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# Stage of chronicity and treatment response in patients with musculoskeletal injuries and concurrent symptoms of depression ☆

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**Abstract**

The present study examined the relation between stage of chronicity and treatment response in patients with work-related musculoskeletal conditions and concurrent depressive symptoms. Also of interest was the role of reductions in pain severity, catastrophic thinking and fear of movement/re-injury as mediators of the relation between chronicity and treatment response. A sample of 80 individuals (38 women, 42 men) with a disabling musculoskeletal pain condition and concurrent depressive symptoms participated in the research. Individuals with work absence of less than 6 months (range 12–26 weeks) were classified as early chronic ($N = 40$), and individuals with work absence greater than 6 months (range 27–52 weeks) were classified as chronic. Both groups were matched on sex, age (±2 years) and severity of depressive symptoms. All participants were enrolled in a 10-week community-based disability management intervention. The early chronic group showed significantly greater reduction in depressive symptoms, and pain symptoms, than the chronic group. Regression analyses revealed that pain reduction, but not catastrophic thinking or fear of movement/re-injury, mediated the relation between chronicity and improvement in depressive symptoms. The results highlight the importance of early detection and treatment of depressive symptoms, given that treatment response decreases over time. The results also suggest that reductions in depressive symptoms might be a precondition to the effective reduction of pain symptoms in this population. Discussion addresses the factors that might contribute to treatment resistance as the period of disability extends over time.


*Keywords:* Pain; Disability; Depression; Catastrophizing; Rehabilitation; Return to work; Musculoskeletal pain; Chronic pain; Fear of movement; Work disability

## 1. Introduction

Numerous investigations have revealed that the duration of pain-related disability is a marker for poor rehabilitation outcomes (Marhold et al., 2001; Waddell et al., 2003; Dunn and Croft, 2006). What remains unclear is why chronicity is associated with poor rehabilitation outcomes. The paucity of research addressing this question has limited conceptual advance in this area, and has likely impacted negatively on the development of effective intervention programs for individuals with long-standing musculoskeletal conditions.

Depression has been identified as a determinant of poor rehabilitation outcomes in individuals with musculoskeletal conditions (Druss et al., 2000; Sullivan and

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Stanish, 2003; Lotters et al., 2006; Sullivan et al., in press). However, a reliable relation between depressive symptom severity and the duration of pain-related disability has not been observed (Campbell et al., 2003; Sullivan et al., 2006). As such, the severity of depressive symptoms is an unlikely candidate as a variable underlying the relation between chronicity and poor rehabilitation outcomes.

It is possible, however, that progressive treatment resistance in depressive symptoms might account for the relation between chronicity and poor rehabilitation outcomes. For example, research has shown that treatment response to pharmacological and psychological interventions for depression decreases as a function of symptom duration (Sotsky et al., 1991; Hirshfeld et al., 1998; Detke et al., 2004). It is possible that depressive symptoms might become more treatment resistant as the duration of pain-related disability extends over time. No research has yet to address the relation between chronicity and treatment-related reductions in depressive symptoms.

Diathesis-stress formulations have been invoked to account for the relation between depression and pain-related outcomes (Banks and Kerns, 1996; Campbell et al., 2003). These models suggest that depressive symptoms arise as a joint function of vulnerability factors and vulnerability-relevant contextual factors. Proceeding from a diathesis-stress formulation, treatment-related reductions in depressive symptoms might be expected to be determined in part by changes in vulnerability factors and changes in vulnerability-relevant contextual factors (Banks and Kerns, 1996; Turk, 2002; Sullivan et al., 2006). It is possible that vulnerability factors or vulnerability-relevant contextual factors might also become resistant to change over time and compromise the magnitude of treatment effects on depressive symptoms.

One objective of the present research was to assess the relation between chronicity and rehabilitation outcome in individuals with musculoskeletal conditions and concurrent depressive symptoms. Rehabilitation outcome was addressed in terms of reductions in pain symptoms, reductions in symptoms of depression and return to work. A second objective of this research was to examine the role of changes in vulnerability factors and changes in vulnerability-relevant contextual factors as mediators of the relation between chronicity and depressive symptom reduction. The second objective was intended to shed light on why depressive symptoms might become treatment resistant as the period of disability extends over time. On the basis of previous research, pain catastrophizing and fear of movement/re-injury were chosen as vulnerability factors and pain severity was chosen as a vulnerability-relevant contextual factor (Sullivan et al., 2006).

## 2. Methods

### 2.1. Participants

A sample of 80 individuals (38 women, 42 men) with a disabling musculoskeletal pain condition participated in the research. All participants had sustained a work-related back injury and were currently receiving WCB wage replacement benefits. Individuals with work absence of less than 6 months (range 12–26 weeks) were classified as early chronic ($N = 40$), and individuals with work absence greater than 6 months (range 27–52 weeks) were classified as chronic. Data were drawn from a larger sample of patients enrolled in a community-based secondary prevention program and were matched on sex, age (±2 years) and Beck Depression Inventory-II (Beck et al., 1996) scores (±2). All participants had Beck Depression Inventory-II scores of 16 or greater as a condition of participation in the treatment program. The mean age of the sample was 40.4 years with a range of 25–58 years.

### 2.2. Procedure

Participants in this research were enrolled in the Pain-Disability Prevention (PDP) Program (Sullivan et al., 2005). The PDP Program is a 10-week standardized intervention aimed at minimizing psychological barriers to rehabilitation progress in individuals suffering from painful musculoskeletal conditions. The patient attends weekly sessions with a psychologist trained in the delivery of the PDP Program. This intervention differs from traditional pain management programs with its primary focus on the reduction of psychological barriers to return-to-work. Activity structuring, activity planning, graded activity involvement, cognitive restructuring and problem-solving are used as primary tools to reduce depressive symptoms and depression-related disability. The PDP Program can best be conceptualized as a life-role reintegration intervention where the objective is to assist clients in resuming participation in life role activities, in spite of the fact that their pain still persists. Standardized assessments of the PDP Program include measures of pain severity, catastrophic thinking, fear of movement/re-injury and depression completed at pre-treatment (Week 1), mid-treatment (Week 4) and post-treatment (Week 9).

The PDP Program was provided by 21 psychologists who were part of a regionally distributed network of community-based practitioners in the province of Nova Scotia, Canada (for more information on the PDP Program and the network of providers, see www.pdp-pgap.com). The PDP Program was provided as an addition to usual medical management and physical therapy. The addition of the PDP Program to traditional rehabilitation treatment is intended to establish 'virtual' multidisciplinary teams at the community-based level (Sullivan et al., 2005). Community-based psychologists were asked to forward copies of PDP Program assessment results (identified only by claim number) to our research centre. The PDP Program assessment results were then linked to WCB administrative data. Due to constraints in the nature of data that could be accessed from the files of the community-based practitioners, information concerning mental health diagnoses and symptom/treatment history was not available for analysis.

Approximately, half ($N = 45$) of participants in this study were also part of the participant sample described in Sullivan et al. (2005). The present sample differs from that described in Sullivan et al. (2005) in that a BDI-II score greater than 16 was a condition of participation in the present study but was not a condition of participation in Sullivan et al. (2005).

### 2.3. Measures

#### 2.3.1. Pain severity

The McGill Pain Questionnaire (MPQ; Melzack, 1975) was used as a measure of pain severity. Respondents endorsed adjectives that best described their pain experience. The Pain Rating Index (PRI) is a weighted sum of all adjectives endorsed, and has been shown to be a reliable and valid index of an individual's pain experience (Turk et al., 1985).

#### 2.3.2. Depression

The Beck Depression Inventory-II (BDI; Beck et al., 1996) was used to measure severity of depressive symptoms. The BDI-II consists of 21 items describing various symptoms of depression. Respondents endorsed phrases that best described how they had been feeling during the past two weeks. The BDI-II has been shown to be a reliable and valid index of depressive symptoms in chronic pain patients (Sullivan and Stanish, 2003; Vowles et al., 2004; Poole et al., 2006).

#### 2.3.3. Catastrophizing

The Pain Catastrophizing Scale (PCS; Sullivan et al., 1995) was used as a measure of catastrophic thinking related to pain. Respondents rated the frequency with which they experienced each of 13 different thoughts and feelings when in pain. The PCS has been shown to have high internal consistency (coefficient alpha = .87), and to be associated with heightened pain, pain behavior and disability (Sullivan et al., 2005).

#### 2.3.4. Fear of movement/re-injury

The Tampa Scale for Kinesiophobia (TSK; Kori et al., 1990) was used as a measure of fear of movement and re-injury associated with pain. Respondents indicated their level of agreement with each of 17 statements reflecting worries or concerns about the consequences of participating in physical activity. The TSK been shown to be internally reliable (coefficient alpha = .77; Vlaeyen et al., 1995), and to be associated with various indices of disability (George et al., 2003).

#### 2.3.5. Medication use

Respondents were asked to indicate the different medications they had been prescribed for their symptoms. For the purpose of the present study, the use of different classes of medication was dichotomized as follows: NSAIDs (y/n), opiates (y/n), hypnotics (y/n), anxiolytics (y/n), low dose antidepressants (y/n), and therapeutic dose antidepressants (y/n).

#### 2.3.6. Return to work

Return to work status was assessed 4 weeks following termination of the PDP Program. Return to work information and claim status were obtained from WCB files. Participants were classified as having returned to work if they had returned to full time pre-injury employment or alternate employment, and their claim was closed. All other participants were classified as not having returned to work.

## 3. Results

### 3.1. Sample characteristics

Sample selection procedures provided matched groups on age, sex and BDI-II scores. As expected there were significant differences in the duration of work absence, with the early chronic group averaging 15.2 weeks of work disability and chronic group averaging 33.3 weeks of work disability, $t(78) = 10.6$, $p < .001$. Early chronic and chronic groups did not differ significantly on the use of NSAIDs, $\chi^2 = 2.0$, ns, opiates, $\chi^2 = .80$, ns, hypnotics, $\chi^2 = .28$, ns, anxiolytics, $\chi^2 = 1.4$, ns, low dose antidepressants, $\chi^2 = 2.8$, ns, or therapeutic dose antidepressants, $\chi^2 = 1.2$, ns (see Table 1).

### 3.2. Rehabilitation outcomes

As expected, the early chronic and chronic groups differed in terms of return-to-work rates. In the early chronic group, 67% of participants returned to work, compared to 37% in the chronic group, $\chi^2 = 7.2$, $p < .01$.

The trajectory of recovery in depression scores for the early chronic and chronic groups is shown in Fig. 1. A two-way (Level of chronicity × Time of assessment) repeated measures analysis of variance (ANOVA) revealed a significant main effect for Time, $F(2, 156) = 22.6$, $p < .001$. The main effect for Level of chronicity did not attain statistical significance, $F(1, 78) = 3.2$, $p < .07$. A significant interaction was also obtained, $F(2, 156) = 8.9$, $p < .001$. Although both groups were comparable on initial depression scores, the response to treatment for the chronic group (13% reduction in BDI-II scores) was more modest than for

Table 1
Sample characteristics at pre-treatment assessment

| | Stage of chronicity | | |
|---|---|---|---|
| | Early chronic | Chronic | *p* |
| Sample size | $N = 40$ | $N = 40$ | |
| (Men/women) | 21/19 | 21/19 | ns |
| Age (years) | 40.1 (8.2) | 41.0 (7.5) | ns |
| Work absence | 15.2 weeks (7.3) | 33.3 weeks (7.8) | .001 |
| BDI-II-T1 | 24.5 (8.1) | 24.0 (6.8) | ns |
| NSAIDs | 35 | 30 | ns |
| Opiates | 20 | 24 | ns |
| Hypnotics | 10 | 8 | ns |
| Anxiolytics | 10 | 15 | ns |
| Low dose antidepressants | 9 | 16 | ns |
| Therapeutic dose antidepressants | 6 | 10 | ns |

*Note.* BDI-II, Beck Depression Inventory-II. Scores on the BDI-II were obtained at pre-treatment assessment. Numbers in parentheses are standard deviations.

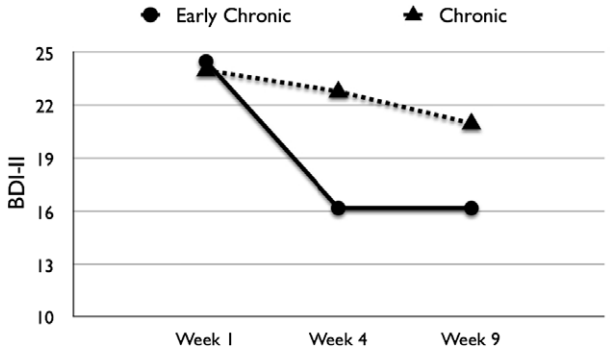


Fig. 1. Changes in depressive symptoms as function of level of chronicity. BDI-II raw scores for participants in the early chronic and chronic groups for assessments conducted pre-treatment (Week 1), mid-treatment (Week 4) and treatment termination (Week 9).

the early chronic group (33% reduction in BDI-II scores), $p < .05$.

Reductions in pain scores across the 10-week intervention program for the early chronic and chronic groups are displayed in Fig. 2. A two-way (Level of chronicity × Time of assessment) repeated measures ANOVA on pain severity scores (MPQ-PRI) revealed a significant main effect for Time, $F(2,156) = 8.6$, $p < .001$, and a significant interaction, $F(2,156) = 9.7$, $p < .001$. Tests of simple effects revealed that the early chronic group obtained significantly lower MPQ scores at Week 9 assessment, $p < .05$, but the two groups did not differ significantly at Week 1 and Week 4 assessments. The early chronic group showed a moderate decrease in pain severity through the course of the treatment program (23% decrease) while the chronic group showed a slight increase in pain severity (4% increase).

### 3.3. Do changes in depression account for the relation between chronicity and rehabilitation outcomes?

Two important rehabilitation outcomes include pain reduction and return to work. In order to examine the role of change in depression as a mediator of the relation between chronicity and rehabilitation outcomes, change scores (Week 1–Week 9) were computed for pain severity, and depression. Mediation is established when the independent variable (e.g., level of chronicity) is associated with both the dependent variable (e.g., rehabilitation outcome) and the hypothesized mediator (e.g., change in depression) (Baron and Kenny, 1986). Complete mediation is established when the relation between the independent and dependent variables is no longer significant, after variance associated with the mediator has been controlled. In order to be considered as a potential mediator, a variable must show a significant association with both independent and dependent variables (Holmbeck, 1997).

Change in BDI-II scores was significantly correlated with change in pain, $r = .44$, $p < .01$. Furthermore, reduction in BDI-II scores was significantly greater for participants who returned to work ($M_{(\text{BDI-II T1–BDI-II T3})} = 7.6$; SD = 5.7) than for participants who did not return to work ($M_{(\text{BDI-II T1–BDI-II T3})} = 2.6$; SD = 9.8), $t(78) = 2.8$, $p < .01$. As such, the conditions for analysis of mediation for change in depressive symptoms, change in pain severity, return to work and level of chronicity were met.

As shown in Table 2, the results of a logistic regression indicated that chronicity was a significant predictor of return to work, Nagelkerke $R^2 = .12$, $p < .001$. A second logistic regression was conducted where change in depressive symptoms was entered in Step 1 of the analysis, and level of chronicity was entered in Step 2. The proportion of variance accounted for by level of chronicity was reduced from 12% to 4%. Nevertheless, level of chronicity significantly predicted return to work status even when controlling for change in depressive symptoms. These results suggest that while change in depressive symptoms is a significant determinant of return to work, change in depressive symptoms only

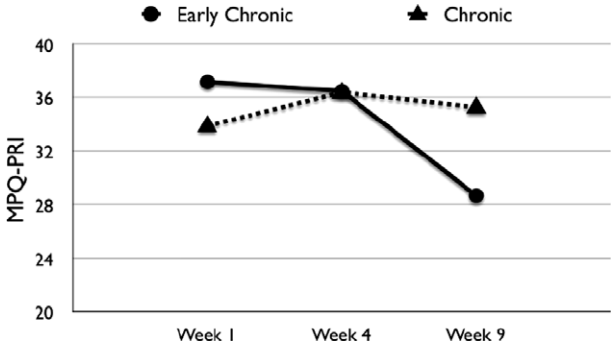


Fig. 2. Changes in pain severity as function of level of chronicity. MPQ-PRI raw scores for participants in the early chronic and chronic groups for assessments conducted pre-treatment (Week 1), mid-treatment (Week 4) and treatment termination (Week 9).

Table 2
Logistic regression examining mediators of the relation between chronicity and rehabilitation outcome (return to work)

| Dependent variable = Return to work | $\Delta\chi^2$ | $R^2_{\text{change}}$ | OR | CI |
|---|---|---|---|---|
| *Regression 1: chronicity and treatment response (return to work)* | | | | |
| Step 1 | | | | |
| Level of chronicity | 7.3 | .12 | .28** | .11–.72 |
| *Regression 2: the mediating role of change in depression* | | | | |
| Step 1 | | | | |
| BDI-II-ch | 7.6 | .12 | 1.10** | 1.0–1.1 |
| Step 2 | | | | |
| Level of chronicity | 4.2 | .04 | .36* | .14–.95 |

*Note.* $N = 80$. $\Delta\chi^2$ = Nagelkerke $R^2$; OR = Odds ratio; CI = 95th percentile confidence interval.

* $p < .05$.

** $p < .01$.

Table 3
Regression analyses examining mediators of the relation between chronicity and change in pain severity

| Dependent variable Change in MPQ-PRI | *B* | $R^2_{change}$ | $F_{change}$ | *p* |
|---|---|---|---|---|
| *Regression 1: chronicity and treatment response (reductions in pain)* | | | | |
| Step 1 | | | | |
| Level of chronicity | −.43** | .18 | 17.8 (1,78) | .001 |
| *Regression 2: the mediating role of change in depression scores* | | | | |
| Step 1 | | | | |
| BDI-II-ch | .34** | .20 | 18.9 (1,78) | .001 |
| Step 2 | | | | |
| Level of chronicity | −.33** | .09 | 11.1 (1,77) | .001 |

*Note*. $N = 80$. Standardized Beta weights are from the final regression equation.
** $p < .01$.

partially mediates the relation between level of chronicity and return to work.

Hierarchical multiple regression analyses were conducted to examine the role of change in depressive symptoms in mediating the relation between level of chronicity and pain reduction. As shown in Table 3, when level of chronicity was the only variable in the analysis, it accounted for 18% of the variance in change in pain symptoms, $F(1,78) = 17.8$, $p < .001$. In a second regression analysis, change in depressive symptoms was entered in Step 1 of the analysis and accounted for 20% of the variance in pain reduction, $F(1,78) = 18.9$, $p < .001$. When controlling for change in depression, the Beta weight for level of chronicity decreased from −.43 to −.33. Nevertheless, the contribution of level of chronicity to the prediction of pain reduction remained significant. These results suggest that change in depressive symptoms partially mediates the relation between level of chronicity and pain reduction.

### 3.4. Mediators of the relation between level of chronicity and reductions in depressive symptoms

Table 4 shows the scores on measures of pain catastrophizing and fear of movement/re-injury at all three test periods over the course of the treatment program (Week 1, Week 4, Week 9). A two-way (Level of chronicity × Time of assessment) repeated measures ANOVA on pain catastrophizing scores (PCS) yielded main effects for Time, $F(2,156) = 33.3$, $p < .001$, and for Level of chronicity, $F(1,78) = 9.8$, $p < .01$. The interaction term was not significant, $F(2,56) = .37$, ns.

A two-way (Level of chronicity × Time of assessment) repeated measures ANOVA on fear of movement/re-injury scores (TSK) revealed main effects for Level of chronicity = 4.0, $p < .05$, Time, $F(2,156) = 12.8$ $p < .001$, and a significant interaction, $F(2,156) = 11.8$, $p < .01$. Tests of simple effects revealed that the early chronic group obtained significantly lower TSK scores at the final evaluation (Week 9) than the chronic group, $p < .05$, but the two groups did not differ on TSK scores on the first two evaluations (Week 1, Week 4).

Change scores (Week 9–Week 1) were computed for pain catastrophizing and fear of movement/re-injury. Table 5 presents the correlations among changes in pain, depressive symptoms, pain catastrophizing and fear of movement/re-injury. Changes in pain severity, pain catastrophizing and fear of movement/re-injury were significantly correlated with changes in depressive symptoms. As such, the conditions for analyses addressing mediators of the relation between level of chronicity and reductions in depressive symptoms were met.

As shown in Table 6, Level of chronicity was a significant predictor of change in depressive symptoms (Regression 1: Beta −.29, $p < .01$). When change in pain severity was entered in the first step of Regression 2, Level of chronicity was no longer a significant predictor of change in depressive symptoms (Beta = −.12, ns). These results of this analysis indicate that change in pain severity mediates the relation between level of chronicity and reduction in depressive symptoms.

The results of Regression 3 revealed that a significant proportion of the variance in change in depressive symptoms was accounted for by change in pain catastrophizing (Beta = .41, $p < .001$). However, Level of chronicity (Beta = −.26, $p < .01$) remained a significant predictor of change in depressive symptoms even when controlling for change in pain catastrophizing. These results indicate that pain catastrophizing does not mediate the relation between level of chronicity and reduction in depressive symptoms.

The results of Regression 4 revealed that a significant proportion of the variance in change in depression was explained by change in fear of movement/re-injury,

Table 4
Scores on measures of pain catastrophizing and fear of movement/re-injury through the course of treatment

| | Stage of chronicity | | | | | |
|---|---|---|---|---|---|---|
| | Early chronic | | | Chronic | | |
| | Week 1 | Week 4 | Week 9 | Week 1 | Week 4 | Week 9 |
| PCS | 25.7 (10.4) | 20.5 (12.8) | 18.7 (14.2) | 32.4 (8.5) | 28.6 (9.5) | 26.5 (11.1) |
| TSK | 42.4 (8.8) | 40.8 (8.5) | 36.3 (9.7) | 43.6 (6.8) | 43.3 (8.0) | 43.3 (8.7) |

*Note*. PCS, Pain Catastrophizing Scale; TSK, Tampa Scale for Kinesiophobia. Numbers in parentheses are standard deviations.

Table 5
Correlations among changes in pain severity, pain catastrophizing and fear of movement/re-injury

| | MPQ-ch | BDI-II-ch | PCS-ch |
|---|---|---|---|
| MPQ-ch | | | |
| BDI-II-ch | .44** | | |
| PCS-ch | .29** | .45** | |
| TSK-ch | .27* | .24* | .48** |

*Note.* $N = 80$. Change scores were computed by subtracting scores obtained at the Week 9 evaluation from the Week 1 evaluation.
* $p < .05$.
** $p < .01$.

when considered alone in the analysis, $R^2 = .05$, $F(1, 78) = 4.1$, $p < .05$. However, the Beta weight for fear of movement/re-injury was no longer significant when Level of chronicity was entered in Step 2. Level of chronicity (Beta = −.24, $p < .05$) remained a significant predictor of change in depression even when controlling for fear of movement/re-injury. The results indicate that fear of movement/re-injury does not mediate the relation between level of chronicity and reduction in depressive symptoms.

## 4. Discussion

The results of the present study are consistent with previous findings indicating that rehabilitation outcomes are more modest as the period of disability extends over time (Waddell, 1998; Dunn and Croft, 2006). Even when matched on sex, age and severity of depressive symptoms, and enrolled in the same standardized intervention program, individuals with work absence greater 6 months were 50% less likely to return to work following treatment than individuals with work absence less than 6 months (Frank et al., 1996; Waddell et al., 2003).

Table 6
Regression analyses examining mediators of the relation between chronicity and treatment response (BDI-II-T1–BDI-II-T3)

| Dependent variable = Change in BDI-II | *B* | $R^2_{change}$ | $F_{change}$ | *p* |
|---|---|---|---|---|
| *Regression 1: chronicity and treatment response* | | | | |
| Step 1 | | | | |
| Level of chronicity | −.29** | .08 | 7.1 (1,78) | .01 |
| *Regression 2: the mediating role of change in pain severity* | | | | |
| Step 1 | | | | |
| MPQ-ch | .39** | .19 | 18.9 (1,78) | .001 |
| Step 2 | | | | |
| Level of chronicity | −.12 | .01 | 1.1 (1,77) | .29 |
| *Regression 3: the mediating role of change in pain catastrophizing* | | | | |
| Step 1 | | | | |
| PCS-ch | .41** | .18 | 17.5 (1,78) | .001 |
| Step 2 | | | | |
| Level of chronicity | −.26** | .07 | 6.8 (1,77) | .01 |
| *Regression 4: the mediating role of change in fear of movement/re-injury* | | | | |
| Step 1 | | | | |
| TSK-ch | .12 | .05 | 4.1 (1,78) | .04 |
| Step 2 | | | | |
| Level of chronicity | −.24* | .05 | 3.8 (1,77) | .05 |

*Note.* $N = 80$. Standardized Beta weights are from the final regression equation.
* $p < .05$.
** $p < .01$.

The present study extends previous research by showing that depressive symptoms become more treatment resistant as the period of work disability extends over time. Participants with duration of work disability less than 6 months showed a 33% reduction in the severity of depressive symptoms while participants with work disability greater than 6 months showed only a 13% reduction in the severity of depressive symptoms. Analyses revealed that reduction in depression scores partially mediated the relation between level of chronicity and rehabilitation outcome, whether addressed in terms of pain reduction or return to work. It appears therefore that one of the reasons why individuals with longer duration of work disability show more modest gains in rehabilitation is that their depressive symptoms become more treatment resistant over time.

In this study, two groups of participants were chosen from a larger pool of injured workers who were matched on age, sex, and severity of depressive symptoms. Matching groups in this manner permitted control over known correlates of treatment response thus permitting more direct examination of the clinical mediators of interest. On the basis of previous research on predictors of depressive symptoms in patients with musculoskeletal conditions, pain severity, pain catastrophizing and fear of movement/re-injury were chosen as potential mediators of the relation between chronicity and treatment response for depressive symptoms (Burns et al., 2003; Campbell et al., 2003; Sullivan et al., 2006).

Regression analyses revealed that reductions in pain catastrophizing and fear of movement/re-injury were significantly associated with reductions in depression but neither mediated the relation between level of chronicity and reduction in depressive symptoms. Comparable reductions in pain catastrophizing were observed in both groups. However, patients in the chronic group obtained higher initial and post-treatment scores on pain catastrophizing. At post-treatment, patients in the chronic group also scored higher on fear of movement/re-injury than patients in the early chronic group. These findings suggest that the absolute score on measures of catastrophizing and fear of movement/re-injury at post-treatment might be more important determinants of treatment outcome than the magnitude of change in scores.

Only reductions in pain severity mediated the relation between level of chronicity and reduction in depressive symptoms. There are different processes by which pain reduction might influence treatment response in pain

patients with depressive symptoms. There is a natural course of recovery from musculoskeletal conditions such that pain symptoms decrease in intensity over the first few months following injury and then plateau (Waddell, 1998; Linton et al., 2005; Von Korff and Miglioretti, 2005; Dunn and Croft, 2006). It is possible that depressive symptoms might be more likely to respond to treatment when pain severity decreases over time. However, the pattern of changes in pain and depressive symptoms is not entirely consistent with the view that a natural course of recovery in pain accounted for the differential rates of reduction in depressive symptoms in the early chronic and chronic groups. In the early chronic group, participants were work disabled for at least three months. Three months would be considered the upper limit of a natural trajectory of recovery for pain symptoms following a soft tissue injury (Waddell, 1998; Von Korff and Miglioretti, 2005). Furthermore, for participants in the early chronic group, pain symptoms showed greatest reduction in the last 5 weeks of the program, while depressive symptoms showed greatest reduction in the first 4 weeks of the program. In other words, depressive symptoms changed before pain symptoms.

It is possible that a recursive process involving bidirectional relations between depression and pain might help explain the pattern of findings. Depressive symptoms might have reacted positively to the behavioural activation component of the program, and in turn, reduction in depressive symptoms might have contributed to a reduction in pain symptoms. The reduction in pain symptoms might have been interpreted by the individual as a sign of improvement, perhaps contributing to further improvement or maintenance of treatment gains.

For patients in the chronic group, depressive symptoms were modestly reduced through the course of the intervention, and pain symptoms remained essentially unchanged. A treatment history characterized by successive treatment failures might be an important factor contributing to treatment resistance in individuals with more chronic symptoms. Given the importance of expectancies in determining treatment outcomes, more pessimistic outcome expectancies might have compromised the potential for reduction in depressive symptoms (Schultz et al., 2004).

On the basis of the present findings, it could be suggested that pain reduction might be an important component of the successful treatment of depressive symptoms associated with pain. However, reducing pain severity in depressed patients with long-standing musculoskeletal pain poses particular challenges. Opioids have been associated with heightened levels of depressive symptoms suggesting that, at least in some patients, opioids might contribute to a lowering of mood (Ciccone et al., 2000). Depressed mood has also been shown to interfere with the efficacy of opioids (Wasan et al., 2005). In terms of the medications currently available for the management of persistent musculoskeletal pain, it is unclear whether treating pain symptoms will yield meaningful reductions in depressive symptoms.

The findings of the present study highlight the importance of early detection and treatment of depressive symptoms in individuals with painful musculoskeletal conditions. Depression continues to be under-detected and under-treated in individuals with musculoskeletal conditions (Sullivan and Robinson, 2006). Assessment of depression is not a routine part of primary medical care. As well, insurers are often reluctant to refer clients for psychiatric or psychological treatment due to concerns about increases in claim costs. However, the present findings suggest that claim costs might only increase when referral for treatment of depression is delayed until chronicity has been established. Claim costs might actually be reduced if treatment for depressive symptoms is implemented early. Delays in referring clients for assessment and treatment of depressive symptoms might mean missing a window of opportunity for effective intervention on depressive symptoms.

Surprisingly, there have been few clinical trials examining the efficacy of antidepressants in depressed patients with musculoskeletal pain (Sullivan et al., 1992; Sullivan and Robinson, 2006). Although antidepressants are frequently prescribed for individuals with persistent pain, the target of treatment is often more likely to be sleep or pain symptoms as opposed to depressive symptoms (Polatin and Dersch, 2004; Mico et al., 2006; Sullivan and Robinson, 2006). As indicated by the present findings, many patients with high levels of depressive symptoms are prescribed antidepressants at doses below the therapeutic range (Polatin and Dersch, 2004). More research is needed in this area in order to provide more effective treatment of patients with comorbid depression and pain.

A number of limitations impact on the interpretation of the findings of the present study. First, chronicity was defined in terms of duration of work absence and not in terms of duration of depressive symptoms. Beyond BDI-II responses indicating that symptoms were present for at least two weeks, no information was available about the date of onset of depressive symptoms. As such, it is unclear whether duration of work absence or duration of depressive symptoms was the key determinant of differences in treatment response.

Another limitation is that depression was operationalised as a high score on a self-report measure of depressive symptoms as opposed to diagnostic interview. To date, the bulk of research on depression associated with musculoskeletal conditions has been conducted with self-report measures (Geisser et al., 1997). Considerable research attests to the validity of the BDI-II as an index of depressive symptoms associated with pain (Poole et al., 2006).

However, there is research to suggest that self-report measures of depressive symptoms have high sensitivity for diagnoses of Major Depressive Disorder (MDD) but low specificity (Bishop et al., 1993; Geisser et al., 2000). Future research will need to address with greater precision, the assessment of duration of depressive symptoms and the degree to which high levels of depressive symptoms reflect a diagnosable mental health condition.

In spite of these limitations, this study provides preliminary evidence that the level of chronicity of a musculoskeletal condition influences response to treatment for individuals who present with high levels of depressive symptoms. The findings further suggest that changes in pain symptoms might play an important role in determining the magnitude of treatment-related reductions in depressive symptoms. The findings highlight the importance of early screening and intervention for depressive symptoms (Rush et al., 2000). Challenges for future research include more systematic efforts to investigate the efficacy of different treatment approaches to the management of depressive conditions associated with pain, particularly for more chronic cases where options for effective pain reduction might be more limited.

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