



Mediators, moderators, and predictors of therapeutic change in cognitive–behavioral therapy for chronic pain

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Abstract

Although cognitive–behavioral therapies (CBT) have been demonstrated to be effective for a variety of chronic pain problems, patients vary in their response and little is known about patient characteristics that predict or moderate treatment effects. Furthermore, although cognitive–behavioral theory posits that changes in patient beliefs and coping mediate the effects of CBT on patient outcomes, little research has systematically tested this. Therefore, we examined mediators, moderators, and predictors of treatment effects in a randomized controlled trial of CBT for chronic temporomandibular disorder (TMD) pain. Pre- to post-treatment changes in pain beliefs (control over pain, disability, and pain signals harm), catastrophizing, and self-efficacy for managing pain mediated the effects of CBT on pain, activity interference, and jaw use limitations at one year. In individual mediator analyses, change in perceived pain control was the mediator that explained the greatest proportion of the total treatment effect on each outcome. Analyzing the mediators as a group, self-efficacy had unique mediating effects beyond those of control and the other mediators. Patients who reported more pain sites, depressive symptoms, non-specific physical problems, rumination, catastrophizing, and stress before treatment had higher activity interference at one year. The effects of CBT generally did not vary according to patient baseline characteristics, suggesting that all patients potentially may be helped by this therapy. The results provide further support for cognitive–behavioral models of chronic pain and point to the potential benefits of interventions to modify specific pain-related beliefs in CBT and in other health care encounters.

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1. Introduction

Cognitive–behavioral therapies (CBT) have been demonstrated to be effective for a variety of chronic pain problems (Keefe and Caldwell, 1997; Morley et al., 1999; Astin et al., 2002; Eccleston et al., 2002; Weydert et al., 2003; Chen et al., 2004). Therapeutic mechanisms underlying the process of patient improvement (“process variables”) in CBT are posited by cognitive–

behavioral theory to be changes in patient cognitions and behaviors. Identification of specific process variables that mediate the effects of CBT on patient outcomes could facilitate refinement of theoretical models and the development of more effective and efficient therapies. Furthermore, knowledge concerning patient characteristics that predict or moderate improvement with CBT could help direct limited resources to those most likely to benefit, match patients with the most appropriate treatments, and tailor interventions to patient characteristics. Little research has been conducted in these areas, which have been highlighted as two of the most important directions for future research related to

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behavioral interventions for chronic pain (Keefe et al., 2002; Nicassio et al., 2004; Vlaeyen and Morley, 2005).

We recently reported the effectiveness of CBT, as compared with an education/attention control condition in a randomized controlled trial (RCT), in improving chronic temporomandibular disorder (TMD) pain and disability (Turner et al., 2006). The CBT intervention was designed to decrease patients' catastrophizing, beliefs that they were disabled by pain and that pain signaled harm, and maladaptive pain coping, and to increase patients' adaptive pain coping and beliefs in their ability to control and self-manage pain. These cognitions and behaviors were selected based on cognitive-behavioral theory, previous research indicating their importance in chronic pain problems and treatment-related improvement, and their being targeted for change in standard cognitive-behavioral pain treatments (Jensen et al., 1994a; Keefe et al., 1999; Turner et al., 2000, 2001; Jensen et al., 2001; Turner and Romano, 2001).

The aim of the current study was to identify mediators, moderators, and predictors of patient improvement with CBT in the RCT. The primary objective was to test the hypothesis that pre- to post-treatment changes in the targeted cognitions and behaviors mediated the effects of CBT on subsequent patient pain and disability. Random assignment of patients to credible treatments with and without the active ingredients of CBT, examination of whether pre- to post-treatment process variable changes mediated CBT effects on subsequent outcomes, and formal statistical tests of mediation offer methodological advantages over previous research. A second objective was to determine whether patient baseline characteristics moderated or predicted treatment effects. These latter analyses were considered exploratory given the paucity of research in this area. For example, little is known concerning whether personality traits affect response to CBT for pain. However, there is some suggestion that patients with shorter symptom duration, less tendency to somatize, less emotional distress, less catastrophizing, and greater perceived ability to control pain are more likely to benefit from cognitive and behavioral treatments (Gale and Funch, 1984; McCreary et al., 1992; Tota-Faucette et al., 1993; Sinclair and Wallston, 2001; McCracken and Turk, 2002; Blanchard et al., 2006).

2. Methods

2.1. Setting, participants, and procedures

The sample for the current study is a subset of the sample in the RCT, which was previously described in detail (Turner et al., 2006). In brief, participants were patients seeking care at a university dental school Orofacial Pain Clinic. Study inclusion criteria were age 18 years or older; a Research Diagnostic Criteria/Temporomandibular Disorders (RDC/TMD) Axis I TMD diagnosis (Dworkin and LeResche, 1992) made by an

oral medicine specialist based on a structured RDC/TMD clinical examination; facial pain for at least three months; facial pain-related disability, as defined by a chronic pain grade of II (high pain intensity and low pain-related disability), III (moderate pain-related disability), or IV (severe pain-related disability) (Von Korff et al., 1992); and the ability to communicate in English. Exclusion criteria were needed for further diagnostic evaluation, pending litigation or disability compensation for pain, current or previous CBT for pain, and major medical or psychiatric conditions that would interfere with ability to participate. Study participants ($N = 156$) were assigned randomly to four individual biweekly sessions over eight weeks of either CBT or an education/attention control condition. The CBT and control conditions also included brief telephone calls to patients in the weeks between the in-person sessions and at 2, 4, 8, 12, 16, 20, and 24 weeks after the fourth in-person session. All participants received treatment as usual at the Orofacial Pain Clinic.

The CBT and control groups did not differ significantly in sociodemographic characteristics, baseline chronic pain grade, or pain duration (Turner et al., 2006). The groups also did not differ significantly in the proportion of participants who completed at least three of the four in-person sessions (CBT: 78%, control: 85%; $P = 0.35$) or in the proportion of participants who completed all follow-up assessments (84% in each group). There were no meaningful differences in RCT results (superiority of CBT on each study outcome measure at one year, using an intent-to-treat analytic approach) when the analyses were repeated only for study participants who completed three or more of the four in-person sessions. Furthermore, the findings did not change when the analyses were repeated using multiple imputation to estimate missing values due to assessment non-response (Turner et al., 2006).

Because the focus of the current study was to assess mediators of CBT effects, we limited the analyses to the 115 study participants (60 education/attention control, 55 CBT) who completed at least three in-person sessions and all assessments. The 115 participants in this analysis sample were compared with study participants not in this sample ($n = 41$) on the demographic variables and the pretreatment scores on the outcome measures. There were no significant differences in age, gender, race, education, or baseline pain intensity or Mandibular Function Impairment (MFIQ) (Stegenga et al., 1993b) masticatory jaw use limitations scores. However, those in the current analysis sample had lower baseline pain-related activity interference scores [mean (SD) = 4.5 (2.3) versus 5.5 (2.6) on 0–10 scale, $P = 0.03$] and MFIQ non-masticatory jaw use limitations scores [mean (SD) = 0.35 (0.17) versus 0.43 (0.18) on 0–1 scale, $P = 0.01$]. Similar to the full RCT sample, 87% of the 115 patients in the current study were females and 85% were Caucasian.

2.2. Measures

2.2.1. Overview

In understanding the process of therapeutic change, it is useful to distinguish between mediators, non-specific predictors, and moderators (Kraemer et al., 2002). A mediator is a variable that is responsible for all or part of the effects of a treatment on an outcome. To be a mediator, a variable must change during treatment, be associated with treatment, and

have an effect on outcome. In the current study, statistical tests of mediation (described in Section 2.3.1) were conducted for putative mediators specified during the RCT design phase based on cognitive-behavioral theory and previous research. Moderators of treatment outcomes are baseline characteristics (that may or may not be theoretically identified) that interact with treatment to affect outcomes (i.e., the effect of treatment on individuals depends on their value of the moderator, which precedes treatment and is not associated with treatment). Non-specific predictors are patient baseline characteristics that predict response in both treatment and control groups. Thus, if there is a significant treatment by baseline characteristic interaction effect, the baseline characteristic is a moderator; if the interaction term is not statistically significant but the baseline characteristic predicts the outcome, the baseline characteristic is a non-specific predictor. The non-specific predictor and moderator variables were assessed prior to patient randomization (baseline/pre-treatment), the mediator variables were assessed at baseline and six months after the last in-person treatment session (the time of the last intervention telephone session), and the outcome measures were obtained at baseline and one year after the last in-person session.

2.2.2. Mediators of outcome

Following the recommendation of Kazdin and Nock (Kazdin and Nock, 2003), we tested multiple variables as possible mediators. According to cognitive-behavioral models of chronic pain, decreasing maladaptive and increasing adaptive patient cognitive and behavioral responses to pain will result in improvement in pain and related problems (Turner and Romano, 2001). Based on prior research establishing their associations with chronic pain problems, we selected the following measures of cognitions and coping responses to test as mediators:

2.2.2.1. Self-efficacy. Self-efficacy for managing TMD was assessed by the 8-item TMD Self-Efficacy Scale (SES), which is a modification (by replacing the word ‘arthritis’ with ‘facial pain’) of the Arthritis Self-Efficacy Scale (Gonzalez et al., 1995; Lorig et al., 1996). Patients rate on a scale from 0 = ‘very uncertain’ to 10 = ‘very certain’, their certainty that they can decrease their pain quite a bit, keep facial pain from interfering with their sleep, keep their pain from interfering with the things they want to do, regulate their activity so as to be active without aggravating their pain, keep the fatigue caused by pain from interfering with the things they want to do, do something to feel better if they are feeling blue, manage facial pain during their daily activities, and deal with the frustration of facial pain. Scale scores are calculated as the mean of the eight ratings, with higher scores indicating greater self-efficacy. We previously reported that this scale had excellent internal consistency (Cronbach’s $\alpha = 0.91$) and validity in the sample of TMD patients enrolled in the RCT (Brister et al., 2006).

2.2.2.2. Pain beliefs. We administered three scales from the Survey of Pain Attitudes (SOPA) (Jensen et al., 1994b): *Disability* (10 items assessing the belief that one’s pain is disabling; e.g., ‘I consider myself to be disabled,’ ‘my pain would stop anyone from leading an active life’), *Harm* (eight items assessing the belief that pain signifies damage and that activity

should be avoided; e.g., ‘the pain that I usually experience is a signal that damage is being done,’ ‘if I exercise, I could make my pain problem much worse’), and *Control* (10 items assessing the belief in one’s personal control over pain; e.g., ‘there are many times when I can influence the amount of pain I feel,’ ‘I have learned to control my pain’). These scales have good test-retest stability, validity, and internal consistency (Jensen and Karoly, 1992; Strong et al., 1992; Jensen et al., 1994b). Study participants were asked to indicate how much they agreed with each item, using a scale of 0 = ‘this is very untrue for me’ to 4 = ‘this is very true for me’. Scores on each scale are calculated as the mean of the summed responses and thus can range from 0 to 4, with higher scores indicating greater agreement with the belief. Although the SOPA Control Scale and the TMD SES are moderately correlated ($r = 0.54$ in our sample), the former scale assesses solely the belief in the ability to control one’s pain, whereas the SES assesses confidence in ability not only to decrease pain but also to manage specific pain-related problems.

2.2.2.3. Pain catastrophizing. Pain catastrophizing was assessed by two scales. The Coping Strategies Questionnaire (CSQ) Catastrophizing Scale has excellent internal consistency (Rosenstiel and Keefe, 1983; Keefe et al., 1989) and has been shown to be associated with various measures of functioning in samples of patients with different pain conditions (Keefe et al., 1987; Keefe et al., 1989; Jensen and Karoly, 1991; Dozois et al., 1996; Martin et al., 1996), including TMD (Turner et al., 2001). Scores can range from 0 to 6, with higher scores indicating greater catastrophizing. The four-item Rumination subscale of the Pain Catastrophizing Scale (PCS) captures aspects of catastrophizing not assessed by the CSQ: ruminative thoughts, worry, and an inability to inhibit pain-related thoughts (Sullivan et al., 1995). The Rumination Scale has been found to be associated with measures of pain and disability (Sullivan et al., 1998; Osman et al., 2000), and has good internal consistency and discriminant validity (Osman et al., 2000). Scores on the scale can range from 0 to 16, with higher scores indicating greater tendency to ruminate about pain.

2.2.2.4. Coping. In the RCT (Turner et al., 2006), there was a significant CBT effect on only one pain coping measure: the Relaxation Scale from the Chronic Pain Coping Inventory (CPCI). Therefore, this was the only coping measure examined in the current study. The CPCI scales have demonstrated internal consistency, test-retest reliability, and validity. Scores on the Relaxation Scale can range from 0 to 7, with higher scores indicating greater use of relaxation strategies to cope with pain.

2.2.3. Non-specific predictors and moderators

2.2.3.1. Demographic and baseline outcome measures. Although most studies have found no relationships between response to CBT and age, gender, race, education, and pain duration (McCracken and Turk, 2002), we explored whether these variables, as well as the baseline values of the mediator and outcome measures, predicted or moderated treatment effects. Based on previous research suggesting their potential importance in chronic pain problems, we also tested predictor/moderator effects for the following baseline measures:

2.2.3.2. Number of pain sites. Study participants were asked whether they had persistent, bothersome pain in the past six months in their head, neck, shoulders, back, arms or hands, buttocks or hips, abdomen/pelvic area, thighs, legs or feet, and whole (or most of the) body. The number of ‘yes’ responses was summed to create the total number of pain sites (range, 0–10).

2.2.3.3. Depression. The 21-item Beck Depression Inventory (BDI) (Beck and Beamesderfer, 1974; Beck et al., 1979) was used to assess depressive symptom severity. The BDI has high internal consistency, adequate test–retest reliability, and validity (Beck et al., 1988), and is a valid screening instrument for depression among patients with chronic pain (Turner and Romano, 1984; Love, 1987; Geisser et al., 1997).

2.2.3.4. NEO Neuroticism and Openness. The NEO Five-Factor Inventory™ (Costa and McCrae, 1992) Neuroticism and Openness scales were administered. Both scales have been demonstrated to have convergent and discriminant validity (Costa and McCrae, 1992). The Neuroticism Scale assesses tendency to experience negative affect (e.g., fear, sadness, anger, embarrassment, and guilt). Individuals high on the Openness Scale tend to be imaginative, curious, and willing to entertain novel ideas (Costa and McCrae, 1992).

2.2.3.5. Somatization. Participants completed the Somatization Scale of the Symptom Checklist-90 (SCL-90) (Derogatis et al., 1976; Derogatis and Cleary, 1977). This measure assesses distress arising from perceptions of bodily dysfunction. Symptoms assessed include cardiovascular, gastrointestinal, and respiratory. We scored the scale after excluding items that assess pain, so that patients’ pain problems would not inflate Somatization scores. Scores can range from 0 to 4, with higher scores indicating greater tendency to report distressing non-specific physical symptoms.

2.2.3.6. Perceived Stress Scale. Study participants completed the 14-item Perceived Stress Scale (PSS) (Cohen et al., 1983), which was designed to measure the extent to which situations in one’s life are perceived as stressful. The PSS has been shown to be valid and reliable (Cohen et al., 1983). Scores can range from 0 to 56, with higher scores indicating greater perceived stress.

2.2.4. Outcomes

2.2.4.1. Pain intensity and activity interference. The Graded Chronic Pain Scale (GCPS) (Von Korff et al., 1992; Von Korff, 2001) was used to assess pain intensity and interference with usual daily activities. The primary outcome measure in the RCT, *activity interference* (Von Korff, 2001), was calculated by averaging 0–10 ratings of pain interference with daily activities, work/housework activities, and recreational/social activities in the past month. *Characteristic pain intensity* was calculated by averaging 0–10 ratings of current pain and average and worst pain in the past month (Dworkin et al., 1990; Von Korff et al., 1992; Von Korff, 2001). The characteristic pain intensity and activity interference scores have good internal consistency, test–retest reliability, and validity (Underwood et al., 1999; Von Korff, 2001).

2.2.4.2. Jaw use limitations. Jaw use limitations were assessed by the MFIQ (Stegenga et al., 1993b). The MFIQ is a 17-item measure with two subscales (masticatory and non-masticatory jaw disability) demonstrated to be sensitive to change with treatment for TMD (Stegenga et al., 1993a). Scores on each subscale have a possible range of 0–1, with higher scores indicating greater limitations.

2.3. Statistical analyses

2.3.1. Mediation

We applied the widely used approach of Baron and Kenny (Baron and Kenny, 1986) to test the hypothesis that changes in multiple specific process variables over the course of treatment (baseline to six months) would mediate the effects of CBT on pain and disability at one year. First, to demonstrate the association between CBT and the outcome variable (the “total effect” of CBT on the outcome), we used regression analysis to examine whether there was an effect of CBT (relative to the control condition) on each outcome measure at one year, controlling for the baseline value of the outcome measure. Second, to demonstrate the association between CBT and the putative mediator, we constructed regression models with the six-month score on each mediator as the dependent variable, and treatment and the baseline score on the mediator as independent variables. Third, to demonstrate the association between the mediator and the outcome after adjusting for treatment and to demonstrate the reduction of the treatment effect on the outcome after adjusting for the mediator, we constructed regression models with both treatment and the mediator (baseline and six-month values) as independent variables and the outcome measure as the dependent variable. We did this separately for each mediator and also for the mediators as a group (i.e., entered baseline and six-month values of each mediator as independent variables).

The mediation effect is referred to as the indirect effect of treatment because it reflects the treatment effect on the outcome through the mediating variable (MacKinnon, 2000). To formally test the mediation effect in the third regression model, we used a version of the Sobel test (Sobel, 1982), which tests whether the indirect effect of treatment on the outcome through the mediator (defined as the product of the treatment to mediator path and the mediator to outcome path) is significantly different from zero. This was done for each mediator individually and for all mediators as a group. The total indirect effect of treatment on the outcome through the mediators as a group was estimated by the sum of the coefficient products for each mediator of the treatment to mediator path and the mediator to outcome path, where the latter coefficient estimate is adjusted for treatment and all other mediators. All coefficients were estimated simultaneously using structural equation modeling (MacKinnon, 2000). We used the bootstrap method of Preacher and Hayes (Preacher and Hayes, 2004) to estimate the indirect effect and bias-corrected 95% confidence interval (CI) for each individual mediator and for all the mediators as a group, based on 1000 bootstrap samples using a Statistical Package for the Social Sciences (SPSS®; SPSS Inc., Chicago, Illinois) macro (<http://www.comm.ohio-state.edu/ahayes/SPSS%20programs/indirect.htm>). This methodology has been recommended as superior to a normal theory approach because it does not require that the sampling distribution of

the indirect effect be normal (Shrout and Bolger, 2002; Preacher and Hayes, 2004). All analyses were conducted using SPSS® version 14.0 for Windows.

2.3.2. Moderators and non-specific predictors

To test whether baseline variables were non-specific predictors or moderators, we constructed linear regression models for each outcome measure. The dependent variable was the one-year score on the outcome measure and the independent variables were the baseline value of the outcome measure, the potential baseline predictor/moderator, treatment group (CBT, education/attention control), and the predictor/moderator X treatment interaction term. Because of the number of statistical tests, we used a *P*-value of 0.01 to define statistical significance. An argument could be made for a more conservative criterion, but given the exploratory, hypothesis-generating nature of these analyses, we did not want to increase the risk of missing true effects.

3. Results

3.1. Mediation analyses

The CBT group, as compared with the education/attention control group, showed significantly greater improvement on each outcome measure at one year (see first row of Tables 1–4). The CBT group also showed significantly greater improvement from baseline to six months on each process variable hypothesized to mediate the effects of CBT. Compared with the control group, CBT participants showed increased perceived control over pain [unstandardized regression coefficient (*B*) = 0.9; 95% CI = 0.7, 1.2], use of relaxation to cope with pain (*B* = 1.1; 95% CI = 0.6, 1.6), and self-efficacy for managing TMD and related symptoms (*B* = 1.7; 95% CI = 1.0, 2.4), as well as decreased belief that they were disabled by pain (*B* = -0.7; 95% CI = -0.9, -0.5), belief that pain indicated bodily harm (*B* = -0.5; 95% CI = -0.7, -0.3), rumination about pain (*B* = -1.9;

95% CI = -3.1, -0.7), and pain-related catastrophizing (*B* = -0.8; 95% CI = -1.2, -0.5).

Tables 1–4 also show the results of the test of the indirect effect of CBT on each outcome measure through each mediator individually and through all mediators as a group. For example, Table 1 shows the results for activity interference; it can be seen that there was a statistically significant indirect effect of CBT on this outcome measure through each process variable except relaxation. Thus, each process measure except relaxation was demonstrated to mediate the effects of CBT on activity interference. The last column of Tables 1–4 shows the percent of the total treatment effect on the outcome explained by the mediators, individually and together. For example, baseline to six-month changes in SOPA Control and Disability scale scores each explained 92% of the total effect of treatment on activity interference at one year (Table 1). The mediators as a group explained 97% of the total treatment effect on activity interference. In the model for activity interference that included all mediators, only the SOPA Disability Scale [estimate (95% CI) = -0.63 (-1.23, -0.23)] and the Self-Efficacy Scale [estimate (95% CI) = -0.46 (-1.05, -0.10)] retained statistically significant mediating effects (results not shown in the table).

Changes in control, disability, and harm beliefs; catastrophizing; and TMD self-efficacy were each demonstrated to mediate the effects of CBT on one-year pain intensity (Table 2). However, there was not a significant indirect effect of treatment on pain through change in rumination or use of relaxation. Changes in beliefs regarding the ability to control one's pain accounted for the largest proportion of the total treatment effect on pain (81%). As a group, the mediators explained 93% of the total effect of CBT on pain intensity. In the model predicting pain intensity from all mediators, no individual process variable retained a significant mediating effect.

Table 1

Mediators of CBT effects on one-year activity interference: total treatment effect and indirect effects of treatment through process variables

Treatment effect	Estimate (95% CI)	% Total effect explained by mediator(s)
Total treatment effect ^a	-1.28 (-2.06, -0.49)	
Indirect effect of treatment through ^b		
Control	-1.18 (-1.87, -0.56)	92
Disability	-1.18 (-1.80, -0.78)	92
Harm	-0.70 (-1.22, -0.34)	55
Rumination	-0.23 (-0.61, -0.01)	18
Catastrophizing	-0.59 (-1.11, -0.31)	46
Relaxation	-0.02 (-0.37, 0.34)	2
Self-efficacy	-0.87 (-1.55, -0.49)	68
All mediators	-1.24 (-2.19, -0.56)	97

^a Unstandardized regression coefficient (95% CI) for treatment (CBT versus control) effects on 1-year activity interference, unadjusted for mediators but adjusted for baseline activity interference.

^b Test of the statistical significance of the indirect effect of treatment (CBT versus control) on activity interference through the mediator(s). The indirect effect is the difference between the total effect of treatment on the outcome (shown in the top row) and the treatment to outcome path coefficient after controlling for the mediator. Estimation of the indirect effect (95% CI) was obtained using the bootstrap method of Preacher and Hayes (2004).

Table 2
Mediators of CBT effects on one-year pain intensity: total treatment effect and indirect effects of treatment through process variables

Treatment effect	Estimate (95% CI)	% Total effect explained by mediator(s)
Total treatment effect ^a	-1.49 (-2.32, -0.67)	
Indirect effect of treatment through ^b		
Control	-1.20 (-1.95, -0.59)	81
Disability	-0.81 (-1.43, -0.42)	54
Harm	-0.36 (-0.91, -0.02)	24
Rumination	-0.22 (-0.62, 0.05)	15
Catastrophizing	-0.44 (-0.94, -0.12)	30
Relaxation	-0.16 (-0.62, 0.12)	11
Self-efficacy	-0.67 (-1.31, -0.25)	45
All mediators	-1.38 (-2.28, -0.54)	93

^a Unstandardized regression coefficient (95% CI) for treatment (CBT versus control) effects on 1-year pain intensity, unadjusted for mediators but adjusted for baseline pain intensity.

^b Test of the statistical significance of the indirect effect of treatment (CBT versus control) on pain intensity through the mediator(s). The indirect effect is the difference between the total effect of treatment on the outcome (shown in the top row) and the treatment to outcome path coefficient after controlling for the mediator. Estimation of the indirect effect (95% CI) was obtained using the bootstrap method of Preacher and Hayes (2004).

With the exception of relaxation coping, each process variable was demonstrated to mediate the effects of CBT on masticatory (Table 3) and non-masticatory (Table 4) jaw use limitations. Belief in one's ability to control pain was the mediator that explained the largest amount of the total treatment effect on both types of jaw use limitations (65% of the CBT effect on masticatory disability and 100% of the effect on non-masticatory disability). In the models predicting masticatory and non-masticatory limitations from all process variables, only self-efficacy retained a statistically significant mediating effect [estimate (95% CI) = -0.05 (-0.10, -0.01) for masticatory and -0.03 (-0.08, -0.01) for non-masticatory].

3.2. Non-specific predictor and moderator analyses

In the tests for moderation effects, only one significant ($P < 0.01$) finding emerged. Baseline MFIQ Masticatory Scale scores interacted with treatment in predicting one-

year MFIQ Masticatory scores ($P = 0.001$). To better understand this effect, we used a median split to divide the patients into two groups: low and high baseline MFIQ Masticatory scores. We then compared CBT versus control condition patients within each of those two groups in terms of their one-year Masticatory scores. Among patients with low baseline masticatory disability, those in the CBT group did not differ significantly from those in the control group on one-year Masticatory scores. However, among patients with high baseline masticatory disability, those in the CBT group had significantly lower masticatory disability at one year as compared with those in the control group [M (SD) = 0.41 (0.25) versus 0.65 (0.18), $P < 0.001$].

Table 5 shows the variables that were statistically significant non-specific predictors of one-year activity interference and non-masticatory jaw use limitations. Patients who at baseline reported more pain sites, depressive symptoms, non-specific physical problems

Table 3
Mediators of CBT effects on one-year MFIQ Masticatory scores: total treatment effect and indirect effects of treatment through process variables

Treatment effect	Estimate (95% CI)	% Total effect explained by mediator
Total treatment effect ^a	-0.17 (-0.24, -0.10)	
Indirect effect of treatment through ^b		
Control	-0.11 (-0.17, -0.07)	65
Disability	-0.07 (-0.12, -0.03)	41
Harm	-0.06 (-0.11, -0.03)	35
Rumination	-0.02 (-0.06, -0.001)	12
Catastrophizing	-0.05 (-0.10, -0.02)	29
Relaxation	-0.03 (-0.07, 0.001)	18
Self-efficacy	-0.08 (-0.13, -0.03)	47
All mediators	-0.14 (-0.22, -0.08)	82

^a Unstandardized regression coefficient (95% CI) for treatment (CBT versus control) effects on 1-year MFIQ Masticatory scores, unadjusted for mediators but adjusted for baseline MFIQ Masticatory scores.

^b Test of the statistical significance of the indirect effect of treatment (CBT versus control) on MFIQ Masticatory scores through the mediator(s). The indirect effect is the difference between the total effect of treatment on the outcome (shown in the top row) and the treatment to outcome path coefficient after controlling for the mediator. Estimation of the indirect effect (95% CI) was obtained using the bootstrap method of Preacher and Hayes (2004).

Table 4

Mediators of CBT effects on one-year MFIQ Non-Masticatory scores: total treatment effect and indirect effects of treatment through process variables

Treatment effect	Estimate (95% CI)	% Total effect explained by mediator(s)
Total treatment effect ^a	-0.10 (-0.16, -0.05)	
Indirect effect of treatment through ^b		
Control	-0.10 (-0.14, -0.06)	100
Disability	-0.07 (-0.11, -0.04)	70
Harm	-0.05 (-0.09, -0.03)	50
Rumination	-0.03 (-0.06, -0.01)	30
Catastrophizing	-0.04 (-0.08, -0.02)	40
Relaxation	-0.02 (-0.05, 0.01)	20
Self-efficacy	-0.05 (-0.10, -0.03)	50
All mediators	-0.10 (-0.17, -0.06)	100

^a Unstandardized regression coefficient (95% CI) for treatment (CBT versus control) effects on 1-year MFIQ Non-Masticatory scores, unadjusted for mediators but adjusted for baseline MFIQ non-masticatory scores.

^b Test of the statistical significance of the indirect effect of treatment (CBT versus control) on MFIQ Non-Masticatory scores through the mediator(s). The indirect effect is the difference between the total effect of treatment on the outcome (shown in the top row) and the treatment to outcome path coefficient after controlling for the mediator. Estimation of the indirect effect (95% CI) was obtained using the bootstrap method of Preacher and Hayes (2004).

(somatization), pain-related rumination, pain-related catastrophizing, and perceived stress had greater activity interference at one year, adjusting for baseline interference. Patients with greater baseline depressive symptom severity had greater non-masticatory disability at one year, adjusting for baseline masticatory disability. No non-specific predictor effects were found for one-year characteristic pain intensity or masticatory disability.

4. Discussion

Pre- to post-treatment changes in patient pain-related beliefs were shown in this study to mediate the effects of CBT on TMD pain and disability at one year. Although concurrent associations between pre- to post-treatment changes in process and outcome measures have been reported previously (Jensen et al., 1994a; Turner et al., 1995; Burns et al., 1998; Nielson and Jensen, 2004), this is the first study to apply, within the context of an RCT, a formal statistical test of mediation using outcomes measured months after the assessment of the process

variables. Two prior studies examined mediation effects in an RCT of CBT for pain (Spinhoven et al., 2004; Smeets et al., 2006); however, both analyzed outcome and process variables assessed simultaneously (pretreatment and post-treatment). Two other studies (Burns et al., 2003a,b) used a cross-lagged panel design to help rule out reverse causation between process and outcome variables, but they did not formally test mediation or randomize patients to CBT versus a control condition.

The study results point to specific process variables that may play the most important roles in patient improvement with CBT. When mediators were examined individually, increased perceived ability to control pain (assessed by the SOPA Control Scale) explained the greatest proportion of the total effect of CBT across outcomes. Increased self-efficacy and decreased belief that one is disabled by pain, belief that pain signals harm, and catastrophizing also played substantial mediational roles. However, when the mediators were analyzed as a group, self-efficacy tended to be the only process variable that had a unique mediating effect. Thus, the mediating effect of perceived control may reflect its associations with the other process variables (changes in the other process variables may all be associated with increased perceived control and with improvement in the outcomes), whereas self-efficacy appears to have an effect independent of the other process variables. In sum, these findings indicate that catastrophizing and control, disability, and harm beliefs may all be important to target in CBT, but that they are interrelated. Efforts to increase patients' self-efficacy for managing pain and related problems may have unique additional benefits.

The findings that increased control beliefs and decreased disability and harm beliefs mediate CBT effects on chronic pain patient outcomes are consistent with

Table 5

Non-specific predictors of outcome at one year^a

Predictor	Slope	99% CI
<i>Outcome = activity interference</i>		
Number of pain sites	0.21	0.02, 0.40
BDI	0.10	0.05, 0.16
Somatization	1.15	0.33, 1.96
Rumination	0.15	0.009, 0.29
Catastrophizing	0.47	0.05, 0.89
Perceived stress	0.93	0.10, 1.75
<i>Outcome = MFIQ Non-Masticatory</i>		
BDI	0.005	0.001, 0.009

BDI, Beck Depression Inventory; MFIQ, Mandibular Function Impairment Questionnaire.

^a Outcome adjusted for baseline value; $P < 0.01$.

results from previous correlational studies. For example, improvement in multidisciplinary pain programs has been shown to be associated with concurrent increases in perceived control over pain and decreases in disability and harm beliefs (Jensen et al., 1994a; Jensen et al., 2001; Nielson and Jensen, 2004), as well as with decreases in pain helplessness (i.e., low control) (Burns et al., 1998). Furthermore, decreases in pain helplessness early in multidisciplinary pain treatment predicted late-treatment decreases in pain (Burns et al., 2003a) and interference (Burns et al., 2003b), but not vice versa. Changes in perceived control over pain and beliefs that pain is a signal of harm/disease also were associated with improvement in TMD pain and jaw functioning after another brief CBT intervention (Turner et al., 1995).

The present findings of a mediation effect for self-efficacy also corroborate previous correlational results. For example, in studies of spouse-assisted coping skills training (SACST) for osteoarthritic knee pain, increases in self-efficacy were associated with improved pain and psychological functioning (Keefe et al., 1999). Increases in self-efficacy were later also found to be associated with increased physical fitness during exercise training and decreased psychological disability after SACST plus exercise training (Keefe et al., 2004). Other studies have also observed associations between increased self-efficacy and improved functioning after pain treatment (Arnstein et al., 2001).

Consistent with past research (Burns et al., 2003a; Spinhoven et al., 2004), changes in catastrophizing as measured by the CSQ were found to mediate the effects of CBT. The other catastrophizing measure, rumination about pain, was not a significant mediator of the CBT effect on pain intensity and was a relatively weak mediator of effects on the other outcomes. In general, changes in CSQ catastrophizing explained less of the effect of CBT on the outcomes than did changes in control, disability, harm, and self-efficacy beliefs. Changes in control, disability, and harm beliefs were also more important than changes in catastrophizing in explaining improvement in a study of multidisciplinary pain treatment (Jensen et al., 1994a; Jensen et al., 2001). However, catastrophizing, but *not* a measure of internal control of pain, mediated the effects of CBT (and of active physical treatment with and without CBT) in a study of chronic low back pain patients (Smeets et al., 2006). This discrepancy could reflect the control measure used. In the Smeets study, the internal control measure had unknown responsiveness to clinical change and patients had fairly high control at baseline, leaving little room for improvement. These mixed results point to the need for further study of catastrophizing as a mediator of CBT effects. Its importance may depend on patient characteristics, treatment components, and measures used.

Only one pain coping measure (relaxation) showed a significant treatment effect and it did not mediate the

effects of CBT on any outcome. Previous studies have also suggested that changes in pain coping may not mediate pain treatment effects (Jensen et al., 1994a; Jensen et al., 2001; Spinhoven et al., 2004). However, failure to find mediation effects for specific pain coping measures does not necessarily indicate that coping is unimportant. Coping strategy effectiveness may vary across patients as well as within patients over time and in different situations. Skills taught in CBT for coping with daily stressors and other problems may be important but were not assessed in this study. Future research that examines within- and between-subject changes in various pain and stress coping responses may shed more light on the role of coping in mediating CBT effects on pain problems. Further study of the relative importance of changes in beliefs versus coping responses will be important for the refinement of theoretical models and therapies. Although changing patient beliefs may ultimately prove to be more important than changing specific pain coping strategies, working with patients to develop individualized pain and stress coping plans may result in the desired changes in the cognitive variables.

The effects of CBT on outcomes generally did not vary according to patient baseline characteristics. However, several non-specific predictors of outcomes were identified. Patients with greater baseline somatization, depressive symptoms, number of pain sites, rumination, catastrophizing, and perceived stress had greater activity interference at one year. Previous studies in different populations also found these factors to be associated with greater concurrent and future disability and with worse treatment outcomes (Gale and Funch, 1984; McCreary et al., 1992; Dworkin et al., 1994; Ciccone et al., 1996; Gureje et al., 2001; Leveille et al., 2001; Dworkin et al., 2002; McCracken and Turk, 2002; Rammelsberg et al., 2003). Patients with these characteristics may require more intensive CBT, and treatment of depression may be advisable before or in conjunction with CBT for pain. The two personality characteristics that were examined, neuroticism and openness, did not predict or moderate treatment effects. This suggests that neuroticism, generally believed to be associated with poor health outcomes (Goodwin et al., 2006), does not preclude benefits from CBT. These findings are also consistent with recent suggestions that examining individuals' cognitive styles may be more helpful than trait-based personality models in understanding response to CBT (Merrill and Strauman, 2004).

Analyses for this study were performed on the subset of RCT participants who completed at least three treatment sessions and all follow-ups in order to use information from all assessments to examine change related to treatment participation. However, the results may not generalize to less compliant patients. Furthermore, the sample consisted primarily of white women. Further research is needed to assess the generalizability of the

findings to individuals with other pain syndromes and sociodemographic characteristics, as well as to other forms of CBT.

Although cognitive-behavioral treatments share certain fundamental characteristics, they vary in goals and techniques (Turner and Romano, 2001). Thus, studies involving different forms of CBT may yield somewhat different results concerning specific mediators. However, the present results, in conjunction with similar findings from previous studies, provide strong evidence that changes in control, disability, and harm beliefs; catastrophizing; and self-efficacy for managing pain mediate the effects of cognitive and behavioral treatments for patients with different chronic pain syndromes. CBT and other treatments for patients with chronic pain may be strengthened by components designed to increase patients' confidence in their ability to control and self-manage their pain and related problems, and to decrease patients' beliefs that they are disabled by their pain and that pain signals harm. An important next direction for research will be the evaluation of CBT interventions refined to more powerfully impact these beliefs and to maintain or even increase these changes over time after the end of treatment.

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