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Purpose: We illustrate the importance of examining whether treatments work either uniquely via mechanisms specified by theory or via mechanisms common to different treatments.

Methods: Secondary data analysis was conducted to examine the effects of CBT and Pain Education mechanisms.

Results: Generally, reductions in catastrophizing were significantly related to outcome improvements irrespective of condition. Knowledge acquired predicted decreases in pain intensity and disability in Pain Education only.

Conclusions: Results underscore the need to assess whether our presumed mechanisms predict outcomes, and illustrate the importance of broadening the assessment of mechanisms beyond those specified by theory. Theory-specific, competing, and common mechanisms must all be assessed to determine why our treatments work.

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Unique and Common Therapeutic Mechanisms in Psychosocial Treatments for Chronic Pain: A Conceptual and Empirical Exploration

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Implications

Results underscore the need to assess whether our presumed mechanisms predict outcomes, and illustrate the importance of broadening the assessment of mechanisms beyond those specified by theory. Identifying and distinguishing the mechanisms that are the true active ingredients of ostensibly different treatments could lead to streamlined and efficient interventions that maximize efficacy. To show the true public health value of psychosocial pain treatments to our constituents, we must be able to demonstrate not only that they produce desirable outcomes, but that they do so exactly because of the time- and energy-consuming therapeutic procedures that the interventions entail.
Abstract

Background: Mechanisms underlying favorable outcomes of psychosocial interventions for chronic pain are unclear. Theory suggests changes in maladaptive cognitions represent therapeutic mechanisms specific to cognitive-behavioral therapy (CBT). Acquired knowledge of pain-related information may represent a mechanism specific to Pain Education.

Purpose: We illustrate the importance of examining whether treatments work either uniquely via mechanisms specified by theory or via mechanisms common to different treatments.

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Conclusions: Results underscore the need to assess whether our presumed mechanisms predict outcomes, and illustrate the importance of broadening the assessment of mechanisms beyond those specified by theory. Theory-specific, competing, and common mechanisms must all be assessed to determine why our treatments work.
Introduction

Chronic pain is a disabling and costly experience for millions of Americans each year.[1] Evidence indicates that a variety of psychosocial interventions are efficacious in reducing pain and suffering and improving general function for people with chronic pain.[e.g.,2,3,4,5,6] The efficacy of cognitive-behavioral therapy (CBT), in particular, has been amply demonstrated for a heterogeneous group of chronic pain conditions,[3,7,8,9,10,11,12] and appears cost effective relative to surgery and medication management.[2] Although extensive findings suggest that CBT is more efficacious than wait-list controls, relatively little attention has focused on the therapeutic mechanisms by which CBT brings about favorable outcomes. As Kopta and colleagues stated in response to a different but closely related literature, “Hundreds of studies have shown that psychotherapy works better than nothing. What is not clear is whether it works for reasons specified by theory (p.3).”[13]

Cognitive-behavioral theory posits that appraisals and interpretations of pain-related stimuli, events and experiences will influence perceived chronic pain severity and level of functioning. A critical ingredient of CBT, based on cognitive-behavioral theory, is that the alteration of maladaptive and irrational pain-related cognitions (to make them more positive and realistic) will lead to reduced pain and improved functioning.[14] As such, reduction in maladaptive appraisals of pain, wrought via cognitive restructuring, is theorized to constitute a therapeutic mechanism for CBT. Research confirms that pain-related cognitions are related to both perceived acute and chronic pain severity, as well as adjustment to chronic pain conditions.[e.g.,15,16,17,18,19,20,21] Research also supports the notion that changes in pain-related cognition may represent a therapeutic mechanism. Maladaptive appraisals have been shown to decrease from pre- to post-CBT treatment, and these changes in cognition correlate with pre- to post-treatment change scores in outcome factors in expected directions.[22,23,24,25] Finally, limited findings using cross-lagged analyses further support the mechanistic role of pain
catastrophizing by showing that early-treatment reductions in pain catastrophizing predict to some extent late-treatment improvements in outcome factors, albeit in an interdisciplinary chronic pain program that included but was not exclusively CBT.[26,26] However, the evidence that cognitive change represents a therapeutic mechanism for CBT for chronic pain comes exclusively from longitudinal designs using correlation methods, and so is far from conclusive regarding any causal role of cognitive change. Much more work using different approaches is sorely needed.

Another method to examine whether CBT works via theoretically-specified cognitive mechanisms is to combine the experimental rigor of a Randomized Controlled Trial (RCT) with correlational methods. Theoretically and procedurally, CBT is closely tied to reductions in maladaptive cognitions. Cognitive change is presumed to be an important therapeutic mechanism of CBT and it is also assumed that cognitive change is a mechanism specific to CBT. Thus, if CBT works via reductions in maladaptive pain-related cognitions, we would expect that changes in such factors would be related to changes in pain and function. A corollary of this proposition is that other theoretically distinct treatments will work primarily via mechanisms other than cognitive changes. Hence, change in cognitive factors should be related to outcome changes to a greater extent in CBT than in another active treatment. However, results of Smeets and colleagues - who conducted secondary analyses of an RCT comparing CBT, active physical treatment (i.e., aerobic and strength training), CBT plus active physical treatment and a wait-list control - suggest otherwise.[28] They found that the three active treatments did not differ significantly on pre- to post-treatment change in pain catastrophizing, and that pre- to post-treatment changes in pain catastrophizing equivalently predicted pre- to post changes in most outcomes.

In the present study, we addressed the issues of mechanism discussed above and evaluated by Smeets et al. [28] and sought also to extend their findings. One limitation of the method described by Smeets et al.,[28] was that they only assessed a cognitive mechanism that was theoretically linked to the
treatment conditions featuring CBT. Assessing mechanisms theoretically linked to other active treatments – in their case, measures of aerobic capacity and/or trunk strength – would allow a more comprehensive test of mechanism specificity. In an effort to provide a springboard for further research specifically designed to address this limitation, we conducted secondary analyses of data from an RCT that compared CBT to an active control condition – Pain Education. This recently reported outcome study examined the feasibility and efficacy of a literacy-adapted, culturally sensitive group CBT program in comparison to a similarly adapted Pain Education intervention.[29] Participants were rural, predominantly African-American people with chronic pain, and characterized by low-socioeconomic status (SES) and low-literacy.[30] The CBT intervention was cognitively-focused and designed to specifically target maladaptive cognitions, whereas the Pain Education intervention was designed to provide factual knowledge about pain, without explicit focus on cognitive change. Thus, cognitive changes were expected to operate as a therapeutic mechanism in CBT, but not in Pain Education.

Pain education alone has usually been found to be insufficient to produce favorable outcomes. Several systematic reviews have reported no clinically significant effect of group education based programs, such as “back schools.”[31,32,33] Notable exceptions include studies in which the educational intervention is based on the biopsychosocial model rather than a biomedical model.[29,34,35,36] In the RCT from which data for the present paper was derived, we found that participants in the Pain Education condition significantly improved on primary outcomes to the same degree as those in the CBT condition.[29] While there is no well-established theory underpinning the rationale for pain education, one might propose that an essential mechanism of efficacy is acquired knowledge of pain-relevant information. However, in a treatment such as CBT, knowledge per se may be less crucial than in Pain Education because the former is heavily weighted towards experiential, skills-building exercises. Thus, we expected in the present study that acquisition of pain-related knowledge conveyed by therapists
during sessions would operate as a therapeutic mechanism in Pain Education to a greater extent than in CBT.

In the original study,[29] 61 people completed the 10-week interventions. Pre- and post-treatment measures of outcomes and the Pain Catastrophizing Scale (PCS)[37]) were taken, and knowledge acquisition of treatment session material was assessed at each session. Pain catastrophizing, a negative mental set about anticipated or actual pain,[38] has been shown to be a consistently strong correlate of pain severity, disability, and mood among people suffering from chronic pain.[18,19,20,21,39,40] Insofar as pain catastrophizing is a maladaptive pain-related cognition, reductions in this factor served as an index of change in maladaptive cognition, and therefore also represented a therapeutic mechanism by which CBT allegedly works to reduce pain and improve functioning. Knowledge acquisition related to chronic pain was expected to act as the primary therapeutic mechanism in the Pain Education condition.

If reductions in pain catastrophizing represent a mechanism specific to CBT (i.e., change in pain-related cognition), then we would expect: a) that pre- to post-treatment changes in the PCS would be greater in CBT than in Pain Education; and b) pre- to post-treatment PCS changes to correlate with pre- to post-treatment outcome changes to a greater degree in CBT than in Pain Education. If the extent of pain-related knowledge gained represents a mechanism specific to Pain Education, then we would expect: a) that knowledge acquisition would be greater in Pain Education than in CBT; and b) that knowledge acquisition would correlate with pre- to post-treatment outcome changes to a greater degree in Pain Education than in CBT.

Methods

Trial Design
This trial compared two treatments for a heterogeneous group of chronic pain conditions in a randomized parallel group design: CBT and Pain Education. Initial screening was conducted over the phone, and all assessments and the 10-week interventions took place within the participants’ primary care clinic. This research was approved by the Institutional Review Board at the University of Alabama, and informed consent was obtained with all patients prior to participation. For additional details, see the reported original trial.[29]

Setting and Participants

Participants were recruited from health clinics in three rural Alabama counties. Study inclusion criteria were: (1) 19 years of age or older; (2) reported having experienced pain most days of the month, for the previous 3 months; (3) if currently taking analgesic or psychotropic medication, must have reported being on the same medication for at least 4-weeks prior to baseline assessment; (4) ability to read and write (in English) at the 2nd grade level or higher as determined by the Wide Range Achievement Test-4 reading/word decoding subtest (WRAT-4);[42] (5) have a home telephone or comparable form of communication. Study exclusion criteria included the following: (1) HIV-related pain and cancer pain because these are associated with malignant disease;[43] (2) significant cognitive impairment, evidenced by a positive screen (score of ‘0’ or ‘1 or 2 with an abnormal clock draw test’) on the Mini-cog;[44] (3) other current psychosocial treatments for any pain condition; (4) schizophrenia, bipolar affective disorder, or seizure disorder not adequately controlled by medication, or current substance abuse as these conditions could result in medical emergencies during treatment.

A total of 83 participants were randomized to treatment (49 CBT and 34 Pain Education), and 61 participants (32 CBT and 29 Pain Education) completed treatment. Because the aim of the present secondary analyses was to examine treatment mechanism, the 61 participants who completed all 10 sessions of treatment and all assessments were used in all analyses. The CONSORT participant flow
diagram and a detailed description of the sample can be found in the original trial report.[29]

Additionally, details describing analyses comparing those participants that completed treatment (i.e., the current sample) to those participants that dropped-out (i.e., failed to complete all 10 sessions) may also be found in the original report.[29]

Intervention protocols

The 10-week CBT and Pain Education intervention protocols implemented in the current study were adapted respectively from Thorn and Ehde et al.[45,46] The adaptations addressed the limited literacy of the sample, tailored the program to rural Alabama patients, and remained sensitive to any adjustments based on differences in income, race/ethnicity, and culture (see Kuhajda et al, 2011 for an overview of the adaptation process).[47] Groups were conducted by a licensed clinical psychologist with extensive experience in the treatment of chronic pain, and an advanced graduate student in clinical psychology served as a co-therapist. Therapists in both conditions sought to maximize patient rapport with therapists, group cohesion, and group discussion related to the weekly topics. The duration of each weekly CBT and Pain Education session was 1.5 hours. Participants in both conditions were given a client workbook with materials and handouts they could follow/discuss during sessions, and read between sessions.

CBT Intervention Description. All CBT sessions followed the same format: (1) pre-session process check; (2) review of previous week’s session; (3) homework review; (4) session treatment objectives; (5) worksheet; (6) assign homework; and (7) post-session process check. Homework assignments included instructions to think about and enact the assignments, and to write thoughts and reactions to them. A general outline of the objectives of each CBT session is as follows: Session 1) establish rapport, explain therapy rationale, goals, format and rules, introduce stress-appraisal-pain connection; Session 2) identification of negative automatic thoughts; Session 3) evaluate automatic thoughts for accuracy;
Session 4) challenge distorted automatic thoughts, construct realistic alternative responses; Session 5) identify intermediate belief systems, challenge negative distorted beliefs, construct new beliefs; Session 6) identify core beliefs, challenge negative, distorted core beliefs, construct new, more adaptive beliefs; Session 7) relaxation exercise, positive coping self-statements; Session 8) expressive writing or verbal narration of expressive writing exercise; Session 9) assertive communication; and Session 10) review concepts and skills learned, provide feedback about helpful and challenging aspects of the treatment. All learning objectives were presented by the group leaders and interactive skills-building exercises and group discussion followed. For further details regarding session structure, see Thorn (2004).[45]

**Pain Education Intervention Description.** All Pain Education sessions followed the same format as the CBT sessions (described above); however, homework was not assigned and the education sessions did not include skills-building exercises related to any of the content. All learning objectives were presented by the group leaders and interactive group discussion followed. A general outline of the objectives of each Pain Education session includes: Session 1) establish rapport, explain therapy rationale, goals, format and rules, introduce concepts in chronic pain treatment; Session 2) Gate Control Theory of Pain, which emphasizes the importance of cognitions and affect in the experience of pain; Session 3) costs of chronic pain; Session 4) acute versus chronic pain; Session 5) sleep (i.e., normal sleep, sleep disorders, sleep hygiene); Session 6) depression and other mood changes associated with chronic pain; Session 7) pain behaviors; Session 8) pain and communication (i.e., assertive, aggressive, and passive communication styles); Session 9) working with health care providers; Session 10) stages of change, review concepts learned, provide feedback about helpful and challenging aspects of the treatment. See Ehde et al. (2005) for further details pertaining to the structure of the Pain Education protocol.[46]

It is important to note that a key difference between the CBT groups and the Pain Education groups was that CBT participants were given interactive skills training in the groups, which they were
then expected to practice via at-home activities in the ensuing week. Although Pain Education participants were given pain-relevant information, including information about the importance of cognitions and behavioral coping, they were not given skills training, nor were they given any homework activities.

**Mechanism Measures**

**Pain Catastrophizing.** The Pain Catastrophizing Scale (PCS) was used to assess patient report of catastrophic thinking about pain.[37] The 13-item measure asks respondents to rate, using a 5-point Likert scale ranging from 0 (not at all) to 4 (all the time), the degree to which they have certain thoughts and feelings when experiencing pain. Higher scores indicate greater use of catastrophic thinking. The PCS has exhibited strong internal consistency ($\alpha=.93$), concurrent and discriminant validity, and high test-retest reliability over a 6 wk period ($r = 0.78$).[37,48,49]

**Acquired Knowledge of Session Material.** Pre- and post-session process checks were adapted from Thorn (2004) and were administered by group therapists at each weekly session.[45] The post-session process check functioned as a proxy for treatment receipt and was completed by participants at the conclusion of each session; it consisted of one question asking “What was the main point you got from today’s group?” The pre-session process check functioned as a proxy for treatment retentions and was completed before each session (except session one) and asked the participant “What was the main point you got from last week’s group?” Responses were quantified (0=Inaccurate and 1=Attempted with at least moderate accuracy) by a research assistant not involved in treatment delivery and blind to participant group assignment. A composite of these two measures was created as an overall proxy for acquired knowledge of session material.

**Outcome Measures**
**Pain Intensity and Pain Interference.** These data were collected via the Wisconsin Brief Pain Inventory (BPI), which consists of 11 items that are rated from 0 to 10.\[^{50}\] Pain intensity (BPI-intensity) scores were obtained from the mean of four items, in which respondents rate their most severe pain, least severe pain, average pain over the past week, and current pain on an 11-point Likert scale ranging from 0 (*no pain*) to 10 (*pain as bad as you can imagine*). Pain interference (BPI-interference) scores were obtained from the seven BPI items that request participants to rate interference due to pain in activities such as mood, sleep, etc. on an 11-point Likert scale ranging from 0 (*no interference*) to 10 (*complete interference*). The BPI has adequate internal consistency ($\alpha=.85$) in a variety of pain populations and concurrent validity with other pain instruments.\[^{50,51}\]

**Perceived Disability.** The Roland-Morris Disability Scale-11 item version (RMDS) provided a self-assessment of limitations due to pain in physical activities, such as dressing, standing, bending, walking, and lifting.\[^{52}\] Participants endorse items that have been true over the past month, and a total score (ranging from 0 to 11) is obtained by summing the number of items endorsed. The 11-item version correlates well with scores on longer 18- and 24-item versions ($r=.949$ and $r=.929$ respectively) and has been shown to have adequate reliability that is comparable to the 24-item version ($\alpha=.88$), and strong concurrent validity.\[^{52}\]

**Depression.** The Center for Epidemiological Studies Depression Scale (CES-D), which has been validated for use in chronic pain patients, was used to assess depression.\[^{53}\] The CES-D consists of 20 items; respondents rate the frequency with which each symptom or feeling occurred during the previous 7 days. Item content is rated on a 4-point scale ranging from 0 (*rarely or less than one day*) to 3 (*most or all of the time, 5-7 days*); total scores range from 0 to 60. Higher scores indicate greater depressive symptoms.\[^{54}\] Reliability and validity are reported to be adequate and similar across a variety of samples from the general population.\[^{55}\]
Life Satisfaction. The Quality of Life Scale (QoLS) is a 7-point self-report scale that manifestly assesses life satisfaction in several areas.[56] Total scores range from 7 to 49 with higher scores indicating greater satisfaction. The QoLS has been shown to correlate moderately with distress, and weakly with measures of functioning and pain intensity, indicating the QoLS is measuring a unique construct distinguished from pain and disability. A psychometric analysis of the QoLS showed it to be internally consistent, reliable across time, and representative of a single construct.[56]

Statistical Analyses

To determine whether pre- to post-treatment changes in PCS scores (and other outcome factors) differed in magnitude between CBT and Pain Education groups, a series of ANCOVAs were conducted on post-treatment values controlling for pre-treatment values. The mean of the nine pre- and ten post–session acquired knowledge composites was computed and used in analyses. An ANOVA was conducted to compare CBT and Pain Education groups on knowledge of session material.

To determine whether the relationships between mechanism measures and outcomes differed as a function of Treatment Group, simple change scores were first computed for PCS and outcome variables by subtracting pre-treatment values from post-treatment values. Next, interaction terms were computed by multiplying PCS change scores by a dummy-coded Treatment Group variable (1 = CBT; 2 = Pain Education), and by multiplying the acquired knowledge composite mean by Treatment Group. Hierarchical regressions were performed for each outcome change score by entering the main effect terms in the first step (i.e., PCS change scores and Treatment Group; Acquired Knowledge Composite and Treatment Group), and entering the PCS x Treatment Group or Acquired Knowledge Composite x Treatment Group interaction terms in the second steps. A significant interaction was revealed by a significant increment in $R^2$ for the second step. Interactions were dissected by generating regression equations linking PCS and outcome change scores separately for each Treatment Group.
Results

_Treatment Group Comparisons on PCS, Acquired Knowledge Composite and Outcome Values_

As reported in Thorn et al.,[29] PCS and all outcome variables, with the exception of RMDS (disability) values, changed significantly from pre- to post-treatment with effects sizes ranging from $\eta^2 = .09$ to $\eta^2 = .269$. In the current study, we focused exclusively on the magnitude of treatment group differences. For PCS scores, the post-treatment difference between CBT and Pain Education controlling for pre-treatment values was nonsignificant [$F(1,58) = 1.84; p > .10; \eta^2 = .031$], although adjusted means for the CBT and Pain Education groups were in expected directions (adjusted $M = 22.0, SE = 2.0$; adjusted $M = 25.8, SE = 2.1$; respectively). Results of an ANCOVA comparing CBT ($M = 9.47; SD = 1.4$) and Pain Education ($M = 9.72; SD = .5$) on Acquired Knowledge values were nonsignificant [$F(1,59) < 1; \eta^2 = .015$], although adjusted means for the CBT and Pain Education groups were in expected directions (adjusted $M = 22.0, SE = 2.0$; adjusted $M = 25.8, SE = 2.1$; respectively). Thus, when pre-treatment differences between groups were controlled, CBT did not produce significantly greater decreases in the putative mechanism, pain catastrophizing, than Pain Education. Moreover, Pain Education did not result in greater acquired knowledge than the CBT condition.

For outcome factors, all CBT vs Pain Education comparisons were nonsignificant [$F$’s < 1.92; $p$’s > .10; $\eta^2$’s < .032]. Thus, CBT and Pain Education produced virtually equivalent effects on outcome factors.

_PCS Change x Treatment Group Effects on Outcome Changes_

For CES-D change scores, the PCS Change x Treatment Group interaction was significant ($t = 2.57; p < .01$). Simple effects tests were conducted by running regressions for PCS and CES-D change scores separately for each treatment group. For CBT, the relationship between PCS and CES-D change
scores was significant (β = .60; p < .01), whereas for Pain Education, this association was nonsignificant (β = -.06; p > .10). Thus, reductions in pain catastrophizing were significantly related to reductions in depressive symptoms in the CBT condition only.

PCS Change x Treatment Group interactions were nonsignificant for all other outcome variable change scores (t’s < 1.0; p’s > .10). However, the main effects for PCS changes on QoLS (β = -.36; p < .01), RMDS (β = .44; p < .01), BPI-intensity (β = .37; p < .01) and BPI-interference changes scores (β = .41; p < .01) were all significant. Thus, reductions in pain catastrophizing were significantly related to outcome factor improvements in intensity, interference, life satisfaction, and perceived disability, irrespective of treatment condition.

**Acquired Knowledge x Treatment Group Effects on Outcome Changes**

For BPI-Intensity changes, Acquired Knowledge x Treatment Group interaction was significant (t = 2.08; p < .04). Simple effects tests were again conducted by running regressions for Acquired Knowledge and BPI-Intensity change scores separately for each treatment group. For CBT, the relationship between Acquired Knowledge and BPI-Intensity change scores was nonsignificant (β = .01; p > .10), whereas for Pain Education, this association was significant (β = -.44; p< .02). For RMDS changes, the Acquired Knowledge x Treatment Group interaction approached conventional levels of significance (t = 1.94; p < .06). Simple effects tests showed that for the CBT group, the relationship between Acquired Knowledge and RMDS change scores was nonsignificant (β = .09; p > .10), whereas for Pain Education, this association was significant (β = -.36; p< .05). Thus, greater knowledge acquisition was significantly related to reductions in pain intensity and disability in the Pain Education condition only.

Acquired Knowledge x Treatment Group interactions were nonsignificant for the other outcome variable change scores (t’s < 1.0; p’s > .10). Unlike for PCS changes, the main effects of Acquired Knowledge on the remaining 3 outcomes were also nonsignificant.
Discussion

The study of why psychosocial treatments work or by what mechanisms has fallen far behind the study of efficacy. A limited amount of research has used correlational methods in the context of longitudinal designs, and found evidence that pain catastrophizing changes are related to outcomes in CBT-based interventions.[22,23,24,25] There are very few studies reporting the efficacy of biopsychosocially-oriented pain education interventions for chronic pain, and none prior to the present study have attempted to examine the effects of a theoretically specified mechanism. More importantly, questions of whether theoretically-specified mechanisms (such as catastrophizing in CBT and acquired knowledge in Pain Education) actually produce effects unique to the relevant treatments have been scarcely addressed. In the current study, we combined an RCT approach with correlational methods to address issues not only of mechanisms, but the degree to which theoretically specified mechanisms are active primarily in the treatments that deliberately target them.

If CBT works via reducing maladaptive cognitions, then changes in pain-related cognition should primarily occur and predict outcomes in CBT, and to a significantly lesser degree, occur and predict outcomes in a distinctly different treatment. Further, if Pain Education works via increasing relevant pain-related knowledge, then such increases should primarily occur and predict outcomes in Pain Education. Results in general did not support the proposition that reductions in pain catastrophizing are a therapeutic mechanism specific to CBT, and so replicate results of Smeets et al.[28] Instead, results suggested that while reductions in pain catastrophizing represent an important therapeutic mechanism, these effects may not be limited to CBT. Extending Smeets et al.’s findings to examine a theoretically specific mechanism associated with the other condition in the RCT, we found evidence that knowledge acquisition of pain-related educational material covered by therapists in session may be an important mechanism associated with psychosocial pain education.
Although the mean PCS change score for the Pain Education group was numerically smaller than the mean change score for the CBT group, the two means were not significantly different. The CBT and Pain Education groups also did not differ significantly on the knowledge acquisition means. These null findings could be a consequence of low statistical power with only about 30 people per condition. Indeed, no significant difference between conditions on any change score was found when pre-treatment baseline scores were statistically controlled. The null finding for PCS could also reveal a larger issue in that the intervention designed to target maladaptive cognition through cognitive restructuring produced pre- to post-treatment pain catastrophizing reductions that were virtually equivalent to an educational intervention that did not specifically target cognitive change. To the degree that participants in the Pain Education condition did reduce their pain catastrophizing, they did so via a protocol that did not explicitly aim to change maladaptive cognitions with well-defined cognitive restructuring techniques and skills training featured in the CBT arm. Similarly, knowledge acquisition of session information by participants may simply be a very general, and hoped for, phenomenon of any active intervention.

Further, the degree to which PCS changed from pre- to post-treatment was related significantly to four of five change scores irrespective of condition. These findings combined with results from Smeets et al.,[28] suggest that reductions in the tendency to catastrophize about pain, brought about through distinct treatment protocols, may be a potent albeit broad therapeutic mechanism, not specific to CBT. Consider that pain catastrophizing reductions predicted outcomes in a Pain Education condition in the present study, and predicted outcomes in a physical training condition in Smeets et al.[28] It may be the case therefore, that strict adherence to cognitive restructuring procedures characterized by CBT may simply not be necessary to achieve these therapeutically important changes in the tendency to think the worst about pain. The one exception to the finding that catastrophizing may be a mechanism common to many treatments was that PCS change scores were related uniquely to CES-D change scores.
only in the CBT group. This finding suggests that CBT-induced reductions in catastrophizing uniquely reduced depressive symptoms in this sample of patients with pain. This may have important implications given that previous research has found that poorer outcomes are associated with the treatment of pain when underlying depression goes undiagnosed and untreated. [57]

It should also be noted that, while not a focus, our Pain Education condition did include cognitive and behavioral principles. For example, one session of the Pain Education protocol was devoted to the Gate Control Model of Pain, which presents pain as a multi-dimensional phenomenon and emphasizes the importance of cognitions and emotions in the pain experience. Moseley has reported that a single educational session explaining the neurophysiology of pain can result in cognitive changes that do not occur with standard pain education that only provides descriptive information about structural pathology of the spine. [58] Furthermore, much of the information presented in our education groups was relevant to adaptive coping, and in conjunction with the supportive group environment, may have elicited cognitive change. Thus, diverse intervention approaches may be efficacious partly to the extent that some aspect of treatment effectively reduces pain catastrophizing. Changes in pain-related maladaptive cognitions and beliefs, hypothesized to be a core and unique feature of CBT approaches, may ironically comprise a mechanism common, and potentially even critical to many efficacious interventions.

Of note, in regards to potential mechanisms specific to pain education, we found that an index of acquired knowledge significantly predicted two out of five outcomes only in the Pain Education condition. No outcomes in CBT were predicted significantly by this knowledge index. On one level, these findings point toward the therapeutic value of simply providing accurate and comprehensible information about pain to people suffering from chronic pain. However, the sample in this study was composed of people living in rural areas with low-literacy and predominantly low-SES, and so may have
benefited to a greater extent from an educational intervention than other groups who routinely experience better healthcare and communication with providers. Still, the principle upheld by Moseley and colleagues that accurate information may help change perceptions and beliefs about the nature of pain may apply to a wide variety of SES and ethnic groups.\cite{58}

On another level, results showing that acquired knowledge only predicted outcomes in the Pain Education group are, to our knowledge, unique. First, minimal systematic attention has been devoted to documenting how control conditions exert beneficial effects. Here, we measured what may be a defining aspect of an education condition – degree of knowledge acquisition – and provide evidence that the education itself may aid patients to improve. Second, unlike previous studies, we tested whether two mechanism indexes theoretically specific to CBT or Pain Education showed common and/or unique effects on outcomes. These kinds of methodological and analytical approaches are potentially groundbreaking in that the results produced will shed light both on the importance of specific techniques and on underlying principles that may transcend individual approaches (e.g., changing pain-related cognitions by whatever means).

A number of caveats should be issued. First, this study used a convenience sample derived from a data set designed to specifically test whether psychosocial interventions for chronic pain were feasible in a low-SES, low-literacy, predominantly African-American rural chronic pain population. Thus, results may not generalize to higher-SES, non-minority populations. Second, the original study was not designed a priori to closely examine therapeutic mechanisms. Due to the sample size, statistical power was low to detect differences between treatment conditions on the magnitude of mechanism and outcome variable changes, and was also low to detect differential relationships between mechanism and outcome changes depending on condition. Additionally, as argued by Laurenceau et al.\cite{59} and Thorn and Burns,\cite{41} the study of therapeutic mechanism requires methodological features not part of
our original study. For example, frequent (e.g., weekly) assessments of putative mechanisms and outcomes were not taken during treatment thereby preventing analysis of lagged and cross-lagged effects, and the examination of temporal patterns of change. Third, although we endeavored to use a mechanism index that was theoretically linked to pain education, the knowledge acquisition check was not originally designed to measure a therapeutic mechanism. Unlike the PCS, reliability and validity data have not been generated. Moreover, the composite measure assessed receipt and retention of session material in general, which did not necessarily pertain only to pain-related knowledge. Finally, we measured only putative specific mechanisms, and did not assess nonspecific factors that can be expected to be active ingredients of any psychosocial pain treatment, such as patient expectations of improvement and the quality of the therapeutic relationship. Limitations notwithstanding, the purpose of this study was to help illustrate the importance of mechanism issues. Results point toward fundamental gaps in our knowledge base regarding psychosocial interventions and how they work.

Research that empirically examines the mechanisms by which meaningful change is realized has vast and far-reaching clinical implications. Identifying and distinguishing the mechanisms – specific and nonspecific – that are the true active ingredients of ostensibly different treatments will guide future work to combine these ingredients and to discard the inert ones. Such research programs could lead to streamlined and efficient interventions that maximize efficacy. To achieve this goal, what are now needed are careful, well-constructed investigations that integrate RCTs with lagged correlation methods, which borrow state-of-the art models and methods from psychotherapy process research. Such investigations need to be designed a priori to measure and analyze candidate mechanisms, as well as competing mechanisms (e.g., those specific to Pain Education in the present study) and nonspecific therapeutic mechanisms common to all viable interventions (e.g., the quality of the therapeutic relationship). To show the true public health value of psychosocial pain treatments to our constituents, we must be able to demonstrate not only that they produce desirable outcomes, but that they do so
exactly because of the time- and energy-consuming therapeutic procedures that the interventions entail.
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