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Transition from acute to chronic pain and disability: A model including cognitive, affective, and trauma factors

Corinna Young Casey [a,*], Melanie A. Greenberg [b], Perry M. Nicassio [c], R. Edward Harpin [d], David Hubbard [e]

[a] Department of Psychiatry, University of California, San Diego, School of Medicine, La Jolla, CA, USA
[b] California School of Professional Psychology, Alliant International University, San Diego, CA, USA
[c] Cousins Center for Psychoneuroimmunology, Neuropsychiatric Institute, University of California, Los Angeles, Los Angeles, CA, USA
[d] Pain Rehabilitation Services, Sharp Healthcare, San Diego, CA, USA
[e] MyoLink, LLC, San Diego, CA, USA

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Abstract

This study evaluated a theoretically and empirically based model of the progression of acute neck and back pain to chronic pain and disability, developed from the literature in chronic pain, cognition, and stress and trauma. Clinical information and standardized psychosocial measures of cumulative traumatic events exposure (TLEQ), depressed mood (CES-D), pain (DDS), physical disability (PDI), and pain beliefs (PBPI) were collected at baseline from 84 acute back pain patients followed at an Acute Back Clinic over 3 months. Path analysis was used for the longitudinal prediction of perceived pain and disability. The predictive model accounted for 26% of the variance in persistent pain intensity and 58% of the variance in perceived physical disability at 3 months. Greater exposure to past traumatic life events and depressed mood were most predictive of chronic pain; depressed mood and negative pain beliefs were most predictive of chronic disability. More cumulative traumatic life events, higher levels of depression in the early stages of a new pain episode, and early beliefs that pain may be permanent significantly contribute to increased severity of subsequent pain and disability. Replication in a larger sample is desirable to confirm these paths. Early detection of elevated depressive symptoms and high trauma exposure may identify individuals at greater risk for developing chronic pain syndromes who may benefit from early multidisciplinary intervention.

Keywords: Acute pain; Chronic pain; Disability; Model; Trauma; Depression; Cognitive; Psychosocial factors

1. Introduction

Chronic pain is a prevalent and costly national health problem. Although most episodes of back pain resolve within 6 weeks (Spitzer, 1987), nearly half of pain sufferers have symptoms which persist and debilitate them for years (Carette, 1994). For those, whose conditions have transitioned from acute to chronic pain (pain persisting for 3 months or longer; International Association for the Study of Pain, 1986), there are often few physical abnormalities.

Although it is clear that psychosocial factors play a role in chronic pain, most studies are cross-sectional or retrospective. The few prospective studies using acute pain samples have not identified specific pathways linking psychosocial factors to pain perpetuation. Integrating the cognitive and trauma literatures into our understanding of pain may elucidate the mechanism(s) through which chronicity develops from acute pain (less than 6 weeks duration; American College of

* Corresponding author. Tel.: +1 858 794 9413; fax: +1 858 876 3128.
E-mail address: cycasey@ucsd.edu (C. Young Casey).

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Rheumatology Clinical Guidelines, 1996). We have developed a theoretically and empirically grounded model (Fig. 1) that represents such an integration. This article describes an initial test of the model in a clinical acute pain sample.

We began with the variables found in prior research to predict pain perpetuation. Acute pain intensity (White et al., 1997; Epping-Jordan et al., 1998), and depressive symptoms (Banks and Kerns, 1996; Geisser et al., 2000), each positively and directly influence the persistence of neck and back pain and disability and are also positively intercorrelated (Von Korff and Simon, 1996; Epping-Jordan et al., 1998). Therefore, we hypothesized that acute pain and disability would predict chronic pain and disability directly, and indirectly, via baseline depression.

Research suggests exposure to severe stressors can permanently change neurobiological processes or structures, negatively affecting arousal thresholds and ability to cope with subsequent stress (van der Kolk, 1996). Thus, past traumas might "hard-wire" individuals to experience more intense distress and pain following an acute injury or inflammation. Cross-sectional studies support a positive relationship between history of traumatic or stressful life events and chronic pain (Boisset-Pioro et al., 1995; Kim et al., 1996; Raphael et al., 2001). Trauma exposure has also been positively associated with depression in healthy populations (Leserman et al., 1998; van der Kolk, 2001) and neck and back pain intensity in pain patients (Saxe et al., 1994). Therefore we posited cumulative trauma exposure would predict more chronic pain and disability directly, and indirectly, via baseline depression.

Previous studies have found a positive association between negative pain beliefs, such as constancy and permanence, and pain chronicity (Wells, 1994; Burton et al., 1995), and between depression and learned helplessness, cognitive distortions, and pessimistic beliefs about the future in healthy samples (see Haaga et al., 1991 for a review). Therefore, we posited that baseline depressive symptoms would positively predict chronic pain and disability both directly, and indirectly, via negative pain beliefs.

This study tests the hypothesized model by evaluating the direct and indirect effects of cumulative trauma exposure, acute pain severity and disability, and baseline depressive symptomology and pain beliefs on chronic pain severity and disability using path analysis (see Fig. 1).

2. Methods

2.1. Recruitment

Participants for this study were recruited from the Sharp Rees-Stealy Acute Back Clinic (ABC) in San Diego County, operated at two locations in the county by one of the largest medical groups in the region. Patients with a new onset of pain are referred to the ABC from their Primary Care Physician (PCP) or an Urgent Care in the Sharp HealthCare system. The time from when the referral is made until the time the patient is seen at the ABC ranges from the same day to less than 1 week. Patients at the ABC were co-evaluated by

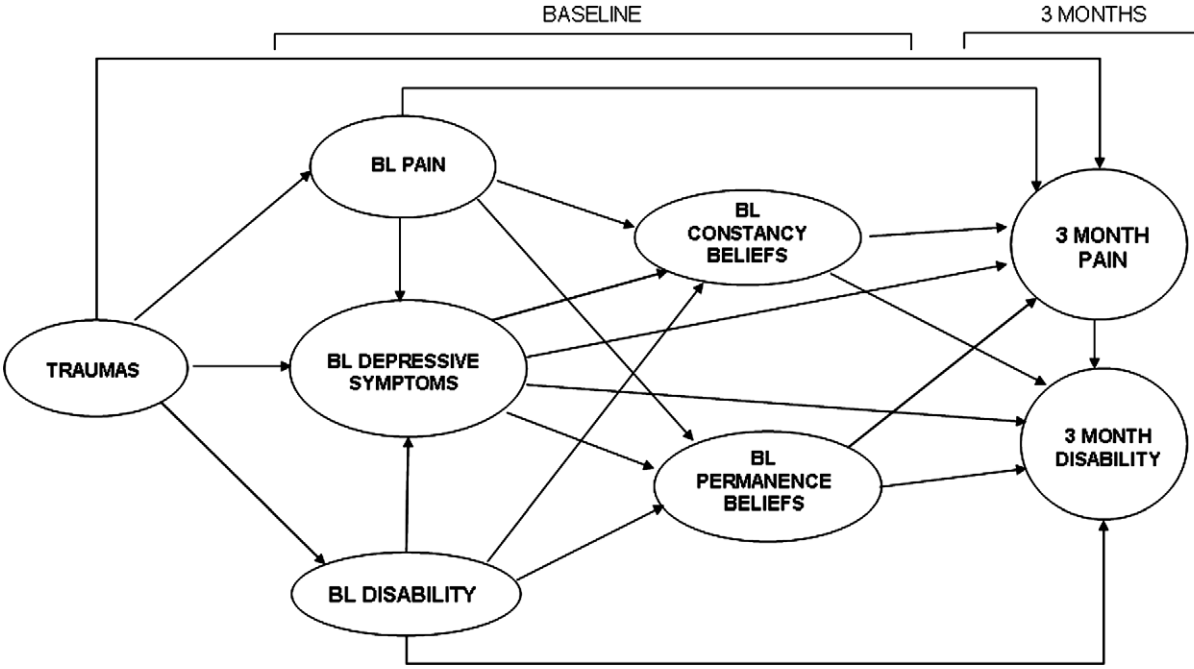

Fig. 1. Hypothesized model of transition from acute to chronic pain and disability.

physical medicine and rehabilitation physicians certified in pain management and a physical therapist and treated according to standard of care guidelines. Patients who reported an occupational injury were referred to their employer's designated workers' compensation treating physician.

2.2. Participants

The present study included 84 English-speaking patients seen at the ABC for new onset neck or back pain of less than 8 weeks duration, who were followed over 3 months, and completed both assessments. An additional 13 patients, who completed only the baseline assessment, were excluded from these analyses. They did not differ significantly from the study group on demographic or medical characteristics or on baseline levels of outcome and independent variables (all p's $> .10$) with the exception of pain constancy beliefs, for which the baseline only patients exhibited stronger beliefs that their pain was constant and unchanging ($p < .01$). This study was approved by the Sharp Healthcare Institutional Review Board, and all participants provided written informed consent prior to entry into the study. Patients were excluded from the study if they had any history of spine surgery, previous neck or back pain within the 6 months prior to their current pain episode, history of any psychotic or delusional disorder, or if they had any serious concurrent pain-related medical conditions. Over a 1 year period, all patients who were evaluated at the ABC and who met study criteria were invited to participate in the study by the principal investigator at the end of their first office visit. Each participant was provided two movie tickets upon completion of the first assessment and $10 after the second as compensation for participation.

The majority of the participants were Caucasian (80%), women (59%), married (61%), in their mid-40s (M(SD) = 46.89(13.6)), and had at least a college degree (67%). Just over half of the participants had a history of previous back pain (57%) and a similar percentage did not have a specific explanation for the cause of their pain (51%); two individuals had a work injury. The mean duration of pain symptoms at initial visit was 3.6 weeks (SD = 2.2 weeks). Using the Diagnostic Classification System of the Quebec Task Force on Spinal Disorders (Spitzer, 1987), the majority of participants (59%) were classified with neck or back pain without any radiation to the extremities while 19% were identified as having neck or back pain with some neurological signs (see Table 1).

Table 1
Medical characteristics ($N = 84$)

Variables	M (SD) or %
History of back pain	57%
Pain duration at baseline (mean # of weeks)	3.6 (2.2)
Quebec Task Force Diagnostic Classifications	
Level 1 – pain without radiation	59%
Level 2 – pain + radiation to proximal extremities	11%
Level 3 – pain + radiation to distal extremities	11%
Level 4 – pain + radiation to upper/lower limb (neuro signs)	19%
Cause of Injury	
Bending/twisting	8%
Do not know/other	51%
Fall	6%
Lifting	11%
Motor vehicle accident	14%
Weights/exercise	7%
Work injury	3%
3-Month follow-up	
Pain status	
Pain free	18%
Less pain	64%
Same	10%
More pain	8%
Sick/disability days	4.31 (14.86)
Weekly medication usage (in number of total pills)	
Over the counter analgesics	21.16 (22.49)
Prescribed opioids, anxiolytics, benzodiazepines	6.86 (14.84)
Prescribed muscle relaxants, sleeping aides	7.44 (15.28)

2.3. Questionnaires

All patients completed the full battery of questionnaires upon enrolling in the study at baseline and then again 3 months later. The battery assessed the following constructs: pain intensity, pain disability, cumulative trauma exposure (administered at baseline only), depression, and pain schemas. The battery also included a comprehensive assessment of demographic, clinical and medical factors.

Pain intensity was assessed at baseline and 3-month follow-up using the Descriptor Differential Scale (DDS; Gracely and Kwilosz, 1988), consisting of 12 analogue scales, each with a distinct descriptor of sensory intensity, covering a full range of pain, which minimize floor and ceiling effects. The 12 scores from each scale are summed and averaged to create a total mean score. The DDS has exhibited excellent reliability and validity and avoids many of the psychometric problems of single-unit visual and numeric analogue scales (Gracely and Kwilosz, 1988). It is also more sensitive than traditional measures to small differences in stimulus intensity (Doctor and Slater, 1993).

Pain disability was assessed at baseline and follow-up using the Pain Disability Index (PDI; Pollard, 1984), a 7-item inventory measuring the degree to which pain interferes with functioning across a range of activities. This instrument demonstrates high internal consistency, reliability $\alpha = .86$ (Tait et al., 1990), and test–retest reliability (ICC = .91; Gronblad et al., 1993).

Traditionally, stressful event measures limit reporting to a 1-year time frame (Brown and Harris, 1978; Murphy and Brown, 1980). Traumatic events, however, can have a significant impact on mental and physical health despite their occurrence years earlier (Leserman et al., 1996), which argues for inclusion of lifetime experiences in trauma questionnaires. The construct of *cumulative trauma exposure* was measured at baseline with the Traumatic Life Events Questionnaire (TLEQ; Kubany et al., 2000), which comprises 23 questions that assess lifetime exposure to 17 types of potentially traumatic events, based on DSM-IV (American Psychiatric Association, 1994) Criterion A for Post Traumatic Stress Disorder. It uses behaviorally specific questions, includes both dichotomous and continuous responses, and assesses qualitative characteristics of events by asking whether the event evoked

"intense fear, helplessness, or horror." It demonstrates content validity, empirical validity against a structured interview ($\kappa = .71$), and 2 month test–retest reliability among men and women in a residential substance abuse program (average $\kappa = .57$, range = .29–.91; Kubany et al., 2000).

Depressive symptomology was assessed at baseline and follow-up by Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1977), a self-report measure that assesses depressive cognition, dysphoric mood, and vegetative signs in the general population. It displays good internal consistency with Cronbach's $\alpha = .86$ and 1 year test–retest reliability ranging from .31 to .54 (Radloff, 1977). While a cutoff score of 16 is recommended for identification of probable clinical depression in the general population (Weissman et al., 1977), a cutoff of 27 is considered a more appropriate norm for clinical depression in the chronic pain population, due to the overlapping somatic symptoms (Geisser et al., 1997).

Pain schemas were assessed at baseline and follow-up by the Pain Behavior and Perception Inventory (PBPI; Williams and Thorn, 1989), which assesses patients' beliefs about their pain and its prognosis. It consists of 16 items and displays good internal consistency on all estimates (Morley and Wilkinson, 1995). It primarily measures four-dimensions of pain beliefs: Mystery, Pain Permanence, Pain Constancy, and Self-Blame. For this study only the Pain Permanence and Pain Constancy factors were included in analyses as they made the most theoretical sense for the longitudinal model of chronic pain and disability proposed in this study. Time, the factor that originally comprised these two subsequent factors, assesses the belief that pain will be enduring. It was also the only factor positively associated with pain intensity in the original development and validation study of the PBPI (Williams and Thorn, 1989). Since the "Time" factor was split into two separate factors, Pain Permanence and Pain Constancy, one study found Pain Constancy to be the only factor significantly and positively associated with pain intensity (Williams et al., 1994). However, the Pain Permanence scale retains most of the construct of the Time scale, that being "Pain is enduring." Due to this discrepancy, this study used both Pain Permanence and Pain Constancy. The Pain Permanence subscale measures the construct of *time* (belief that pain will be enduring) while the Pain Constancy subscale measures beliefs that pain is always there.

2.4. Diagnostic category

To control for the different sources and physiological pathways of pain, this study employed the Diagnostic Classification System of the Quebec Task Force on Spinal Disorders (Spitzer, 1987) to categorize pain diagnoses at baseline. It is based mostly on simple clinical criteria that encompass the majority of cases encountered in clinical practice. The classification system includes 11 categories, based on history, clinical examination and response to treatment. Categories 1–3 refer to the localization of the pain (e.g., pain without radiation or pain with radiation), category 4 reflects the results of clinical examination (i.e., neurologic signs), categories 5–7 reflect results of objective assessments, such as radiologic, laboratory, or electronic devices (i.e., nerve root compression, spinal stenosis). This study used only categories 1–7, which are the only ones that may be determined at the initial visit.

2.5. Utilization of services

At baseline and at 3 months, each participant was also asked to report his or her utilization of all pain-related treatments and services (e.g., number of office visits, urgent care/ER visits, physical therapy visits), medications and their frequency of use, and complementary or alternative treatments (e.g., heating pads, yoga, acupuncture, chiropractic care). To confirm the validity of self-reported traditional treatment data, an extensive chart review examined utilization of services, referrals for other treatments (e.g., corticosteroid injections, pain clinic, orthopedic referrals), tests, prescribed medications, and prior histories of neck/back pain.

2.6. Statistical analysis

Descriptive characteristics were calculated for all outcome variables. To identify the independent predictors of chronic disability and to test the hypothesized model, a series of hierarchical multiple regression analyses employing path analytic procedures was conducted. Before regression analyses were performed, the data were examined to ensure the variables met the essential underlying assumptions of normality, linearity, homoscedasticity, and non-multicollinearity. The first model was a standard multiple linear regression analysis predicting pain and disability at 3 months from baseline measures. Next, multivariate regression models evaluated the unique contribution of each mediator/predictor variable to each subsequent predictor variable in the path analysis. Pearson correlations were conducted between potential covariates and the outcome variables. Potential covariates were variables theoretically or empirically associated with pain and depression in prior research. These included demographic factors (i.e., gender, ethnicity, marital status, socioeconomic status), pain-related factors (e.g., history of previous pain episodes, pain duration of current episode), and concurrent medications and medical treatments (see Section 2.5). Significant covariates ($p < .05$) were added to the regressions for each relevant path. Based on the outcomes of the path analysis where possible mediators were identified, the Aroian version of the Sobel test (Aroian, 1944/1947; Sobel, 1982) was applied to test whether the indirect effects of the paths via the mediators were significant.

3. Results

3.1. Summary characteristics

Table 2 provides correlations and means of the study variables at both time points. None of the predictor variables was highly intercorrelated at .90 or higher (Tabachnik and Fiddell, 2000). The criterion variables, pain and disability, were not correlated with each other or themselves at baseline. Pain intensity and disability were both quite elevated at baseline and remained above the clinical threshold at 3 month follow-up. Pain intensity mean scores at baseline ($M = 11.12$, SD = 4.34), and 3 months ($M = 7.24$, SD = 5.2) were within one standard deviation of healthy population scores ($M = 6.8$; Atkinson et al., 1997), however, the disability mean score at baseline for this study sample ($M = 33.06$,

Table 2
Correlations among demographic,predictor and criterion variables and means and standard deviations for each variable

Variables	1	2	3	4	5	6	7	8	9	10	11	Baseline		3 months	
												M	SD	M	SD
1 Pain dur (weeks)	–											–	–	–	–
2 Prev pain	−.14	–										–	–	–	–
3 Hispanic	−.19	.18	–									–	–	–	–
4 BL pain***	.02	−.00	−.09	–								11.1	4.3	7.2	5.2
5 BL disability***	−.29**	.29**	.26*	.00	–							33.1	18.7	17.3	15.4
6 Depression**	−.04	.15	.04	.24*	.48***	–						16.1	10.9	12.8	12.0
7 Pain Beliefs Permanence**	−.11	.18	.08	.13	.24*	.30**	–					−.9	.9	−.6	1.3
8 Pain Beliefs Constancy**	−.09	.16	.12	.11	.46***	.45**	.15	–				.2	1.1	−.7	1.3
9 Trauma exposure	−.05	.08	−.06	−.02	.20	.17	−.08	.17	–			11.2	8.5	–	–
10 3 months pain	.23*	−.00	.11	.15	.08	.40**	.23*	.18	.25*	–		–	–	–	–
11 3 months disability	.06	.29*	.05	.01	.46***	.55***	.52**	.25*	.17	.54**	–	–	–	–	–

Note: BL, baseline.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

SD = 18.68) was nearly equivalent to that of chronic pain patients (average pain duration = 4.9 years) with "low disability" ($M = 32.5$–34.5, SD = 8.42–9.32; Tait et al., 1990). Ninety-eight percent of the patients experienced at least one traumatic life event, and the mean number of different types of traumas experienced was 5.0, which is comparable to that of normative samples ($M = 6.4$) but lower than that of PTSD patients ($M = 10.7$). The mean number of traumatic life events reported at baseline was 11.45 (SD = 1.0), which is slightly lower than that of the normative population ($M = 16.8$) and significantly lower than the clinical mean for individuals with PTSD ($M = 32.0$; Kubany et al., 2000). The mean depression score of 16.14 at baseline, when the spine injuries were still acute, was equal to the suggested cutpoint score of 16 for probable major depression in the general population (Craig and Van Natta, 1978) while the follow-up mean score of 12.8 dropped below threshold for clinical depression but remained significantly higher than the general population mean of eight (Weissman et al., 1977). The only variable with means below those of a chronic pain population was pain beliefs. The constancy beliefs improved over time, such that people did not feel their pain was as constant at follow-up ($M = -.68$, SD = 1.25) as they did at baseline ($M = .18$, SD = 1.14). The means at both time points were below the chronic pain population mean of .47. In contrast, the pain permanence beliefs worsened over time (baseline $M = -.93$, SD = .89; follow-up $M = -.57$, SD = 1.25) but remained lower than the chronic pain population mean ($M = .06$).

3.2. Preliminary analyses

Paired *t*-tests, for dependent samples, assessed for significant differences between the means of the baseline scores and the 3-month follow-up scores for each variable using an alpha level that controlled for multiple comparisons ($p < .01$). Scores on each of the measures improved significantly between baseline and 3-month follow-up, except for the PBPI Permanence subscale. Scores worsened significantly on this scale assessing permanent and long-lasting pain beliefs.

Pearson correlations revealed that Hispanic ethnicity, shorter pain duration, and history of previous neck/back pain were significantly positively correlated with baseline disability. At follow-up, baseline pain duration was positively related to 3 month pain; and baseline pain intensity was significantly positively correlated with 3 month pain and history of previous neck/back pain episodes was significantly correlated with 3 month disability. Each of these covariates was included in the regression analyses for each relevant path in the model.

3.3. Path analysis

3.3.1. Path model of chronic pain

Summary results of the hierarchical multiple regression analysis predicting pain intensity at 3-month follow-up after entry of the four independent variables, baseline levels of pain and disability, and of the covariate, baseline pain duration, are depicted in Table 3. The model, with all variables included on the last step, accounted for 26% of the total variance in 3 month pain. Higher trauma exposure and more depressive symptomology at baseline independently and significantly predicted greater pain at follow-up, over and beyond initial pain duration.

3.3.2. Path model of chronic disability

The combined model postulated that cumulative trauma, more disability at baseline, more depressive symptoms, stronger beliefs that pain would be

Table 3
Multiple regression analysis for baseline predictors of 3 month pain intensity

Step	Predictor variable	*B*	SEB	β	sr^2	*p*	ΔR^2
1	Pain duration (# weeks at baseline)	.55	.21	.26	.05	.02	.05
2	Acute pain intensity (baseline)	.04	.12	.04	.00	ns	.04
	Acute disability (baseline)	.00	.03	−.10	−.00	ns	
3	Cumulative trauma (total # of events)	.12	.06	.22	.05	.03	.05
4	Depressive symptoms (baseline)	.12	.05	.28	.06	.02	.09
5	Pain permanence beliefs (baseline)	.86	.56	.16	.02	ns	.03
	Pain constancy beliefs (baseline)	−.02	.46	−.01	−.00	ns	

Note: $R^2 = .26$ ($N = 84$, $p = .002$).

permanent, stronger beliefs that pain is constant, together with 3 month pain, would predict more perceived disability at 3-month follow-up (see Table 4). After all the variables, including the covariate (history of previous neck/back pain) and baseline pain and disability, were entered, the combined model was highly significant $F(7, 76) = 14.25$, $p < .001$. Together the seven variables accounted for 58% of the variance in 3 month pain-related disability (see Table 4). History of neck or back pain did not contribute significantly to the total variance over and beyond the other variables in the model. While cumulative traumatic life events (at baseline) significantly predicted more pain at follow-up ($sr^2 = .05$, $p < .05$), it did not significantly improve the path model for chronic disability. Baseline depression, pain permanence beliefs, and chronic pain intensity were significant and independent predictors of 3 month disability, with the latter two variables explaining the greatest amount of variance in disability.

3.4. Mediation

Examination of Fig. 2 revealed the possibility of two mediators in the model: (a) baseline depression as a mediator between baseline disability and chronic pain disability and (b) pain permanence beliefs as a mediator between depression and chronic pain disability. A mediator is an intervening variable, such that in a case where variable *X*, which is assumed to affect *Y*, no longer affects variable *Y* after variable *M* (mediator) has been controlled, such that the original $X \rightarrow Y$ path is zero or reduced significantly in absolute size (Baron and Kenny, 1986). To determine whether baseline depression or pain permanence beliefs were mediators, the Aroian version of the Sobel test (Aroian, 1944/1947; Sobel, 1982) was applied, as recommended by Baron and Kenny.

Baseline depression was first tested as a possible mediator between baseline disability and 3 month disability. The direct effect of the path from baseline disability to 3 month disability was .22 while the indirect effect of the path when depression was included was .10. In this path, depression mediated the effect of baseline disability on 3 month disability ($z = 2.28$, $p < .05$, see Fig. 2). The pain permanence beliefs variable was also tested as a mediator in the baseline depression to chronic pain disability path. The original effect of the path from baseline depression to chronic pain disability was .49, while the mediated effect was .36. The reduction in the effect with inclusion of the hypothesized mediator variable was not statistically significant ($Z = 1.82$, n.s.); therefore, belief in pain permanence did not mediate the path between baseline depression and 3 month disability (Fig. 3).

4. Discussion

This study tested an etiological model of chronic pain and disability in patients recruited during the first few weeks of an acute pain episode. The model accounted for 26% of the variance in chronic pain and 58% of the variance in chronic disability.

Table 4
Multiple regression analysis for predictors of 3 month disability

Step	Predictor	*B*	SEB	β	sr^2	*p*	ΔR^2
1	Previous pain	4.51	2.59	.14	.02	.09	.07
2	Acute pain intensity (baseline)	−.45	.30	−.12	.01	ns	.16
	Acute disability (baseline)	.18	.08	.22	.03	.02	
3	Cumulative trauma (total # of events)	.05	.14	.03	.00	ns	.01
4	Depressive symptoms (baseline)	.37	.14	.27	.04	.008	.14
5	Pain permanence beliefs (baseline)	5.64	1.43	.32	.09	<.0001	.12
	Pain constancy beliefs (baseline)	−1.12	1.21	−.08	.00	ns	
6	Chronic pain intensity (3 months)	1.04	.28	.32	.08	<.0001	.08

Note: $R^2 = .58$ ($N = 84$, $p < .0001$).

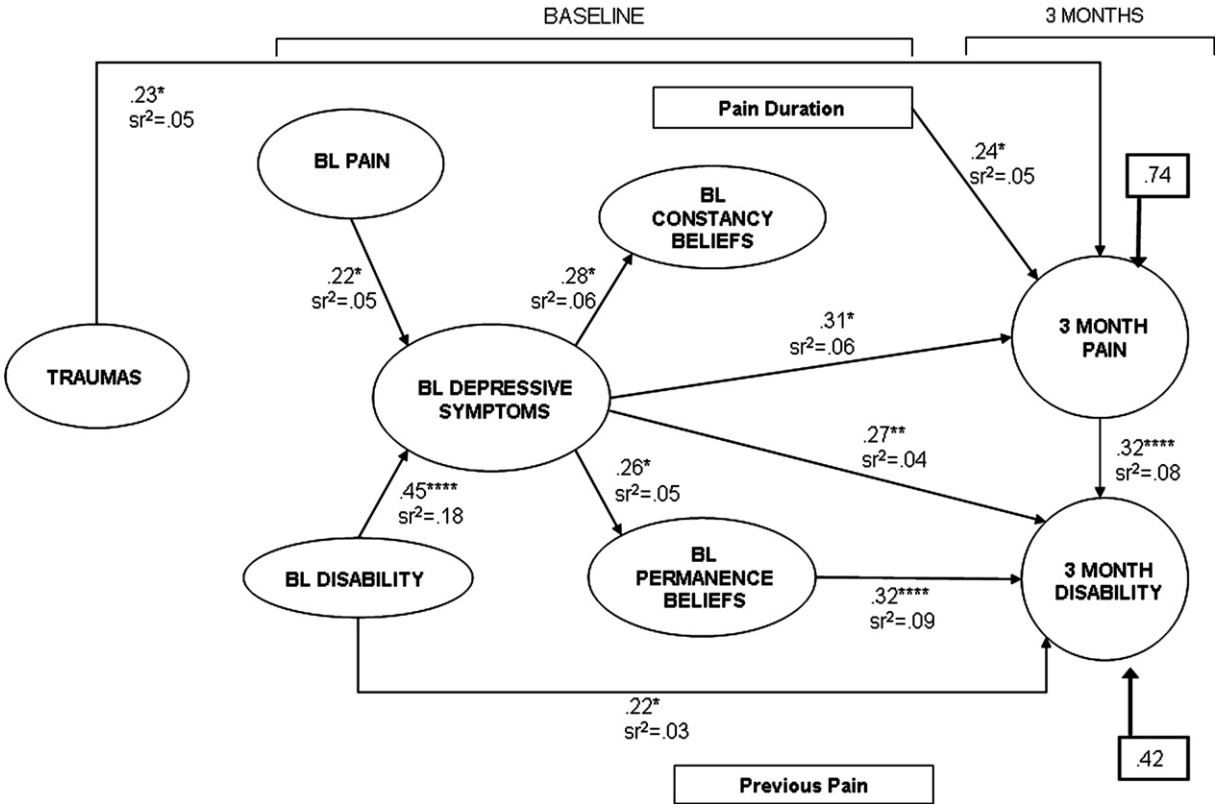

Fig. 2. Path analytic model of transition from acute to chronic pain and disability ($R^2 = .58$). *Note:* Standardized path coefficients (β) and squared semipartial correlations (sr^2) are reported for each path. "BL", baseline. Solid lines indicate $p < .05$ significant paths. Paths with associated p values $\geqslant .05$ are not depicted in this figure. $^{*}p < .05$. $^{**}p < .01$. $^{****}p < .0001$.

Baseline depressive symptoms and pain permanence beliefs were the most powerful predictors of chronic disability, uniquely accounting for nearly half of the variance predicted by the full model. Depressive symptoms and uncontrollability beliefs may lead to passive coping and avoidance, thereby exacerbating disability. With one exception (Gatchel et al., 1995) other prospective studies of acute pain support the relationship of initial depression to disability persistence (Hasenbring et al., 1994; Pincus et al., 2002).

These findings also highlight the bidirectional relationship between depression and disability. Acute disability directly and positively predicted disability at 3 months and was also indirectly related to this outcome via higher baseline depressive symptoms and more negative pain beliefs. Functional disability due to a pain condition may strain relationships or interfere with valued activities, leading to depression and associated motivational deficits, which then exacerbate disability.

Acute pain intensity did not directly predict 3 month disability, although it was indirectly, positively related to disability through initial pain permanence beliefs. This path is consistent with cognitive vulnerability and learned helplessness models of depression (e.g., Seligman, 1975; Beck et al., 1979) and indicates their potential applicability to pain populations. Numerous studies have now shown that baseline pain intensity, acute or chronic, does not necessarily predict subsequent disability (von Korff et al., 1993; Long et al., 1996; Epping-Jordan et al., 1998). Therefore, we concur with Atkinson

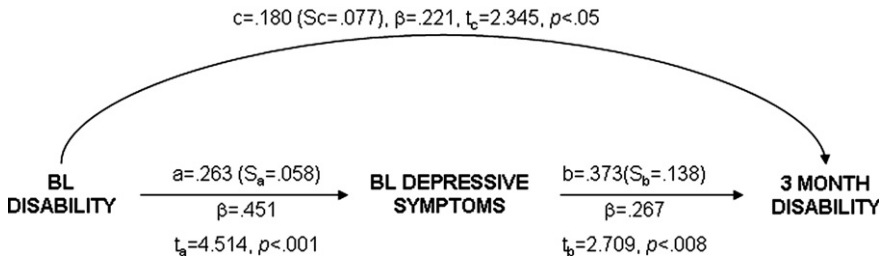

Fig. 3. Sobel test of mediation (Aroian version; Sobel, 1982; MacKinnon et al., 1995). Evaluation of baseline (BL) depressive symptoms as a mediator between baseline (BL) and 3 month disability ($t = 2.28$, $p < .05$).

et al. (1997) that acute disability is a more important contributor to chronicity than acute pain.

Surprisingly, cumulative trauma exposure did not predict more disability at either time point. Results are consistent with Yaari et al. (1999), who found Holocaust survivors reported more pain and depressive symptoms but similar levels of self-care and role functioning, relative to chronic pain controls. Proximal factors, including pain perceptions and emotional reactions, may supercede historical factors in predicting chronic disability.

Cumulative trauma exposure at baseline independently predicted greater pain intensity at 3 months over and beyond the other predictors but was not related to them. The magnitude of effect for trauma exposure was similar to that of depression. There may be a separate process not previously studied in the pain literature. Past traumas may influence physiologic systems or cognitive–affective processes that perpetuate pain, though they may not affect the acute sensation of pain.

In contrast to earlier studies (Linton, 1997), acute pain intensity did not directly predict 3 month pain but did predict chronic pain indirectly via baseline depression. Due to simultaneous measurement of pain and depression, the direction of causality is not established. Some evidence suggests depression is a risk factor for an acute onset of intense and/or disabling pain (Carroll et al., 2004; Larson et al., 2004).

Surprisingly, baseline pain beliefs did not predict chronic pain, although permanence beliefs positively predicted greater 3 month disability. In contrast negative pain beliefs reliably predict greater pain and disability in chronic pain patients (Jensen et al., 1991; Stroud et al., 2000;). It may be that other model variables, such as depression or trauma exposure, superceded pain beliefs in predictive value. Alternatively, pain beliefs may play a greater role in chronic pain, which is actually less controllable and more persistent than acute pain.

This cohort of acute neck and back pain patients is similar to acute and chronic pain patients in the pain literature and the national low back pain study (Long et al., 1996) with regard to race, marital status, age, and education (Craufurd et al., 1990; Potter and Jones, 1992; Burton et al., 1995). However, no demographic, medical history or treatment factor significantly predicted chronic pain or disability. These findings support the growing literature that contends the progression to chronic pain and disability is more dependent on psychosocial and occupational factors than on medical characteristics of the spinal condition (Valat et al., 1997).

4.1. Significance

This is one of only a few prospective studies to test a theoretically based model of the progression to chronic pain and disability. Results challenge some assumptions regarding chronic disability development and highlight the potential role of trauma history. Although previous studies have found more trauma or abuse histories in chronic pain patients than healthy controls, this is the first study to link trauma to the development of chronic pain in an acute sample. The fact that depressive symptoms were the strongest independent, positive predictor of subsequent pain and disability has implications for clinical practice. Screening for elevated depressive symptoms in patients presenting with acute pain may be an important tool in chronicity prevention efforts.

4.2. Limitations

The most significant limitation is the sample size of 84 participants, which was sufficient for a multiple regression with eight predictors, but fell short of the 10–20 participants per parameter recommended by statisticians for path analysis. This could result in less reliable and valid parameter estimates; path coefficients may be unstable, reflected by high standard errors, and their magnitudes could change with a new sample. Further, because of insufficient power to calculate an overall goodness of fit index, model fit could not be compared with plausible alternative models. Path coefficients are very sensitive to model specification, including possible omitted or extraneous variables and are dependent on sample characteristics. Therefore, the findings are preliminary and in need of replication with a larger sample. The current model was, however, grounded in theory and research and accounted for substantial variance in pain and disability; results are suggestive and provide an impetus for further research and theory development.

Another limitation was reliance on self-report measures of primary variables, which are subject to recall and social desirability biases. Further, although all measures were internally consistent, a few had less than ideal test–retest reliability. Including in vivo assessment of pain behaviors would have provided additional objective measurement of pain (Keefe and Block, 1982; Prkachin et al., 2002). Assessment of past traumas was retrospective; obtaining independent confirmation of lifetime traumas represents a significant methodological challenge. Emotional traumas are, however, more easily remembered than neutral events (Christianson and Loftus, 1991; Christianson, 1992). Self-reports of potential treatment covariates were validated by chart review examining service utilization, treatment referrals, medications, and prior histories of neck/back pain.

Because of higher dropout among those with greater pain constancy beliefs, these data may underestimate the relationship of constancy beliefs to other model variables. There are also some limits to external generalizability. Participants were recruited from a referral-based clinic within one medical group and may therefore comprise treatment-seekers with more serious

conditions. The generalizability of this study is limited to those of similar demographic characteristics, i.e., college educated, Caucasian adults, in Southern California with particular treatment-seeking preferences, who may be less likely to characterize their pain as constant.

4.3. Future directions

The most important future research priority is to replicate these findings in a longitudinal study using a larger sample of acute pain patients, sufficient to estimate goodness of fit. Extension of this research with diverse pain samples and using multiple assessment modalities across longer follow-up periods is also recommended.

Future studies should also focus on the role of trauma in the perpetuation of pain and disability following an acute pain incident. Cumulative trauma exposure did not predict chronic pain through depression and negative pain beliefs, or through acute pain intensity, as hypothesized. Future research should explore alternative potential mediators of this effect. Neurobiological research (van der Kolk, 1996, 2001; Siegel, 2001) suggests that trauma can produce long-standing negative changes in brain structures and processes, which affect information processing and emotional regulation The anterior cingulate cortex is activated during both physical pain and social distress, suggesting possible common neurobiological pathways (Eisenberger et al., 2006). Studies suggest trigger points located in the intrafusal muscle fibers, which are sympathetically mediated and innervated by psychological stress may perpetuate muscle pain (Hubbard and Berkoff, 1993; McNulty et al., 1994). These theories may bridge gaps unexplained by the current study.

Anxiety and its effects on information processing may also link past traumas to pain chronicity. Traumas are encoded in memory as fear structures (Foa and Kozak, 1986) that are easily activated and promote hypervigilance for threat (Chemtob et al., 1988). Anxiety-related constructs, such as fear-avoidance (McCracken et al., 1992; Waddell et al., 1993) or passive coping (Brown and Nicassio, 1987; Keefe et al., 1989), are positively correlated with pain behavior and disability. Klenerman and colleagues (1995) demonstrated that fear-avoidance behaviors accounted for 66% correct classification of acute back pain injuries that became chronic at 12 months. Generalization of anxiety reactions associated with past traumas could plausibly impede recovery from an acute pain incident.

5. Conclusions

This study was an initial test of a new, theoretically derived model of the development of persistent pain and disability following an acute pain incident. The findings contribute to the growing body of empirical studies demonstrating that the transition to chronic pain syndromes is more a function of serious life stressors and cognitive–affective factors than medical factors.

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