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# Reduction of Pain Catastrophizing Mediates the Outcome of Both Physical and Cognitive-Behavioral Treatment in Chronic Low Back Pain

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**Abstract:** **The aim of this study was to examine whether treatments based on different theories change pain catastrophizing and internal control of pain, and whether changes in these factors mediate treatment outcome. Participants were 211 patients with nonspecific chronic low back pain (CLBP) participating in a randomized controlled trial, attending active physical treatment (APT, n = 52), cognitive-behavioral treatment (CBT, n = 55), treatment combining the APT and CBT (CT, n = 55), or waiting list (WL, n = 49). Pain catastrophizing decreased in all 3 active treatment groups and not in the WL. There was no difference in the change in internal control across all 4 groups. In all the active treatment groups, patients improved regarding perceived disability, main complaints, and current pain at post-treatment, and no changes were observed in the WL group. Depression only changed significantly in the APT group. Change in pain catastrophizing mediated the reduction of disability, main complaints, and pain intensity. In the APT condition, pain catastrophizing also mediated the reduction of depression. Not only cognitive-behavioral treatments but also a physical treatment produced changes in pain catastrophizing that seemed to mediate the outcome of the treatment significantly. The implications and limitations of these results are discussed.**

***Perspective:*** ***This article shows that treatment elements that do not deliberately target cognitive factors can reduce pain catastrophizing. Reduction in pain catastrophizing seemed to mediate the improvement of functioning in patients with chronic low back pain. The results might contribute to the development of more effective interventions.***



***Key words:*** *Chronic low back pain, catastrophizing, pain control, rehabilitation, mediation, outcome.*

In the treatment of chronic low back pain (CLBP), different treatments have been developed and studied. There is substantial evidence that exercise therapy, cognitive-behavioral therapy, and multidisciplinary treatment are more effective than doing nothing or a waiting list control treatment. But there is no strong evidence that one of the abovementioned active treatments is more effective than the other.[17,18,31,37] Furthermore, the treatment effects are not impressive because most effect sizes are not greater than 0.5, and a substantial proportion of patients do not seem to benefit at all. To improve the effectiveness/efficacy of treatment, we need insight into what mechanisms are responsible for the desired outcomes and what kind of treatment is the best for a particular patient.

Recently, Vlaeyen and Morley[57] proposed to approach these questions by using the moderator-mediator distinction as described by Baron and Kenny.[5] Moderators provide the answer to the question "in what circumstances does the treatment work?," whereas mediators concern the question "how does the treatment work?". Some examples of moderators are dose of treatment and

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client expectancy, whereas examples of mediators are changing beliefs and behavior contingencies.

Pain catastrophizing and internal control seem to be important factors in the development of chronic pain and disability[22,48,50] and also have been shown to mediate the outcome of multidisciplinary treatment.[10,11,23-25] Most studies consisted of uncontrolled designs, but Spinhoven et al[44] were the first to show that pain catastrophizing and internal control mediated the outcome of cognitive-behavioral treatment as compared with a waiting list control group. Unfortunately, the outcome measures were composite scores, and a common measure for disability, the main outcome of rehabilitation treatment, was not available. So far, no study has examined the mediating role of pain catastrophizing and internal control in a pure physical treatment for CLBP.

Studying the potential mediating factors of a physical, cognitive-behavioral, and multidisciplinary treatment might increase our knowledge regarding the working mechanisms of such treatments and further improve the development of more effective treatments.

Recently, we performed a randomized controlled trial (RCT) in which 3 active treatments based on 3 frequently used theories regarding the development and treatment of CLBP (the deconditioning theory, the cognitive-behavioral theory, and the biopsychosocial theory) were compared with a waiting list control treatment (WL). The treatments were an active physical therapy (APT) to improve aerobic fitness level and low back muscles strength/endurance, a cognitive-behavioral therapy with operant treatment principles and problem-solving techniques to improve coping with daily problems, stress, and pain (CBT), and multidisciplinary therapy combining the APT and CBT (CT).

The aim of the current analysis was to test whether pain catastrophizing and internal control mediated the outcome in terms of pain, depression, and functional disability in patients with CLBP who received APT, CBT, or CT.

## Materials and Methods

This study is part of a larger RCT (ISRCTN22714229) of which the methods are extensively described elsewhere.[42] The main aim of this RCT was to test the hypotheses that the 3 active treatments are more effective than the WL, and that the combination of both single treatment elements is more effective than the single treatments. A short description of participants, treatments, measures, and study design is presented with special emphasis on the putative mediating factors.

### *Participants*

Patients with CLBP were referred by general practitioners and medical specialists to 3 Dutch outpatient rehabilitation centers. The main inclusion criteria for participation in the RCT were disability caused by nonspecific LBP of more than 3 months, age between 18 and 65 years, and ability to walk at least 100 meters without interruption. Exclusion criteria were vertebral fracture, spinal inflammatory disease, spinal infections or malignancy, current nerve root pathology, spondylolysis or spondylolisthesis, lumbar spondylodesis, medical comorbidity making intensive exercising impossible (eg, cardiovascular or metabolic disease), ongoing diagnostic procedures or treatment for their CLBP at the time of referral, psychopathology that would hamper individual or group processes, not proficient in Dutch language, pregnancy, and substance abuse that could interfere with the rehabilitation treatment.

Patients were requested to stop other therapies for their back complaints, except pain medication.

Of the 309 patients who were referred to the study, 82 (26%) did not participate. The main reasons were not meeting the criteria (n = 31), clear preference for a particular treatment (n = 20), and logistic problems (n = 15). Another 4 patients were excluded immediately after randomization because they did not meet the criteria (other medical diagnosis). Of the 223 patients who started the treatment (APT, n = 53; CBT, n = 58; CT, n = 61; WL, n = 51), 11 patients did not complete any questionnaire immediately after the end of treatment (5%), and 1 patient did not complete the Pain Cognition List (PCL) questionnaire immediately after treatment. Complete results were available for 211 patients (APT, n = 52; CBT, n = 55; CT, n = 55; WL, n = 49). A summary of the baseline data of the total study population is presented in Table 1. Comparison of the baseline variables between those who did and those who did not complete questionnaires immediately after treatment did not show significant differences.

### *Study Design*

After referral by a consultant in rehabilitation medicine, the patient was invited for a meeting with the research assistant, during which further information about the trial was provided, and inclusion criteria were checked. If eligible for participation, the patient was asked to give his/her informed consent for participation. One to several weeks later the patient was invited to participate in the first assessment (pretreatment). After this assessment participants were allocated at random to 1 of the 3 possible interventions or the WL condition. Randomization took place per group of 4 patients at a time in each rehabilitation center. After 10 weeks of therapy the immediately post-treatment assessment took place.

The medical ethics committee of the Rehabilitation Foundation Limburg and Institute for Rehabilitation Research at Hoensbroek in The Netherlands approved the study protocol.

### *Interventions*

All interventions started with a group meeting of a maximum of 4 patients during which the rationale of the particular therapy was explained. Emphasis was put on the responsibility of the patient for making plans to keep on being active after the treatment. Each treatment lasted 10 weeks and started with the explanation of the rationale of that particular treatment. A written summary of the rationale was given to the patients.

**Table 1. Baseline Variables for All Patients in the 4 Therapy Groups (Total n = 211)**

| *Variables* | *APT (n = 52)* | *CBT (n = 55)* | *CT (n = 55)* | *WL (n = 49)* |
|---|---|---|---|---|
| Age (y) | 43.00 ± 8.84 | 42.02 ± 9.47 | 41.58 ± 10.07 | 40.63 ± 11.29 |
| Gender (% male) | 59.6 | 40.0 | 61.8 | 49.0 |
| Education (%) | | | | |
| Low | 67.3 | 60.0 | 60.0 | 63.3 |
| Middle-high | 32.7 | 40.0 | 40.0 | 36.7 |
| Work status (%) | | | | |
| Full-time | 32.7 | 32.7 | 25.5 | 24.5 |
| Partial sick leave/disability pension | 19.2 | 25.5 | 34.5 | 18.4 |
| Full sick leave/disability pension | 42.3 | 32.7 | 32.7 | 42.9 |
| No job/retired | 5.8 | 7.3 | 7.3 | 14.3 |
| Duration of LBP (mo) | 57.87 ± 76.28 | 69.87 ± 75.86 | 56.18 ± 70.62 | 44.65 ± 72.07 |
| Duration of disability (mo) | 29.27 ± 37.67* | 50.05 ± 62.94* | 35.65 ± 55.71* | 24.20 ± 33.26* |
| Radiation of pain (%) | | | | |
| No radiation | 9.6 | 16.4 | 14.5 | 16.3 |
| Above knee | 36.5 | 27.3 | 41.8 | 42.9 |
| Below knee | 53.8 | 56.4 | 43.6 | 40.8 |
| Previous back surgery (%) | 17.3 | 10.9 | 14.5 | 16.3 |
| Trauma preceding LBP (%) | 17.3 | 20.0 | 10.9 | 28.6 |
| PCL-catastrophizing | 40.44 ± 13.94 | 41.09 ± 11.92 | 38.82 ± 11.85 | 38.27 ± 11.92 |
| PCL-internal control | 16.77 ± 3.58 | 15.67 ± 4.09 | 16.53 ± 3.21 | 15.91 ± 4.02 |
| RDQ | 14.15 ± 3.74 | 13.87 ± 3.55 | 13.67 ± 3.66 | 13.82 ± 3.86 |
| PSC | 74.25 ± 14.60 | 74.84 ± 16.24 | 71.95 ± 17.14 | 77.18 ± 11.08 |
| Current pain | 51.06 ± 26.78 | 49.07 ± 23.92 | 45.62 ± 23.94 | 50.73 ± 25.86 |
| BDI | 10.52 ± 7.62 | 10.67 ± 7.11 | 9.35 ± 6.28 | 9.85 ± 7.81 |

Values presented as means and standard deviation or percentage.

Abbreviations: APT, active physical therapy; CBT, cognitive behavioral therapy; CT, combined therapy; WL, waiting list; LBP, low back pain; PCL, Pain Cognition List; RDQ, Roland Disability Questionnaire; PSC, patient-specific complaints; BDI, Beck Depression Inventory.

*Significant differences between therapy groups ($P < .05$).

All patients were asked to attend as much as possible the sessions offered during the treatment.

To assure sufficient contrast between the 3 different treatments and to avoid incorporating possible confounding elements, all therapists were instructed not to discuss general aspects concerning back pain origin, anatomy, and ergonomics. No other interventions than those that were chosen for the APT, CBT, or CT took place. In case of acute and severe psychosocial stress or pathology (severe depression, high risk for suicide, or personal problems the patient did not wish to discuss during the group treatment), a consultation of a clinical psychologist or social worker was possible. During this consultation the therapist tried to find out what the exact problem was and consecutively, when judged necessary, arranged for professional help outside the rehabilitation center.

All therapists received an extensive training before the start of the trial. They attended refresher courses; two 1-day courses during the first year and 1 each year during the next 2 years of the trial. For each intervention detailed treatment manuals were used by all therapists. The clinical psychologists and social workers had at least 5 years of experience in treating CLBP patients.

## APT

In a group of 4 maximum, patients were invited to perform 30 minutes of aerobic training on a bicycle (65% to 80% heart rate maximum) and 75 minutes of strength and endurance training of their lower back and upper leg muscles (3 series of 15 to 18 repetitions in a dynamic-static manner) 3 times a week during 10 weeks. The exercises consisted of leg extension while sitting on knees and hands, trunk lifting and lifting both legs while lying prone on a couch. During the exercises assistive weight by a pulley system or extra weight placed on the body of the patient was used, depending on the calculated load (70% of 1-Repetition Maximum, which allowed 15 to 18 repetitions until muscular fatigue occurred). No kind of cognitive-behavioral intervention was given, and patients did not receive homework assignments. The training was supervised by 2 physiotherapists.

## CBT

From a theoretical point of view, Aldrich et al[2] hypothesized that patients with chronic pain tend to persevere in their attempt to solve an unsolvable problem, namely pain, despite the experience of repeated failure. This perseverance might keep them hypothetically stuck in a vicious circle of chronic pain. To intervene in this process it might be more important to help patients to identify and cope with the consequences of pain in everyday life. Problem-solving training (PST) can help the patients to redefine their problem(s) and focus more on other individually relevant daily life goals that can be achieved by using graded activity (GA) techniques. Therefore, CBT consisted of operant behavioral GA training[14,41] and

PST.[32,33,52] The GA started with 3 group sessions followed by a maximum of 17 individual sessions of 30 minutes. During the GA, the therapist focused on a time-contingent gradual increase or pacing of 3 activities that were considered important and relevant for the patient's personal situation. After a baseline, activity tolerance level was calculated and final treatment goals were set, and the patient started performing the selected activities following quotas for each day, starting from 70% to 80% of the baseline with gradually increasing activity levels toward the final treatment goals. The patient was instructed only to perform the agreed amount of activity and not to perform less or more, even when he/she felt capable of doing so. The daily performance was graphically registered in a personal diary, and therapists were instructed to discuss these graphs regularly with the patient, while positively reinforcing any progress toward the preset goals. Contrary to GA therapy that has been studied in other trials and to create as much contrast as possible with the APT, no physical therapy elements (eg, muscle strengthening or aerobic exercises) were incorporated.

The PST started with 3 initial sessions in which the rationale of training and the skill of positive problem orientation were discussed. Sessions 4 to 10 focused on problem definition and formulation, generation of alternatives, decision making, implementation, and evaluation. Patients received a course book with additional information, a summary of each session, and homework assignments. The training of the skills and application were the main focus of the therapy, rather than one specific problem area. Patients were free to select their own personal problem areas. After each session, homework assignments were given to practice the skills in everyday life situations. A clinical psychologist or social worker, specifically trained to guide this intervention, provided 10 sessions of 1 ½ hours to a maximum of 4 patients at a time.

### CT

CT consisted of APT in combination with the PST, both offered in the same frequency and duration as described before. The patient was told that he/she first had to gain enough aerobic fitness and strength before increasing his/her activities. The GA was not started until the third week, and it started with the selection of the 3 patient-relevant activities. By the end of the fourth week the final goals and daily quota were set. In total, 19 sessions with a total duration of 11 hours were given.

### WL

The patients assigned to the WL were requested to wait 10 weeks, after which they were offered a regular individual rehabilitation treatment. During the waiting period, patients were not allowed to participate in diagnostic or therapeutic procedures because of their CLBP.

## *Assessment*

Assessments (questionnaires) were carried out before treatment and immediately after 10 weeks of active treatment or waiting by research assistants who were not aware of the patient's assigned treatment.

## *Outcome Measures*

### Disability

The Roland Disability Questionnaire (RDQ) was used. This questionnaire has been shown to be a valid and reliable instrument in the evaluation of CLBP treatment.[8,16,39,46,47] In this sample the internal consistency was sufficient (Cronbach alpha = .73).

### Patient-Specific Complaints

The patient-specific main complaints were identified by using the patient-specific approach method from Tugwell et al.[9,49] The patient has to indicate the 3 most limited functional activities and to rate the difficulty in performing these activities during the previous week on a 100-mm visual analog scale (VAS). This method appeared to be valid, reliable, and of sufficient responsiveness.[8,36]

### Pain

A 100-mm VAS with "no pain" on the left side and "unbearable pain" on the right side was used to measure the current pain intensity. Relevance, validity, and reliability have been sufficiently tested for patients with LBP.[13,38,45]

### Depression

The level of depression was measured by the Beck Depression Inventory (BDI),[6,60] a reliable, valid, and widely used questionnaire in CLBP research.[7] The internal consistency in this population was excellent (Cronbach alpha = .86).

## *Mediating Factors*

### Pain Catastrophizing and Internal Control of Pain

The subscales pain catastrophizing and internal control of pain of the Pain Cognition List (PCL) were used to measure pain catastrophizing and internal control of pain. The reliability and stability of this questionnaire have been proved to be sufficient in CLBP patients.[53,56] The validity of the subscales was supported by the meaningful pattern of correlations with other relevant constructs.[56] For example, the subscale of pain catastrophizing showed correlations of approximately .70 with the catastrophizing subscale of the Coping Strategies Questionnaire (CSQ).[40] Furthermore, pain catastrophizing correlated with negative emotions such as depression (.66 with BDI)[6] and fear (.51 with Tampa Scale for Kinesiophobia [TSK]).[55] The subscale internal control of pain correlated positively with other measures of internal locus of control measures, .37 with subscale perceived control of the CSQ and .32 with Multidimensional Health Locus of Control Scale (MHLC).[58]

The PCL is a 39-item self-report questionnaire with a 5-point Likert scale answering categories ranging from

"completely disagree" to "completely agree". The pain catastrophizing subscale consists of 16 items, and the total score ranges from 16 to 80. Some items are "My thoughts are always concentrating on the pain," "I feel like an unlucky person," "I think that fate has struck me." The higher the score, the more the person is catastrophizing. Internal consistency appeared to be excellent in our sample (Cronbach alpha = .89). The internal control of pain subscale consists of 5 items. Typical items are "I know a way to decrease my pain a little," "I think that I can influence my pain positively." Two questions need to be scored inversely (eg, "I think that I can't do anything against my pain"), and next a total score of these 5 items is calculated. The total score ranges from 5 to 25, with a higher score indicating increased control of pain. The internal consistency also appeared to be sufficient (Cronbach alpha = .68).

## Compliance and Treatment Integrity

To check whether patients were compliant with the allocated treatment and the therapists did follow the treatment manual instructions, each therapist kept precise records on the presence during treatment, the amount of exercise (duration, intensity of exercising by monitoring heart rate during cycling, and amount of repetitions and weight displaced during muscle training), and choice of activities and increase in time of these activities for the GA training. At the end of treatment each therapist made copies of the graphs in which the patient recorded the amount of activities he/she performed during the treatment and at home.

In accordance with other studies and guidelines, the presence at the treatment had to be at least 2/3 of the maximal amount of administered sessions to conclude that the treatment was of sufficient intensity.[1,52]

During the trial an independent researcher made several observations of all active treatments (on-site, audio and video recordings). The independent researcher analyzed whether the therapists did not engage in delivering treatments outside the manual, for example, decatastrophizing during the APT.

## Statistical Analysis

All statistical analyses were carried out according to the intention-to-treat principle. All patients, including withdrawals from treatment and patients with poor compliance, remained in the group to which they were randomized.

To check whether treatment differentially influenced pain catastrophizing and internal pain control, linear regression analyses were performed with the score of the putative mediating factor at the post-treatment assessment as the dependent variable. The variables age, gender, treatment center, the baseline score of the putative mediating factor, and the baseline value of the outcome of interest always stayed in the regression analysis as covariates. Variables for which, despite randomization, differences between treatment groups at baseline were found ($P < .10$) were added to the regression equation as a covariate.

To account for possible dependence of the outcomes within the groups of 4 patients who were randomized together, a random intercept term for these groups was included in all models by using multilevel analyses (SPSS mixed linear; SPSS Inc, Chicago, Ill).

Dummy variables for the 3 active treatments were made, and the coefficients for these dummies were the estimated treatment effects with respect to WL (reference group). Differences between CT and APT and CBT, respectively, were estimated similarly.

Effect sizes are estimated as Hedges's *g*.[21]

Mediation can be investigated by the 3-step method described by Baron and Kenny.[5] Mediation is suggested when the change in the putative mediating factors is significantly related to treatment as the independent variable, outcome is significantly related to treatment as the independent variable, and finally, the relationship between outcome and treatment decreases (or goes to zero) when the change in the mediating factor is entered into the equation.

Three successive multilevel regression analyses were conducted. The first analysis was performed to test whether pain catastrophizing and internal control of pain significantly changed while comparing the 3 active treatment groups with the WL (reference treatment). The treatment was the independent variable, and pain catastrophizing or internal control immediately post-treatment was the dependent variable, thereby controlling for age, gender, baseline value of pain catastrophizing or internal control of pain, treatment center, and variables that turned out to be unequally divided between treatment groups at baseline. When the change in pain catastrophizing or internal control was not significantly different between the WL and 1 of the 3 active treatments ($P < .05$), the procedure was terminated at this point because this variable could no longer be regarded as a potential mediating factor for that particular active treatment.

The second multiple regression analysis was performed by using the outcome measure immediately post-treatment as dependent variable and treatment as independent variable, thereby controlling for age, gender, baseline value of outcome measure, baseline score for the mediator, treatment center, and variables that turned out to be unequally divided between treatment group at baseline. The WL was the reference treatment.

The final regression model was analyzed with the putative mediating factor added to the independent variables of the second model and the outcome measure immediately post-treatment as the dependent variable. Mediation was established when the regression coefficient of the effect of treatment on the outcome measure with the mediating factor taken into the analysis was substantially lower than the regression coefficient of treatment in the second regression analysis. The change in regression coefficients for treatment between models 2 and 3 was tested for significance by using Sobel's *t* test.[3,29,43]

# Results

## Randomization Check and Group Dependence

The baseline characteristics are shown in Table 1. Demographic variables showed similar distribution for all treatment groups ($P > .10$), except for gender, which was controlled for in the analyses. Regarding disease characteristics, only the duration of disability showed a significant difference between treatment groups and was entered into the regression analyses as a covariate.

The multilevel regression analyses showed that the dependence within randomization groups was usually small, intraclass correlations being never larger than .10 with only one exception; the second regression model on the patient-specific complaints showed an intraclass correlation of .15.

## Compliance and Integrity

When applying the criterion of at least 2/3 attendance of all possible sessions, 84.6% of all patients attending the APT had a training of sufficient intensity. Of all CBT patients, 81.8% and 80% had a sufficient number of sessions of GA and PST, respectively. For the CT patients, 80% had sufficient physical training and 69.1% sufficient GA and PST.

Further analysis of the records showed that the APT therapists seemed to have adhered very well to the treatment manual. They kept accurate records of the adjustments of the training and explained the reasons for adjustment carefully. Adjustments were also in accordance with the treatment manual. Analysis of the records of the GA therapists and graphs filled out by the patients showed that the selection of 3 activities, the activity tolerance level, and the gradual increase or pacing of these activities were satisfactory overall. The same applied to the PST therapists.

The independent researcher analyzed the observations (on-site, video and audio tapes) and concluded that the APT therapists did not use additional decatastrophizing methods, and the PST and GA therapists did not incorporate physiologic training principles.

## Treatment Effects on Mediating and Outcome Measures

### Mediating Factors

In Table 2, the mean PCL scores in the WL at post-treatment and the mean difference of this score for each active treatment in comparison to the WL are presented.

Pain catastrophizing significantly decreased in all 3 active therapies when compared with the WL. Because internal control of pain did not change significantly in all active treatments and therefore could not be a mediating factor, no further analyses for this factor were carried out.

### Outcome Measures

The changes of outcome measures in each active treatment in comparison with the post-treatment value of the

**Table 2. Effects of APT, CBT, and CT as Compared With WL on Putative Mediating Factors and Outcome Measures and Effect Sizes**

| *Dependent Variable* | *WL, Mean ± SD* | *APT, Mean Difference (95% CI)** | *APT, Effect Size (95% CI)* | *CBT, Mean Difference (95% CI)** | *CBT, Effect Size (95% CI)* | *CT, Mean Difference (95% CI)* | *CT, Effect Size (95% CI)* |
|---|---|---|---|---|---|---|---|
| PCL-pain catastrophizing | 38.71 ± 12.94 | −5.18 (−8.73 to −1.62)† | .43 (.13 to .72) | −4.23 (−7.79 to −.67)‡ | .35 (.06 to .64) | −4.23 (−7.73 to −.73)‡ | .35 (.06 to .63) |
| PCL-internal control of pain | 16.16 ± 3.65 | .37 (−1.20 to 1.94) | .10 (−.31 to .51) | .69 (−.86 to 2.23) | .18 (−.23 to .53) | .70 (−.84 to 2.24) | .18 (−.22 to 0.59) |
| RDQ | 13.91 ± 4.82 | −2.38 (−4.19 to −.56)‡ | .44 (.10 to .77) | −3.09 (−4.89 to −1.28)† | .56 (.23 to .89) | −2.50 (−4.29 to −.72)† | .46 (.13 to .78) |
| Current pain | 53.35 ± 22.6 | −9.14 (−17.74 to −.55)‡ | .36 (.02 to .70) | −15.64 (−24.23 to −7.06)† | .61 (.28 to .95) | −8.60 (−17.08 to −.11)‡ | .34 (.01 to .67) |
| PSC | 74.59 ± 14.59 | −10.31 (−19.38 to −1.23)‡ | .49 (.06 to .92) | −15.55 (−24.51 to −6.60)† | .74 (.31 to 1.17) | −16.97 (−25.85 to −8.08)† | .81 (.39 to 1.23) |
| BDI | 9.63 ± 7.89 | −2.09 (−3.86 to −.32)‡§ | .32 (.05 to .59) | −1.65 (−3.42 to .12) | .25 (−.02 to .52) | .04 (−1.73 to 1.79) | −.01 (−.27 to .26) |

Mean differences, confidence intervals (CIs), and corresponding *P* values were estimated while adjusting for age, gender, treatment center, baseline score of outcome measure, duration of disability, and dependence within randomization groups.

Abbreviations: APT, active physical therapy; CBT, cognitive-behavioral therapy; CT, combined therapy; WL, waiting list; SD, standard deviation; PCL, Pain Cognition List; RDQ, Roland Disability Questionnaire; PSC, patient-specific complaints; BDI, Beck Depression Inventory.

*Values are the mean difference between treatment and WL; WL score is the score at posttreatment.

†$P < .01$.

‡$P < .05$.

§APT > CT, $P < .05$.

**Table 3. Regression Coefficients and Standard Errors (SEs) of 3 Successive Multiple Regression Analyses on the Mediation Effect of Pain Catastrophizing on Disability, Pain, Patient-Specific Complaints, and Depression**

| | Regression Coefficient 1 | SE | Regression Coefficient 2 | SE | Regression Coefficient 3 | SE | Sobel's t | P Value |
|---|---|---|---|---|---|---|---|---|
| RDQ | | | | | | | | |
| APT vs WL | −5.252 | 1.745 | −2.415 | .905 | −1.284 | .820 | −2.754 | .006 |
| CBT vs WL | −4.214 | 1.749 | −3.140 | .905 | −2.217 | .814 | −2.269 | .023 |
| CT vs WL | −4.158 | 1.718 | −2.524 | .889 | −1.636 | .801 | −2.278 | .022 |
| Current pain (VAS) | | | | | | | | |
| APT vs WL | −5.192 | 1.791 | −9.282 | 4.287 | −4.703 | 4.042 | −2.526 | .012 |
| CBT vs WL | −4.159 | 1.794 | −15.826 | 4.302 | −12.165 | 4.027 | −2.103 | .035 |
| CT vs WL | −4.043 | 1.766 | −8.685 | 4.231 | −5.124 | 3.958 | −2.081 | .037 |
| PSC* | | | | | | | | |
| APT vs WL | −5.104 | 1.779 | −10.389 | 4.536 | −6.723 | 4.313 | −2.472 | .013 |
| CBT vs WL | −4.177 | 1.782 | −15.647 | 4.489 | −12.665 | 4.246 | −2.102 | .036 |
| CT vs WL | −4.110 | 1.759 | −17.010 | 4.436 | −14.192 | 4.200 | −2.096 | .036 |
| BDI*† | | | | | | | | |
| APT vs WL | −4.803 | 1.711 | −2.010 | .901 | −.605 | .751 | −2.698 | .007 |

*NOTE.* Model 1, pain catastrophizing as dependent variable and treatment as independent variable; model 2, outcome as dependent variable and treatment as independent variable; model 3, outcome as dependent variable and treatment as well as pain catastrophizing as independent variables.

Abbreviations: APT, active physical therapy; CBT, cognitive-behavioral therapy; CT, combined therapy; WL, waiting list; RDQ, Roland Disability Questionnaire; PSC, patient-specific complaints; BDI, Beck Depression Inventory; VAS, visual analog scale.

*One patient did not complete PSC at post-treatment; 2 patients did not complete BDI at pretreatment.

†Data for CBT vs WL and CT vs WL not presented because there was no significant difference on BDI score.

WL, as well as effect sizes, are shown in Table 2. Significant differences between CT and APT or CBT, respectively, are indicated. Although the active treatments were quite different, all 3 treatments were apparently equally effective regarding the reduction of disability, patient-specific complaints, and current pain. APT was the only treatment that was effective in reducing the depression score, even when compared with the CT. Because CBT and CT did not significantly change the BDI score in comparison with the WL, no further analyses regarding mediation on BDI for these 2 treatments were performed.

No other significant differences between CT and the other 2 active treatments were found.

### Change in Pain Catastrophizing as a Mediating Factor of Treatment Outcome

Table 3 displays the regression coefficients and their standard errors as well as Sobel's *t* test and level of significance for the change of the coefficient after correction for pain catastrophizing for each active therapy when compared with the WL group. Correcting for pain catastrophizing significantly changed the regression coefficients in APT, CBT, and CT for the RDQ, patient-specific complaints, and current pain, suggesting that pain catastrophizing mediated the decrease in disability, major complaints, and pain. Because APT was the only treatment that showed a significant improvement regarding depression when compared with WL, the mediating role of pain catastrophizing on depression could only be investigated and was also established for this treatment.

## Discussion

The aim of this study was to test whether pain catastrophizing and internal control of pain mediated the outcome of a physical treatment, a cognitive-behavioral treatment, and a combined treatment in CLBP. While being compared with the waiting list, all 3 active treatments significantly decreased the level of pain catastrophizing but unexpectedly not the level of internal control of pain. The active treatments also showed a significant decrease of disability, current pain, and patient-specific complaints. The depression score only significantly decreased in the physical treatment (APT). The reduction of pain catastrophizing significantly mediated disability, patient-specific complaints, and current pain when the patients received either one of the active treatments. In the APT, pain catastrophizing also mediated the level of depression, but it should be noted that the mean baseline score of the BDI was already relatively low (mean score, 10.1 ± 7.18), and by using the cutoff score of ≥21 as recommended by Geisser et al,[15] only 7.6% of all patients could be classified as being depressed at baseline. Therefore, the clinical relevance of the improvement of the BDI score might be questioned.

In general, the overall effect sizes for the outcome measures are comparable to the results presented in recent reviews and meta-analysis on different active treatments.[17-19,31,37]

The results regarding the effect of CBT and CT on pain catastrophizing are comparable with the results of a previous RCT in CLBP[44] and an uncontrolled study in fibromyalgia.[34] The mean effect sizes of pain catastrophizing

are also comparable to the overall mean effect size of negative coping in a meta-analysis on CBT for chronic pain.[31]

In another study comparing a cognitive-behavioral treatment, an operant-behavioral treatment, and a waiting list control group, the use of coping strategies (pain catastrophizing decreased and use of hoping and praying increased) was an important factor in the final treatment results.[51] Interestingly, this change was not only found in the cognitive-behavioral treatment, which was explicitly aimed at increasing the use of these coping skills, but also in the operant-behavioral treatment. In contrast, Ostelo et al[35] found that a behavioral-GA program and care as usual in patients with LBP of more than 6 weeks after first time lumbar disk surgery did not significantly change pain catastrophizing in both groups. In an uncontrolled study of 54 patients of the original 83 patients who started a CBT program, Woby et al[59] showed that changes in increased perception of pain control were related to the reduction of disability and not changes in pain catastrophizing.

This is the first study showing that the effects of a physical treatment in which no cognitive-behavioral treatment elements were incorporated seemed to be mediated by the decrease in pain catastrophizing. One explanation might be that patients were exposed to a number of rigorous activities, thereby challenging the idea that pain is a sign of impending threat. Similar findings were reported by Mannion et al,[27] who found that an active therapy program appeared capable of modifying fear-avoidance beliefs, possibly as a result of the positive experience of completing the prescribed exercises without undue harm.

It was suggested earlier that changes in pain impact and pain catastrophizing occurred after improved motor functioning during physical training,[54] which is in line with the more general assumption that cognitions can best be altered by treatment techniques producing changes in motor behavior.[4] This change in motor functioning might have resulted in increased confidence in one's own body and consequently created greater possibilities regarding performing activities. Otherwise, it is possible that increased physical capacity (muscle strength, muscle endurance, and aerobic capacity) might have had a positive effect on performing activities, which eventually resulted in a reduced level of pain catastrophizing.

Apparently the interventions that are supposed to alter maladaptive thoughts and improve coping with pain and pain disability did not show a greater decrease of pain catastrophizing than the physical treatment. It is possible that when combined with the operant behavioral GA treatment, the specific cognitive training element (PST) was not able to also change pain catastrophizing. A recent study showed that the same PST added to an operant GA treatment did have an additional effect in decreasing sick leave and disability in patients with subacute sick leave caused by LBP, although no differential effects were found for pain catastrophizing, which decreased in both interventions.[52] One possibility for the lack of differential effects in our study might be the compliance rate. A substantially high number of patients did not attend sufficient sessions of PST and GA (20% in CBT and 31% in CT). On the other hand, in APT and CT, 15% and 20%, respectively, of all patients also did not attend sufficient physical training sessions. Although this difference in attendance rate between APT and CBT/CT, respectively, is not very substantial, we cannot rule out that this is a possible reason why APT is equally effective in decreasing pain catastrophizing.

Another explanation might be insufficient treatment integrity. Although the independent observer did not encounter the use of physiologic training principles in the GA and PST and all therapists had at least 5 years of experience in treating CLBP patients and attended training and supervision sessions throughout the study, it should be taken into account that the observations were performed by only one independent observer, and the reliability of his observations has not been tested. Therefore, it cannot be totally ruled out that the therapist did use the most proper treatment principles. Otherwise, compared with the study of van den Hout et al,[52] our CBT and CT showed at least an equal and even a tendency to a larger decrease of pain catastrophizing, indicating at least a comparable effectiveness.

A further possibility might be that the cognitive changes described in the abovementioned studies on CBT were not caused by the CBT specifically. In these multidisciplinary treatments, also physical and occupational therapies were incorporated, which could have caused cognitive changes. Several other studies suggested that improvements in physical capacities (muscle strength, cardiovascular capacity, etc) indeed account for favorable outcomes.[10,20,28,30] Furthermore, the present study results might be caused at least partially by nonspecific factors of the treatment such as a clear treatment rationale, a highly structured and standardized treatment program, and emphasis on active participation and responsibility for generalization.[26,44]

In conclusion, this study showed that treatment elements that do not deliberately target cognitive factors but instead concentrate on letting the patients experience that performing physical exercises or normally hampered activities in a controlled environment is still possible are responsible for a substantial cognitive change.

The lack of change in internal control of pain was not expected. This might be attributed to the fact that the cognitive treatment consisting of the PST and the operant behavioral GA training was not specifically aimed at changing the internal control of pain. In the study of Spinhoven et al,[44] which did show an increase of control of pain, the cognitive treatment specifically aimed at increasing pain control and self-efficacy beliefs. Another explanation might be the internal control of pain subscale of the PCL itself. Currently there is no information about the responsiveness of this scale. In addition, the baseline score seemed already moderate to high in all treatment groups (mean score ranged from 15.6 to 16.6). However, in the absence of normative values for the

healthy population, it is difficult to judge whether there existed a ceiling effect for this measure.

A weakness of this study and most previous studies alike is that the results are based on self-report only, which might be influenced by a number of self-serving biases. On the other hand, by using the 3-step process for assessing mediation as described by Baron and Kenny[5] and statistically testing the results with Sobel's *t* test, the mediating effect of pain catastrophizing seems quite solid. To examine whether we are not merely measuring correlation and that the change in mediator really precedes the change in outcome, Burns et al[11,12] suggested using cross-lagged correlation. In their uncontrolled study on chronic pain patients attending a multidisciplinary treatment, they collected data on pain catastrophizing, helplessness, depression, and the outcome (multidimensional pain inventory) at before, during, and after treatment. An early change in cognitions appeared to predict late treatment change of pain and disability but not the other way around.

Although we performed a controlled study, we were not able to collect data at mid-treatment, and a cross-lagged correlation was not possible. Therefore, the direction of causality among the variables remains ambiguous. Taking the results of Burns et al[11,12] into consideration, it seems that the direction of mediation is from pain catastrophizing to outcome measures and not the other way around. More research, especially on temporal precedence of factors that mediate the effects of our rehabilitation treatments for CLBP, is warranted. Once we have detected the most potent mediators, we can start to develop treatments that show the best results in changing these mediators. It might contribute to the development of interventions that have increased cost-effectiveness and can be geared onto the mechanisms that produce the effects at which we aim.[57]

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## References

1. ACSM: ACSM position stand on the recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in adults. Med Sci Sports Exerc 30:975-991, 1998

2. Aldrich S, Eccleston C, Crombez G: Worrying about chronic pain: Vigilance to threat and misdirected problem solving. Behav Res Ther 38:457-470, 2000

3. Aroian LA: The probability function of the product of two normally distributed variables. Annals of Mathematical Statistics 18:265-271, 1944

4. Bandura A: Self-efficacy: towards a unifying theory of behaviour change. Psychol Rev 84:191-215, 1977

5. Baron RM, Kenny DA: The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. J Pers Soc Psychol 51: 1173-1182, 1986

6. Beck AT, Rush AJ, Shaw BF, Emmery G: Cognitive Therapy of Depression. New York, NY, Wiley and Sons, 1979, pp 389-399

7. Beck AT, Steer RA, Garbin MG: Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. Clin Psychol Rev 8:77-100, 1988

8. Beurskens AJ, de Vet HC, Koke AJ: Responsiveness of functional status in low back pain: A comparison of different instruments. Pain 65:71-76, 1996

9. Beurskens AJ, de Vet HC, Koke AJ, Lindeman E, van der Heijden GJ, Regtop W, Knipschild PG: A patient-specific approach for measuring functional status in low back pain. J Manipulative Physiol Ther 22:144-148, 1999

10. Burns JW, Johnson BJ, Mahoney N, Devine J, Pawl R: Cognitive and physical capacity process variables predict long-term outcome after treatment of chronic pain. J Consult Clin Psychol 66:434-439, 1998

11. Burns JW, Glenn B, Bruehl S, Harden RN, Lofland K: Cognitive factors influence outcome following multidisciplinary chronic pain treatment: A replication and extension of a cross-lagged panel analysis. Behav Res Ther 41:1163-1182, 2003

12. Burns JW, Kubilus A, Bruehl S, Harden RN, Lofland K: Do changes in cognitive factors influence outcome following multidisciplinary treatment for chronic pain? A cross-lagged panel analysis. J Consult Clin Psychol 71:81-91, 2003

13. Carlsson AM: Assessment of chronic pain. I. Aspects of the reliability and validity of the visual analog scale. Pain 16:87-101, 1983

14. Fordyce WE: Behavioral Methods for Chronic Pain and Illness. St Louis, Mo, Mosby, 1976

15. Geisser ME, Roth RS, Theisen ME, Robinson ME, Riley JL 3rd: Negative affect, self-report of depressive symptoms, and clinical depression: relation to the experience of chronic pain. Clin J Pain 16:110-120, 2000

16. Gommans IHB, Koes BW, van Tulder MW: Validiteit en responsiviteit Nederlandstalige Roland Disability Questionnaire: Vragenlijst naar functionele status bij patiënten met lage rugpijn. Ned T Fysiother 107:28-33, 1997

17. Guzman J, Esmail R, Karjalainen K, Malmivaara A, Irvin E, Bombardier C: Multidisciplinary bio-psycho-social rehabilitation for chronic low back pain. Cochrane Database Syst Rev: CD000963, 2002

18. Hayden JA, van Tulder MW, Malmivaara AV, Koes BW: Meta-analysis: Exercise therapy for nonspecific low back pain. Ann Intern Med 142:765-775, 2005

19. Hayden JA, van Tulder MW, Tomlinson G: Systematic review: Strategies for using exercise therapy to improve outcomes in chronic low back pain. Ann Intern Med 142:776-785, 2005

20. Hazard RG, Fenwick JW, Kalisch SM, Redmond J, Reeves V, Reid S, Frymoyer JW: Functional restoration with behav-

ioral support: A one-year prospective study of patients with chronic low-back pain. Spine 14:157-161, 1989

21. Hedges LV, Olkin I: Statistical Methods for Meta-analysis. Orlando, FL, Academic Press, 1985

22. Jensen IB, Bergstrom G, Ljungquist T, Bodin L, Nygren AL: A randomized controlled component analysis of a behavioral medicine rehabilitation program for chronic spinal pain: Are the effects dependent on gender? Pain 91:65-78, 2001

23. Jensen MP, Turner JA, Romano JM, Lawler BK: Relationship of pain-specific beliefs to chronic pain adjustment. Pain 57:301-309, 1994

24. Jensen MP, Turner JA, Romano JM: Changes in beliefs, catastrophizing, and coping are associated with improvement in multidisciplinary pain treatment. J Consult Clin Psychol 69:655-662, 2001

25. Keefe FJ, Caldwell DS, Williams DA, Gil KM, Mitchell D, Robertson C, Martinez S, Nunley J, Beckham JC, Crisson JE, Helms M: Pain coping skills training in the management of osteoarthritic knee pain: A comparative study. Behav Ther 21:49-62, 1990

26. Kole-Snijders AM, Vlaeyen JW, Goossens ME, Rutten-van Molken MP, Heuts PH, van Breukelen G, van Eek H: Chronic low-back pain: What does cognitive coping skills training add to operant behavioral treatment? Results of a randomized clinical trial. J Consult Clin Psychol 67:931-944, 1999

27. Mannion AF, Müntener M, Taimela S, Dvorak J: Comparison of three active therapies for chronic low back pain: Results of a randomized clinical trial with one-year follow-up. Rheumatology 40:772-778, 2001

28. Mayer TG, Polatin P, Smith B, Smith C, Gatchel R, Herring SA, Hall H, Donelson RG, Dickey J, English W: Spine rehabilitation: Secondary and tertiary nonoperative care. Spine 20: 2060-2066, 1995

29. McKinnon DP, Lockwood C, Hoffman JM, West SG, Sheets V: A comparison of methods to test mediation and other intervening variable effects. Psychol Methods 7:81-104, 2002

30. Mellin G, Harkapaa K, Hurri H, Jarvikoski A: A controlled study on the outcome of inpatient and outpatient treatment of low back pain. Part IV. Long-term effects on physical measurements. Scand J Rehabil Med 22:189-194, 1990

31. Morley S, Eccleston C, Williams A: Systematic review and meta-analysis of randomized controlled trials of cognitive behaviour therapy and behaviour therapy for chronic pain in adults, excluding headache. Pain 80:1-13, 1999

32. Nezu AM: Efficacy of a social problem-solving therapy approach for unipolar depression. J Consult Clin Psychol 54: 196-202, 1986

33. Nezu AM, Perri MG: Social problem-solving therapy for unipolar depression: An initial dismantling investigation. J Consult Clin Psychol 57:408-413, 1989

34. Nielson WR, Jensen MP: Relationship between changes in coping and treatment outcome in patients with fibromyalgia syndrome. Pain 109:233-241, 2004

35. Ostelo RW, de Vet HC, Berfelo MW, Kerckhoffs MR, Vlaeyen JW, Wolters PM, van den Brandt PA: Effectiveness of behavioral graded activity after first-time lumbar disc surgery: Short term results of a randomized controlled trial. Eur Spine J 12:637-644, 2003

36. Ostelo RW, de Vet HC, Knol DL, van den Brandt PA: 24-item Roland-Morris Disability Questionnaire was preferred out of six functional status questionnaires for post-lumbar disc surgery. J Clin Epidemiol 57:268-276, 2004

37. Ostelo RW, Tulder M, Vlaeyen J, Linton S, Morley S, Assendelft W: Behavioural treatment for chronic low-back pain. Cochrane database of systematic reviews Online Update Software:Cd002014, 2005

38. Revill SI, Robinson JO, Hogg MIJ: The reliability of a linear analog for evaluating pain. Anaesthesia 31:1191-1198, 1976

39. Roland M, Morris R: A study of the natural history of back pain. Part I: Development of a reliable and sensitive measure of disability in low-back pain. Spine 8:141-144, 1983

40. Rosenstiel AK, Keefe FJ: The use of coping strategies in chronic low back pain patients: Relationship to patient characteristics and current adjustment. Pain 17:33-44, 1983

41. Sanders SH: Operant conditioning with chronic pain: Back to basics, in Gatchel RJ, Turk DC (eds): Psychological Approaches to Pain Management: A Practitioner's Handbook. New York, NY, The Guilford Press, 1996, pp 112-130

42. Smeets RJEM, Vlaeyen JWS, Hidding A, Kester ADM, van der Heijden GJMG, van Geel ACM, Knottnerus JA: Active rehabilitation for chronic low back pain: Cognitive-behavioral, physical, or both? First direct post-treatment results from a randomized controlled trial [ISRCTN22714229]. BMC Musculoskel Dis 7:5, 2006

43. Sobel ME: Asymptomatic confidence intervals for indirect effects in structural equations models, in Leinhart W (ed): Sociological Methodology 1982. San Francisco, Calif, Jossy-Bass, 1982, pp 290-312

44. Spinhoven P, Ter Kuile M, Kole-Snijders AM, Hutten Mansfeld M, Den Ouden DJ, Vlaeyen JW: Catastrophizing and internal pain control as mediators of outcome in the multidisciplinary treatment of chronic low back pain. Eur J Pain 8:211-219, 2004

45. Sriwatanakul K, Kelvie W, Lasagna L, Calimlim JF, Weis OF, Mehta G: Studies with different types of visual analog scales for measurement of pain. Clin Pharmacol Ther 34:234-239, 1983

46. Stratford PW, Binkley J, Solomon P, Gill C, Finch E: Assessing change over time in patients with low back pain. Phys Ther 74:528-533, 1994

47. Stratford PW, Binkley JM, Riddle DL, Guyatt GH: Sensitivity to change of the Roland-Morris Back Pain Questionnaire: Part 1. Phys Ther 78:1186-1196, 1998

48. Sullivan MJ, Rodgers WM, Kirsch I: Catastrophizing, depression and expectancies for pain and emotional distress. Pain 91:147-154, 2001

49. Tugwell P, Bombardier C, Buchanan WW, Goldsmith CH, Grace E, Hanna B: The MACTAR Patient Preference Disability Questionnaire: An individualized functional priority approach for assessing improvement in physical disability in clinical trials in rheumatoid arthritis. J Rheumatol 14:446-451, 1987

50. Turk DC, Okifuji A: Psychological factors in chronic pain: Evolution and revolution. J Consult Clin Psychol 70:678-690, 2002

51. Turner JA, Clancy S: Comparison of operant behavioral and cognitive-behavioral group treatment for chronic low back pain. J Consult Clin Psychol 56:261-266, 1988

52. van den Hout JH, Vlaeyen JW, Heuts PH, Zijlema JH,

Wijnen JA: Secondary prevention of work-related disability in nonspecific low back pain: Does problem-solving therapy help? A randomized clinical trial. Clin J Pain 19:87-96, 2003

53. Vlaeyen JWS, Geurts SM, Kole-Snijders AMJ, Schuerman JA, Groenman NH, van Eek H: What do chronic pain patients think of their pain? Towards a pain cognition questionnaire. Br J Clin Psychol 29:383-394, 1990

54. Vlaeyen JWS, Haazen IW, Schuerman JA, Kole-Snijders AM, van Eek H: Behavioural rehabilitation of chronic low back pain: Comparison of an operant treatment, an operant-cognitive treatment and an operant-respondent treatment. Br J Clin Psychol 34:95-118, 1995

55. Vlaeyen JWS, Kole-Snijders AM, Rotteveel AM, Ruesink R, Heuts PHTG: The role of fear of movement/(re)injury in pain disability. J Occup Rehabil 5:235-252, 1995

56. Vlaeyen JWS, van Breukelen G, Nooyen-Haazen IWCJ, Stomp-van den Berg SGM, Kole-Snijders AMJ: De Pijn Cognitie Lijst. Maastricht, the Netherlands, Pijn Kennis Centrum academisch ziekenhuis Maastricht, 2003

57. Vlaeyen JWS, Morley S: Cognitive-behavioral treatments for chronic pain: What works for whom? Clin J Pain 21:1-8, 2005

58. Wallston KA, Wallston BS, DeVellis R: Development of the Multidimensional Health Locus of Control (MHLC) Scales. Health Educ Monogr 6:160-170, 1978

59. Woby SR, Watson PJ, Roach NK, Urmston M: Are changes in fear-avoidance beliefs, catastrophizing, and appraisals of control, predictive of changes in chronic low back pain and disability? Eur J Pain 8:201-210, 2004

60. Zitman FG, Griez EJL, Hooijer C: Standaardisering depressievragenlijsten. Tijdschr Psychiatr 31:114-135, 1989