavenar 1980) and a 54-item symptom checklist (Pennebaker 1982). The Pain-Induced Fear Scale correlated significantly with the Zung Anxiety Scale [r (168) = .57, $p < .01$] and with the number of physical symptoms experienced over a period of a month [r (185)

=.49, $p < .01$]. In the present study, the coefficient α was .90. The mean and standard deviation for this scale were 8.70 and 7.66, respectively.

Pain disability index

Perceived pain disability was assessed approximately 3 months after the initial assessment of goals and fear of pain using the Pain Disability Index (PDI; Pollard 1984). The PDI assesses disability across seven areas of functioning, including family/home, recreation, social activities, occupation, sexual behavior, self-care, and life-support activity. Factor analyses have indicated that both a one-factor and a two factor model appear to yield reasonable fit (Chibnall and Tait 1994; Tait et al. 1987, 1990). This widely used instrument has been found to possess extensive evidence of validity. For example, correlations have been reported between scores on the PDI and pain behaviors, post-surgical status, inpatient versus out patient status, employment status, circumstances of pain onset, and litigation status (Chibnall and Tait 1994; Jerome and Gross 1991; Pollard 1984; Tait et al. 1987, 1990). In the present study, the coefficient α was .91. The mean and standard deviation for this scale were 33.49 and 17.29, respectively.

Center for epidemiological studies depression scale

Depression was also measured at the 3-month follow-up using the 20-item Center for Epidemiological Studies Depression Scale (CES-D: Radloff 1997). The CES-D has been widely used in many settings and with a variety of participants, including those with chronic pain. It has been found to have adequate reliability (e.g., α of .85 and 1 month retest reliability of .67) and strong predictive validity. In the present study, the coefficient α was .92. The mean and standard deviation for this scale were 21.51 and 13.44, respectively.

Results

Correlational analyses among the main study variables

As hypothesized, significant ($p < .05$) negative associations were found between goal self-efficacy and all the other study variables, including participant's scores on the CESD and the PDI (rs ranged from –.20 to –43). Goal conflict and severity were significantly ($p < .001$) correlated with pain-induced fear ($r = .37$ and .46, respectively), CESD scores (.36 and .45, respectively), and PDI scores (.35 and .55, respectively). Pain-induced fear, in turn, was significantly ($p < .001$) correlated with CESD and PDI scores, $r = .66$ and .62, respectively. The correlation between CESD and PDI scores was also significant, $r = .59$, $p < .001$.

Structural equation analysis

We used structural equation modeling to test our hypothesis that pain-induced fear would fully mediate the effects of goal self-efficacy and goal conflict, and would partially mediate the effects of severity on both the CESD and PDI.

Table 1 Model fit and difference tests for direct effects of goal self-efficacy, goal conflict, and severity on CESD and PDI Scores

Model	χ^2	*df*	Exact *p*	χ^2:*df*	AIC	RMSEA
1. Saturated model	<.001	3	1.00	<.000	–6.00	<.001
2. Drop self-efficacy direct effects	.588	5	.98	.117	–9.41	<.001
3. Drop goal conflict direct effects	4.936	7	.66	.705	–9.06	<.040
4. Drop severity direct effects	21.596	9	.01	2.399	3.59	.119
Model comparison	χ^2change	*df*	Exact *p*			
1 vs 2	.587	2	.75			
2 vs 3	4.34	2	.12			
3 vs 4	16.66	2	<.001			

First, we tested a saturated model in which we specified the direct effect of all exogenous variables on both depression and disability. In light of the significant association between the CESD and the PDI scores, we included a path specifying the partial regression coefficient between each outcome. In effect, this partial regression coefficient can represent any excluded factor that may account for common variation or any residual relation controlling for the effects of the exogenous variables (cf., Loehlin 2004).

As predicted, the direct effects of goal self-efficacy and goal conflict on both the CESD and PDI scores were not significant. Furthermore, consistent with our hypothesis, the direct effects of pain severity were significantly related to these two outcomes ($p < .05$). As can be seen in Table 1, the saturated Model 1 provided an excellent fit to the data, $\chi^2(100, 3) < .001$, $p = 1.00$, AIC = –6.00, RMSEA < .001.

Next, we systematically trimmed the saturated model by dropping, in sequential fashion, the direct effects of goal self-efficacy, goal conflict, and severity on both CESD and PDI scores. Dropping the paths representing the direct effects of goal self-efficacy in Model 2 did not produce a significant degradation in fit compared to Model 1, χ^2 change (100, 2) = 0.587, $p = .75$. Similarly, dropping the paths representing the direct effects of goal self-efficacy in Model 3 did not produce a significant reduction in fit compared to Model 2, χ^2 change (100, 2) = 4.34, $p = .12$. However, when we dropped the paths representing the direct effects of severity on both the CESD and PDI in Model 4, the degradation in fit relative to Model 3 was, in fact, significant, χ^2 change (100, 2) = 16.66, $p < .001$. Consequently, we retained Model 3, which fit the data very well, χ^2 (100, 7) = 4.936, $p > .66$, AIC = –9.064, RMSEA = .040.

Figure 1 depicts Model 3 with standardized estimates of each path. Goal self-efficacy, goal conflict, and severity exerted significant direct effects on pain-induced fear (highest $p < .01$). Severity and pain-induced fear, in turn, exerted significant direct effects on both CESD and PDI scores (highest $p < .05$).

Estimation of indirect effects

To examine the extent to which the effects of goal self-efficacy, goal conflict, and pain severity on depression and disability were mediated by pain-induced fear, we computed indirect effects. This computation was achieved by multiplying the unstandardized path coefficients from each of these exogenous variables to pain-induced fear by the unstandardized path coefficients from pain-induced fear to CESD and PDI scores. Table 2 presents the indirect effects for each exogenous variable on both the CESD and

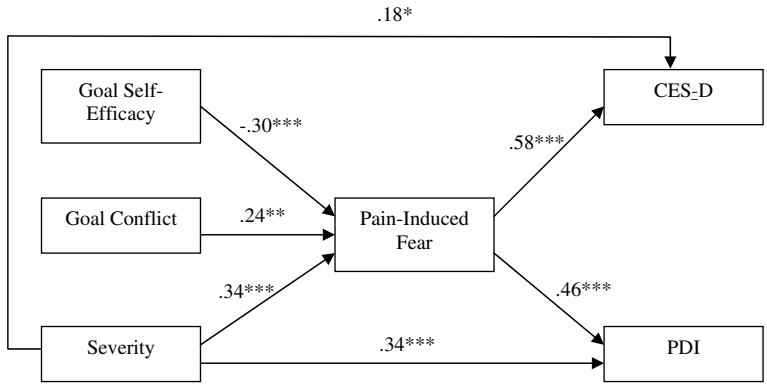

Fig. 1 Summary of the mediational model of the relations among goal self-efficacy, goal conflict, severity, pain-induced fear, CESD, and PDI. *$p < .05$, ***$p < .001$. All path coefficients are standardized estimates

Table 2 Sobel tests for the indirect effects of goal self-efficacy, goal conflict, and severity

Dependent	*a*	*b*	*ab*	S_{ab}	*z*
CESD					
Goal self-efficacy	–64	1.02	–.65	.20	–3.19**
Goal conflict	.37		.38	.14	2.71**
Severity	.68		.69	.20	3.54***
PDI					
Goal self-efficacy	–.64	1.04	–.67	.22	–3.01**
Goal conflict	.37		.38	.15	2.60**
Severity	.68		.71	.21	3.30***

All coefficients are unstandardized estimates

** $p < .01$, *** $p < .001$

the PDI. Using the Sobel procedure (MacKinnon et al. 1995; Sobel 1982), the mediated effects for goal self-efficacy, goal conflict, and pain severity on CESD and PDI scores were all found to be significant (highest $p < .01$).

Whereas goal self-efficacy exerted a negative mediated effect on both the CESD and PDI, goal conflict and pain severity each exerted positive mediated effects on these outcomes. Relative to goal conflict, the magnitude of the mediated effects was larger for goal self-efficacy and pain severity. The effects of goal conflict and goal self-efficacy on the two dependent measures were fully mediated by pain-induced fear as evidenced, first, by their non-significant direct effects on the CESD and PDI scores, and, second, by their significant indirect effects on both indices via pain-induced fear. By contrast, the effects of severity on CESD and PDI scores were only partially mediated by pain-induced fear, as evidenced by severity's significant direct effects on depression and disability and its significant indirect effects on CESD and PDI scores via pain-induced fear.

When a variable has significant direct and indirect effects, the magnitude of a mediated effect can be gauged by computing the percentage of a variable's total effect that is indirect. Of our three exogenous variables, only severity exerted *both* direct and indirect effects. Using the standardized coefficients in Fig. 1, the mediated effect for

pain severity on CESD scores via pain-induced fear was .20 (.34 × .58), and its direct effect on the CESD was 0.18. Thus, over half of the total effect of pain severity on depression was indirect. Similarly, the mediated effect of pain severity on PDI-assessed disability via pain-induced fear was 0.16 (.34 × .46), and its direct effect on the PDI was .34. Thus, approximately one-third of the total effect of pain severity on disability scores was indirect.

Discussion

Research on CLBP has focused on an assortment of biological and psychosocial constructs as putative causes, facilitators (moderators and mediators), and/or maintenance conditions. Adequate treatment and prevention of CLBP clearly depend upon an appreciation for all of the relevant biopsychosocial dimensions that might serve to place individuals at risk for debilitating long-range maladaptive outcomes, such as depression and disability (Garofalo and Polatin 1999). In recent years, various cognitive indices of pain-related fear have emerged as important components of maladjustment in CLBP because, among other things, these fear indices have been shown to relate negatively with cognitive functioning (e.g., coping, decision-making, attentional control) and positively with self-perpetuating avoidance behaviors (Asmundson et al. 1999; Crombez et al. 1999; McCracken and Gross 1993). Less well understood, however, has been the role of pain-induced fear per se. The present study made use of structural equation modeling to evaluate the incremental utility of a motivational perspective that emphasizes the role of goal-based self-efficacy, goal conflict, and of pain severity as antecedents of pain-induced fear, which, in turn, was expected to have the most proximal influence on later assessed depression and disability.

In general, our results tended to support the proposed structural model as tested in a sample of community-residing adults with CLBP. As expected, pain-induced fear revealed strong direct paths to both depression and disability in our middle-aged sample of low back pain sufferers. Also as predicted, our goal-based indices were strongly linked to pain-induced fear, with self-efficacy revealing a significant inverse relation and conflict exhibiting a significant positive relationship. Particularly important was the finding that, in the prediction of disability and depression, neither goal-based self-efficacy nor goal conflict exerted direct effects, thus supporting the mediational role of pain-induced fear (see Fig. 1).

Pain severity had a significant direct effect upon both disability and depression. The aversive qualities of pain, indexed by the PCP: S Severity sub-scale, are supported centrally by multiple neurotransmitter systems and by various somatosensory pathways. The semantics of intense and frequent pain transcends bodily arousal, and can be ''read'' as threat, diminished expectancies, loss of reward, and frustration as well. Thus, it is not surprising that pain severity was found to have a direct effect upon perceptions of disability and negative affectivity. In fact, a recent analysis of industrial workers with low back pain (Gheldof et al. 2006) found that pain intensity was a strong predictor of both functional and social disability. Perhaps more interesting, in the present context, is severity's indirect influence on both disability and depression via pain-induced fear. Perhaps the physiological arousal linked to the interoception of severe pain gives rise to fear at multiple levels (see Power and Dalgleish 1997), possibly including the schematic appraisal that one's basic survival goals are being threatened as well as the prepositional

appraisals that one may become incapacitated (i.e., disabled) and should withdraw from active pursuit of rewarding activities (i.e., depression). The dampening effects of pain and pain-induced fear upon the motivation to actively engage the world is reflective of the well-known inhibitory relation between aversive and appetitive systems in animals (Dickinson and Pearce 1977) and merits further study in human motivation.

It is important to be cautious and to bear in mind that structural equation models require the a priori specification of relationships among variables. Thus, the finding that the fit of a particular model to the data is good does not rule out the possibility that other models may also fit the data well, or that the direction of causality might plausibly have been reversed (Kline 1998). However, causal confidence is aided, in the present case, by our use of a short-term longitudinal design. Depression and disability could not logically have precipitated fear assessed 3 months earlier. And it is difficult to imagine how pain-induced fear could have ''caused'' the varied goal ratings.

It is also important to note that the present findings are limited in several ways. First, the data are exclusively of the self-report variety. Therefore, the obtained relationships may partially reflect the effects of shared method variance. Second, the participants in the present research consisted of adults recruited by persons unknown to them rather than by the staff of a clinic where they are patients, as is the norm for research on predictors of pain adjustment. This fact doubtless carries disadvantages and advantages. Nonetheless, we are constrained in our ability to compare our findings to those reported in much of the extant literature. Additionally, relative to the larger sample of 2407 pain patients, our group of 100 low back pain participants represents persons willing to participate in additional, time-consuming research (for pay); and they may, therefore, possess psychological characteristics that render them distinct from low back pain patients recruited from clinical settings.

One obvious direction for future investigations would be to explore the connections between the current index of pain-induced fear and higher-order cognitive fear appraisals/attitudes such fear avoidance beliefs, fear of re-injury, anxiety sensitivity, and catastrophizing. Investigating other aspects of goal cognition and the role of emotions other than fear represent two additional important directions for research on chronic pain (cf., Hamilton et al. 2004; Keefe et al. 2001). The systematic examination of multiple, ongoing goals being pursued by chronic pain patients, particularly goals that tend to be mutually facilitative or mutually disruptive, would provide a broader motivational base than that provided in the present research. Also, in the present study, we limited our focus to a content appraisal of goal self-efficacy and to a structural index of goal cognition (goal conflict). However, other health researchers have explored the influence of *styles* or profiles of motivational functioning upon pain adaptation. Schmitz et al. (1996), for example, found that dispositional differences in the tenacious (assimilative) versus flexible (accommodative) styles of goal cognition were linked to different chronic pain adjustment outcomes. In general, the connection between pain intensity and disability/depression proved to be moderated by levels of accommodative thinking. It would therefore be instructive to examine the relationship between so-called assimilative/accommodative styles and the present measure of pain-induced fear.

The skillful modulation (i.e., activation and deactivation) of one's emotional states and of their communicative expression, including not only negative emotions such as fear, anger, and anxiety, but also positive emotions such as enjoyment, may hold important untapped clinical benefits for pain sufferers. Without denying the clinical importance of controlling fear and other negative affective responses in persons with chronic pain, we can point to data from several research groups that suggest greater

scrutiny of positive affect and its regulation. Positive emotions have been empirically linked to adaptive patterns of thought and behavior that are distinct from those precipitated by negative emotions. Fredrickson (2000) and her colleagues have shown, for example, that positive emotions can help to down-regulate the cardiovascular effects of negative mood states. Similarly, Isen and her colleagues (e.g., Isen et al. 1987) reported that positive affect can facilitate problem-solving. Dreisbach and Goschke (2004) demonstrated, in a controlled laboratory context, that positive affect increases cognitive flexibility. Finally, Zautra and his colleagues (e.g., Zautra et al. 2001) have found that, among rheumatoid arthritis patients, the presence of positive affect can serve, during painful episodes, to interrupt the pain $\rightarrow$ negative affect link. Thus, in the future, the relation between goal cognition, positive affect, and indices of well-being might profitably be explored in a manner similar to that of the present study.

Finally, speculating about the therapeutic implications of our findings, we wonder whether, in treating patients with pain-related fears, it might be useful and cost-effective to assess their current goal appraisals and inter-goal conflicts, and then devote a session or two to the process of goal re-organization. Although cognitive regimens and exposure-based therapies have been shown to be clinically effective for pain patients, their effects may well be magnified if conflicts between therapy and non-therapy goals and low self-efficacy expectations were also addressed.

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