Is reduction in pain catastrophizing a therapeutic mechanism specific to cognitive-behavioral therapy for chronic pain?

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ABSTRACT

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). We illustrate the importance of examining whether treatments work either uniquely via mechanisms specified by theory or via mechanisms common to different treatments. Secondary data analysis was conducted to examine the effects of reduction in pain catastrophizing on outcomes following CBT and Pain Education. Generally, reductions in pain catastrophizing were significantly related to outcome improvements irrespective of CBT or Pain Education condition. Results underscore the need to assess whether mechanisms presumed to operate specifically in one treatment do indeed 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.

KEYWORDS

Chronic pain, Cognitive-behavioral therapy, Pain Education, Pain catastrophizing, Mechanisms

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., 2–6]. The efficacy of cognitive-behavioral therapy (CBT), in particular, has been amply demonstrated for a heterogeneous group of chronic pain conditions [3, 7–12] 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 psychother-

Implications

Practice: In order to develop streamlined and efficient interventions that maximize efficacy, there is a need to identify and distinguish the mechanisms that are the true active ingredients of ostensibly different treatments.

Policy: To show the true public health value of psychosocial pain treatments to our constituents, we need to 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.

Research: Continued research is needed to assess whether our presumed mechanisms predict both short- and long-term outcomes, and research is also needed that broadly examines mechanisms beyond those specified by theory.

apy 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 events affect subsequent emotional and behavioral responses [14, 15]. Repeated patterns of irrational or otherwise maladaptive appraisals and interpretations may underlie problematic emotional and behavioral responses (e.g., depression). A tenet of cognitive-behavioral *therapy* is that the alteration of maladaptive patterns of appraisals and interpretations through techniques of cognitive restructuring can lead to a correction of these problematic responses. Cognitive change is therefore a therapeutic mechanism specified by cognitive-behavioral theory to promote adaptive emotional and behavioral responses to events and experiences. The stipulation from cognitive-behavioral theory that cognitive change is crucial for meaningful emotional and behavioral change, coupled with the particular techniques of cognitive restructuring designed expressly to bring about cognitive changes suggests that cognitive change is a therapeutic mechanism *specific* to CBT [16].

An abundance of evidence suggests that pain-related cognitions are related to both perceived acute and chronic pain severity, as well as adjustment to chronic pain conditions [e.g., 17–23]. Pain catastrophizing, a set of negative irrational cognitions about anticipated or actual pain [24], has been shown to be a consistently strong correlate of pain severity, disability, and mood among people suffering from chronic pain [20–23, 25, 26]. Similar to CBT in general, CBT for *chronic pain* holds that the alteration of irrational and maladaptive appraisals and beliefs about pain-related stimuli and events via cognitive restructuring techniques will favorably influence perceived chronic pain severity and level of functioning [27]. Such cognitive changes—such as those of pain catastrophizing—may be seen as a therapeutic mechanism specific to CBT for pain, and findings indeed support this notion.

In CBT-based interventions, maladaptive appraisals have been shown to decrease from pre- to posttreatment, and these cognitive changes have been shown to correlate with pre- to posttreatment changes in pain-related outcomes (e.g., pain intensity, perceived disability, and mood) in expected directions [28–31]. Finally, limited findings using cross-lagged analyses further support the mechanistic role of changes in pain-related cognitions by showing that early treatment reductions in pain catastrophizing predict to some extent late treatment improvements in pain-related outcomes, albeit in an interdisciplinary chronic pain program that included but was not limited to CBT [32, 33]. 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 to fully demonstrate the role of cognitive change in CBT for pain.

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. If CBT works specifically via reductions in maladaptive pain-related cognitions, we would expect that changes in such cognitions would be substantial in CBT compared to control conditions or other kinds of active treatments and would be related to pain-related outcome changes to a greater extent in CBT than in other treatments. 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 [34]. They found that the three active treatments did not differ significantly on pre- to posttreatment change in pain catastrophizing, and that pre- to posttreatment changes in pain catastrophizing equivalently predicted pre- to post-treatment changes in most pain-related outcomes. Contrary to theory and widely held beliefs, these findings suggest that change in

maladaptive cognition may represent a mechanism common to many active treatments that are not necessarily designed to target cognitive change.

In the present study, we conducted a secondary analysis utilizing a combined RCT with correlational methods approach to further investigate the issues of CBT mechanism discussed above. The current research examines the relations evaluated by Smeets et al. [34], in another sample and with a different comparison condition. The recently reported RCT examined the feasibility and efficacy of a literacy-adapted, culturally sensitive group CBT program in comparison to a similarly adapted Pain Education intervention [35]. Participants were rural, predominantly African-American people with chronic pain, and characterized by low socioeconomic status (SES) and low literacy [36]. Typically found to be largely inert, systematic reviews have reported no clinically significant effect of group education-based programs, such as “back schools [37–39].” Notable exceptions include studies in which the educational intervention is based on the biopsychosocial model rather than a biomedical model [35, 40–42], which was the case in this RCT. Speaking to the active nature of our Pain Education condition, we found that participants in the Pain Education condition significantly improved on primary outcomes to the same degree as those in the CBT condition [35]. The CBT intervention was cognitively focused and designed to specifically target maladaptive cognitions, whereas the Pain Education intervention was designed to provide factual, biopsychosocial knowledge about pain, without explicit focus on cognitive change. As such, while Pain Education was expected to be an active treatment control, it was not expected to engender, or function via, change in maladaptive cognition. Thus, cognitive changes were expected to operate as a therapeutic mechanism in CBT, but not in Pain Education.

In the original study [35], 61 people completed the 10-week interventions. Pre- and posttreatment measures of pain-related outcomes and the pain catastrophizing scale (PCS) [43] were taken. Insofar as pain catastrophizing is a maladaptive pain-related cognition, reductions in pain catastrophizing 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. 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 posttreatment changes in the PCS would be greater in CBT than in Pain Education and (b) pre- to posttreatment PCS changes to correlate with pre- to posttreatment pain-related outcome changes to a greater degree in CBT than in Pain Education.

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 of the University of Alabama, and an informed consent was obtained from all patients prior to participation. For additional details, see the reported original trial [35].

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 second grade level or higher as determined by the Wide Range Achievement Test-4 reading/word decoding subtest [44]; and (5) have a home telephone or comparable form of communication, i.e., cell phone. Study exclusion criteria included the following: (1) HIV-related pain and cancer pain because these are associated with malignant disease (i.e., progressive worsening of symptoms possibly resulting in death) which entails a different symptom trajectory than chronic pain [45]; (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 [46]; (3) other current psychosocial treatments for any pain condition; and (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 ten 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 [35]. 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 ten sessions) may also be found in the original report [35].

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. [47, 48]. 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. [49] for an overview of the adaptation process). Groupings 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 h. Participants in both conditions were given a client workbook with materials and handouts they could follow/discuss during sessions and read between sessions. The client workbooks consisted of handouts that provided a summarization of the main points covered throughout each weekly session. Thus, the client workbooks mirrored the session content (described below) that comprised the CBT and Pain Education conditions. In the CBT condition, the participants were also given worksheets that assisted with the skill-building exercises (see the Kuhajda et al. for more details) [50]. Each session's handouts were distributed to the participants at the beginning of the session.

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 (see Thorn for details on pre- and post-session checks [47]; these brief open-ended questionnaires were used to determine what aspects of each session were meaningful and retained by the participant). 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 skill-building exercises and group discussion followed. Skill-building exercises

included in-group, therapist-guided completion of the various coping skills taught during each session. For example, in session 6, the group leader collaboratively demonstrated a cognitive-restructuring exercise using a participant example; in session 7, the group leader implemented a guided relaxation; and in session 8, the group leader guided an expressive writing period. For further details regarding session structure, see Thorn [47].

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 skill-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, and 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; and session 10—stages of change, review concepts learned, provide feedback about helpful and challenging aspects of the treatment. See Ehde et al. for further details pertaining to the structure of the Pain Education protocol [48].

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 PCS was used to assess patient report of catastrophic thinking about pain [43]. The 13-item measure asks respondents to rate, using a five-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 ($\alpha=0.93$), concurrent and discriminant validity, and high test-retest reliability over a 6-week period ($r=0.78$)

[43, 51, 52]. Adequate internal consistency was found in the current sample ($\alpha=0.94$).

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 [53]. 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 ($\alpha=0.85$) in a variety of pain populations and concurrent validity with other pain instruments [53, 54]. In the current sample, the BPI-intensity and BPI-interference scales both had adequate internal consistency ($\alpha=0.86$ and $\alpha=0.91$, respectively).

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 [55]. 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 ($r=0.949$ and $r=0.929$, respectively) and has been shown to have adequate reliability that is comparable to the 24-item version ($\alpha=0.88$) and strong concurrent validity [55]. Adequate internal consistency for the 11-item version was demonstrated in the current sample ($\alpha=0.84$).

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 [56]. 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 four-point scale ranging from 0 (*rarely or less than 1 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 [57]. Reliability and validity are reported to be adequate and similar across a variety of samples from the general population [58]. Internal consistency was found to be adequate in the current sample ($\alpha=0.89$).

Life satisfaction—The Quality of Life Scale (QoLS) is a seven-point self-report scale that manifestly assesses life satisfaction in several areas [59]. 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 [59]. In the present sample, adequate internal consistency of the QOLS was demonstrated ($\alpha=0.86$).

Statistical analyses

To determine whether pre- to posttreatment changes in PCS scores (and other outcome factors) differed in magnitude between CBT and Pain Education groups, a series of ANCOVAs were conducted on posttreatment values controlling for pretreatment values. To determine whether the relationships between changes in pain catastrophizing and outcomes differed as a function of treatment group, simple change scores were first computed for PCS and outcome variables by subtracting pretreatment values from posttreatment values. Next, interaction terms were computed by multiplying PCS change scores by a dummy-coded treatment group variable (1=CBT; 2=Pain Education). 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) and entering the PCS $\times$ treatment group interaction term in the second step. A significant interaction was revealed by a significant increment in R^2 for the second step. The interactions were dissected by generating regression equations linking PCS and outcome change scores separately for each treatment group.

RESULTS

Treatment group comparisons on PCS and outcome values
As reported by Thorn et al. [35], PCS and all outcome variables, with the exception of RMDS (disability) values, changed significantly from pre- to posttreatment with effect sizes ranging from $\eta^2=0.09$ to $\eta^2=0.269$. In the current study, we focused exclusively on the magnitude of treatment group differences. For PCS scores, the posttreatment difference between CBT and Pain Education controlling for pretreatment values was nonsignificant [$F(1, 58)=1.84$; $p>0.10$; $\eta^2=0.031$], 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). Thus, when pretreatment differences between groups were controlled, CBT did not produce significantly greater decreases in the putative mechanism, pain catastrophizing, than Pain Education.

For outcome factors, all CBT vs. Pain Education comparisons were nonsignificant [$F_s<1.92$; $p_s>0.10$; $\eta^2_s<0.032$]. Thus, CBT and Pain Education produced virtually equivalent effects on outcome factors.

PCS change $\times$ treatment group effects on outcome changes
For CES-D change scores, the PCS change $\times$ treatment group interaction was significant ($t=2.57$; $p<0.01$). 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 ($\beta=0.60$; $p<0.01$), whereas for Pain Education, this association was nonsignificant ($\beta=-0.06$; $p>0.10$). Thus, reductions in pain catastrophizing were significantly related to reductions in depressive symptoms in the CBT condition only.

PCS change $\times$ treatment group interactions were nonsignificant for all other outcome variable change scores ($t_s<1.0$; $p_s>0.10$). However, the main effects for PCS changes on QoLS ($\beta=-0.36$; $p<0.01$), RMDS ($\beta=0.44$; $p<0.01$), BPI intensity ($\beta=0.37$; $p<0.01$), and BPI interference changes scores ($\beta=0.41$; $p<0.01$) 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.

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 pain-related outcomes in CBT-based interventions [28–31]. More importantly, questions of whether theoretically specified mechanisms (such as catastrophizing in CBT) 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 mechanism, but the degree to which a theoretically specified mechanism may be active primarily in the treatment that deliberately targets it.

If CBT works via reducing maladaptive cognitions, then changes in pain-related cognition should primarily occur and predict pain-related outcomes in CBT, and to a significantly lesser degree, occur and predict outcomes in a distinctly different treatment. Results in general did not support the proposition that reduction in pain catastrophizing is a therapeutic mechanism *specific* to CBT, and so replicate the results of Smeets et al. [34]. Although the mean PCS change score for the Pain Education group was numerically smaller than the mean PCS change score for the CBT group, the two means were not significantly different. This null finding 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 pretreatment 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 posttreatment 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.

Further, the degree to which PCS changed from pre- to posttreatment was related significantly to four of five pain-related outcome change scores *irrespective of condition*. These findings combined with the results from Smeets et al. [34] suggest that reductions in the tendency to catastrophize about pain, brought about through distinct treatment protocols, may be a potent albeit broad therapeutic mechanism that is not specific or limited to CBT. Consider that pain catastrophizing reductions predicted pain-related outcomes in a Pain Education condition in the present study and predicted pain-related outcomes in a physical training condition in Smeets et al. [34]. 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 [60].

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 multidimensional 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 [61]. 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 that is common, but potentially quite critical to many efficacious interventions.

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, the results may not generalize to higher SES, nonminority 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. Third, as argued by Laurenceau et al. [62] and Thorn and Burns [63], the study of therapeutic mechanism requires methodological features not part of our original study. 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. A further limitation of the methods described by Smeets et al. [34] and used here was that a *cognitive* mechanism that was theoretically linked to the treatment conditions featuring CBT was the only mechanism assessed. Measuring mechanisms theoretically linked to *other* active treatments—such as measures of aerobic capacity for Smeets et al. [34] and knowledge acquisition for the present study—would allow a more comprehensive test of mechanism specificity. Additionally, we measured only a putative-*specific* mechanism, 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. Finally, in order to comprehensively understand how treatments produce long-term, sustained patient benefit, future studies should examine outcomes at follow-up assessments and the mechanisms associated with these outcomes. For example, it is possible that changes in catastrophizing wrought via CBT might yield more durable change than those obtained with EDU, as well as a greater long-term association with outcomes. Limitations notwithstanding, the purpose of this study was to help *illustrate* the importance of mechanism issues. The results point toward fundamental gaps in our knowledge base regarding psychosocial interventions and how they work.

The results of Smeets et al. [34] in conjunction with those of the present study suggest that change in maladaptive cognition—a mechanism assumed to be specific to CBT-based treatments—may operate as

a common mechanism across interventions that are as procedurally diverse as CBT, physical exercise, and pain education. Far from dooming CBT, these results offer the intriguing possibility that changing irrational pain-related cognitions by whatever means may be a core feature of many efficacious approaches. At the same time, these results also underscore how little we truly know about how our psychosocial interventions for chronic pain produce the salutary outcomes reported in study after study. 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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