

Coping self-efficacy as a mediator between catastrophizing and physical functioning: treatment target selection in an osteoarthritis sample

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Abstract The purpose of this study was to assess the relative effects of coping self-efficacy and catastrophizing on physical functioning. Over a 9-month period, studying changes in self-efficacy as possible mediator between catastrophizing changes and physical functioning changes might provide evidence for the most promising treatment target. Data came from a randomized, longitudinal controlled trial comparing exercise, self-management and the two combined to treat 254 individuals with early knee osteoarthritis. A secondary analysis using a bootstrapped linear mixed-effects mediational model produced estimates of both the direct and indirect effects. Results indicated that self-efficacy partially mediated the effect between catastrophizing and physical functioning suggesting that self-efficacy was the more direct treatment target compared to catastrophizing. Treatments targeting both self-efficacy and catastrophizing may have greater impact on physical functioning compared to treatments that focus on only one.

Keywords Coping · Self-efficacy · Catastrophizing · Osteoarthritis · Treatment

All interventions aim to either reduce a negative target or enhance a positive target. Psychology embraces this two factor approach with a surging interest in preventative (e.g., acceptance and commitment therapy Biglan et al. 2008) and positive psychology (Seligman and Csikszentmihalyi 2000). Psychologists now explore various positive treatment targets for problems across many areas. One area—arthritis—bridges medicine and psychology due to the nature of the degenerative physical disease and its psychological impact on patients. Researchers showed, for example, that strengthening positive affect (Zautra et al. 2005), self-efficacy (Keefe et al. 1996; Lorig et al. 2001), and pro-health behaviors (e.g., exercise Ettinger et al. 1997) resulted in lower pain and greater functioning. Researchers also showed that decreasing mood disturbance (Smith and Zautra 2008), negative life events (Potter and Zautra 1997), and maladaptive pain coping strategies (Keefe et al. 1997) resulted in improved functioning. For the purposes of our study, we focused on self-efficacy as a positive target and catastrophizing as a negative target to assess this dualistic treatment approach and to determine whether one treatment target might offer greater potential than the other.

Self-efficacy—the belief that one can successfully perform a specific behavior to achieve a particular outcome (Bandura 1977)—predicts functional status in arthritis (Marks et al. 2005) and other chronic pain conditions (Marks et al. 2005; Sharma et al. 2003; Maly et al. 2006c; Focht et al. 2005). Some of the evidence linking self-efficacy to chronic pain comes from the osteoarthritis outcomes research, where arthritis self-efficacy is defined

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as the belief that one can manage arthritic pain and other stressors generated by arthritis. Osteoarthritis patients with high self-efficacy report and display greater physical functioning and report less pain than those with low arthritic self-efficacy (Pells et al. 2008; Focht et al. 2005).

Targeting a positive strength such as self-efficacy seems warranted given the empirical support in the arthritis treatment literature. Longitudinal trials (e.g., Rejeski et al. 2001; Sharma et al. 2003) showed that long-term functional benefits came with improved self-efficacy. Specifically, higher self-efficacy resulted in less self-reported deterioration over time. Cross-sectional studies merely confirm these findings (Maly et al. 2006a, b) and provided greater conceptual clarity for the role of self-efficacy in the self-appraisal process. Self-efficacy tends to correspond far more favorably with self-reported outcomes than with objectively measured outcomes (Harrison 2004)—a finding that reinforces the connection between self-efficacy and self-appraisal. Furthermore, studies examining self-efficacy in osteoarthritis patients often ignore other psychological processes. By studying self-efficacy in isolation, it is difficult to gain insights into how self-efficacy operates; thus a study that focuses on both self-efficacy and an alternative, contrasting treatment target might help clarify each variable's role in the change process.

Catastrophizing serves as a reasonable alternative treatment target to self-efficacy. Degenerative conditions such as osteoarthritis cause irreversible damage that accumulates—placing substantial burden on patients who must cope with pain, stress, and disability. People cope with pain in many ways. Researchers found that both positive and negative outcomes arise from different coping strategies (Rosenstiel and Keefe 1983). Catastrophizing is one maladaptive coping strategy that has been linked to negative outcomes in chronic pain patients (Sullivan et al. 2001b; Burns et al. 2003a, b), including diminished physical functioning (Keefe et al. 2000; Turner et al. 2002; Vowles et al. 2008). Catastrophizing—the tendency to focus on pain and magnifying, even dramatizing, the possible negative consequences (Keefe et al. 2004)—reflects the interpretations and reactions to chronic pain rather than the severity of the pain itself. Reducing negative targets such as catastrophizing has less empirical support for primary treatment of arthritis compared to targeting positive strengths. In some cases, however, changes in maladaptive coping influence other mediating variables such as positive and negative affect (Zautra et al. 1995). Other research indicates a direct correspondence between these maladaptive coping styles (e.g., catastrophizing) and reported pain or disability (Watkins et al. 1999).

It remains to be seen whether targeting positive processes leaves the negative processes unaffected since they tend to be related in most studies. That is, as treatment

increases catastrophizing, self-efficacy wanes—either as a consequence or simply concurrently from other unmeasured third processes. The goal of this study was to examine whether self-efficacy mediates the effect between catastrophizing and physical functioning in people who have just been diagnosed with osteoarthritis. Diminished physical functioning in osteoarthritis patients is a key concern to researchers and clinicians, since low physical functioning places patients at an increased risk for cardiovascular disease and other serious health consequences by inhibiting physical activity (Berlin and Colditz 1990; Oguma and Shinoda-Tagawa 2004; Lakka et al. 1994; de Groot et al. 2008). Patients' coping style plays a significant role in their ability to remain functionally independent (Keefe et al. 1987a, b).

The effects of self-efficacy and catastrophizing relative to physical functioning is also of practical importance for selecting optimal targets for psychosocial interventions. Interventions ought to be based on strong theory that is empirically supported (DeVellis and Blalock 1993). In accord with that line of reasoning, we tested the hypothesized mediation model with catastrophizing, self-efficacy, and functional status. Since the two predictors—catastrophizing and self-efficacy—consistently showed an inverse relationship (Keefe et al. 1997), we expected that catastrophizing would affect physical functioning but the influence would be mediated by self-efficacy. Catastrophizing, in other words, would reduce self-efficacy and, in turn reduce physical functioning. We expected that mediational ordering on the basis of two studies (Pells et al. 2008; Shelby et al. 2008) that tested the mediational model in the order we specify here. The mediation model consists of three primary hypotheses including (1) a negative relationship between catastrophizing and self-efficacy; (2) a negative relationship between catastrophizing and physical functioning; and (3) a positive relationship between self-efficacy and physical functioning. A fourth hypothesis—the one central to this study—suggests that the relationship between catastrophizing and physical functioning (hypothesis 2) would be mediated by self-efficacy. Tests of the fourth hypothesis would be strengthened by showing that the mediation effect exists when self-efficacy and not catastrophizing serves as the mediator (5th hypothesis).

Assessing the independent effects of catastrophizing and self-efficacy on outcomes presents some complexities to researchers. First, both constructs—or at least their measures—tend to correlate with measures of mood (e.g., depression, anxiety, etc.) (Sullivan et al. 2001a; Burns et al. 2003). Estimating their independent effects might be difficult without taking mood and mood-correlated constructs (e.g., pain, sex, age) into consideration. Second, these constructs may change at different rates, under different influences, and at different times. For example, transient pain

symptoms may affect catastrophizing less (Turner et al. 2004) than self-efficacy (Smarr et al. 1997). Conversely, self-efficacy might be the easier of the two outcomes to change via simple treatment (Lorig et al. 2004). Such contrasting influences might obscure the relationship between two cognitive constructs, especially in prospective data. Furthermore, the relationship among the three variables might not conform to a set order whereby one variable (e.g., self-efficacy) mediates the relationship between the two others but rather a moderation model where the relationship between catastrophizing and physical functioning depends upon a person's self-efficacy level. Thus, an alternative to the mediation model might be a moderation model that complicates further the causal priority. Our aim was to account for these factors and provide the most conservative estimate for our aforementioned hypotheses. Estimating the potential different effects of self-efficacy and catastrophizing may help clinicians and researchers both select and examine treatment strategies in the future.

Patients and methods

Design

Treatment duration data (baseline, 3-month and 9-month) from a non-blinded, 24-month randomized treatment trial of resistance training and self-management for osteoarthritis (the KNEE study) served as the basis for our study. The trial aimed to compare the effects of three interventions: a resistance training program, a self-management program, and a combined resistance training and self-management program (see McKnight et al. 2010) for primary results). The study was carried out at the University of Arizona Arthritis Center in Tucson, AZ and approved by the institutional review board. All study participants gave written informed consent prior to randomization.

Participants

KNEE Study participant eligibility criteria were (1) between the age of 35 and 64 years; (2) reported pain on most days in 1 or both knees; (3) duration of symptoms of less than 5 years; (4) had Kellgren and Lawrence classification (KL) (Kellgren and Lawrence 1957) grade II radiographic evidence of knee osteoarthritis in one or both knees; and (5) self-reported disability due to knee pain for at least 3 of the following: descending or ascending stairs, walking, kneeling, or performing daily activities. Potential participants were excluded if they had (1) an uncontrolled medical condition that precluded safe participation or prevented completion of the study (e.g., heart disease, blood pressure or respiratory conditions); (2) any neurological condition that

could affect coordination; (3) inflammatory arthritis (e.g., rheumatoid or psoriatic arthritis); (4) previous knee surgery; (5) KL grades III or IV radiographic evidence of osteoarthritis in one or both knees; (6) a BMI > 37.5 Kg/m²—individuals over that limit were advised to follow a weight loss program and achieve stable weight for 6 months prior to participation; (7) a knee corticosteroid injection in the previous 3 months; (8) plans to move from the local area; or (9) plans to become pregnant during the study period.

Staff recruited participants from the local community, assessed eligibility by telephone, and if appropriate scheduled them for a radiographic exam administered by a staff rheumatologist. Individuals meeting all eligibility criteria were followed for a run-in period ($\bar{X}_{days} = 73$) and then randomly assigned to one of three treatment groups—a resistance training group, a self-management group, or a combined treatment group. Males and females were allocated separately in random blocks to ensure a equivalent distribution of sex among the groups.

Interventions

All three groups participated in treatment that specifically targeted self-efficacy either through resistance training, education, or both. Previous analyses (McKnight et al. 2010) showed that group assignment had no effect on the outcomes but all three groups improved both physically and psychologically. To avoid redundancy with previous publications and focus more on the relevant aspects of the data, we provide a cursory account of the randomized treatment trial here. The resistance training group received 9 months of supervised sessions aimed to enhance participants flexibility, balance, range of motion, and muscle strength. The self-management group received 9-month education intervention—based on previous self-management programs (Lorig and Holman 2003)—that targeted coping and self-efficacy skills. A third group received a combined intervention consisting of both resistance training and self-management. The interventions focused on self-efficacy via either behavioral methods (i.e., resistance training) or cognitive methods (i.e., psychoeducation through the self-management program), and all three groups showed roughly equivalent, significant treatment outcomes. Since there were no significant differences between treatment conditions, we present all results below on the entire sample.

Primary variables

Our study focused on three primary measures—each measured at three different intervals—prior to treatment (0 months), after the initial intensive treatment (3 months) and after the treatment booster sessions (9 months). These

times represent critical periods in the treatment process where we expected a linear change over time for all relevant variables.

Catastrophizing

We used the catastrophizing subscale from the Coping Strategies Questionnaire (CSQ; Rosenstiel and Keefe 1983). The CSQ is a 42-item instrument with 7-point Likert scales (0 = never to 6 = always) that assesses the use of six different cognitive coping strategies (diverting attention, reinterpreting pain sensations, ignoring pain sensations, coping self-statements, praying or hoping, and catastrophizing) and one behavioral strategy (increasing activity level). Researchers have used the CSQ extensively in the evaluation of rheumatology diseases and previous research supports its reliability (Cronbach's α , 0.77–0.89). For the purposes of the current study, we used the sum score of the 6-item catastrophizing subscale.

Self-efficacy

We used the Arthritis Self-Efficacy Scale (ASES; Lorig and Holman 1998)—a 20-item self-efficacy measure developed specifically for individuals with arthritis. The ASES yields 3 scores: self-efficacy for physical function (FUNCTION), for pain management (PAIN), and for controlling other arthritis symptoms (OTHER Sx). Previous research indicates strong internal consistency ($0.90 < \alpha < 0.94$) and test-retest reliability ($0.85 < r_{tt} < 0.90$) for each scale. Our intent was to measure general self-efficacy (SE) so we combined the three subscales via a unit weighted factor scoring procedure (i.e., standardizing the subscales and taking the mean of the z-scores). The FUNCTION subscale typically correlates strongly with measures of physical functioning (see Covariates section below); consequently, we chose to combine the three subscales into a single factor score. Contrary to our expectations, combining the subscales actually attenuated the relationship between our our measure of self-efficacy and self-reported physical functioning (compare $r_{PAIN,PhysFunc} = 0.4$, $r_{FUNCTION,PhysFunc} = 0.4$, and $r_{OtherSx,PhysFunc} = .38$ with $r_{SE,PhysFunc} = .22$).

Physical functioning

We used the physical functioning subscale (10-items) from the Short-Form-36 Health Survey (SF-36; Ware and Sherbourne 1992)—a 36-item measure of overall health status. The SF-36 includes scales to measure the following dimensions: bodily pain, physical functioning, role limitations due to physical health, social functioning, general mental health, role limitations due to emotional problems,

vitality and general health. Each category score was re-coded into a 0–100 score, oriented so that high values represent more favorable states. All questions referred to problems in the past four weeks. Several studies reported the appropriateness and validity of this generic instrument for the evaluation of overall health status in knee osteoarthritis. The reported psychometric properties are for validity (correlation coefficients ranging from 0.39 to 0.85), internal consistency (Cronbach's alpha 0.73–0.96 for the different subscales) and test-retest reliability (correlation coefficients ranging from 0.60 to 0.90).

Covariates

Several variables potentially confound and inflate the relationship between our three primary variables. Depression level, age, sex, and arthritis severity tend to be related to all three measures and thus inflate the correlation and may spuriously indicate mediation when, in fact, no mediation exists among the variables. Depression was measured using the Center for Epidemiological Studies Depression Scale (CES-D; Radloff 1977)—a 20-item scale used in many observational and randomized controlled treatment studies over the last 25 years and total scores reflect complex but useful indication of mood problems in arthritis samples (e.g., Sheehan et al. 1995). Depression confounds the estimate of catastrophizing (Sullivan and D'Eon 1990; Sullivan et al. 2001a, b); failing to account for depression would affect measures of catastrophizing due to substantial mood level differences within the sample. Furthermore, since both catastrophizing and self-efficacy change according to depression level, we chose to eliminate any potential confound that would inflate the correlation between the two variables. Age and sex were measured by single items that asked respondents their date of birth and biological sex at baseline. Both of these variables confound measures of catastrophizing (Keefe et al. 2004; Santavirta et al. 2001) and coping self-efficacy (Miller and Cronan 1998) as well by inflating the correlation between the two. Previous research (Santavirta et al. 2001) also suggested that age (Santavirta et al. 2001) and sex (Keefe et al. 2000) both moderated the relationship between coping strategies, coping self-efficacy and self-reported well-being. Our aim was to eliminate any potential for our unbalanced sample (>75 % female) to spuriously influence the mediation model results. Finally, arthritis severity was measured using a standard visual analog scale (0–100 range) that asked the respondent to rate the current severity of their arthritis symptoms. Since arthritis symptoms often strongly relate to both catastrophizing (Keefe et al. 2000a, b) and self-efficacy (Prior and Bond 2004), we chose to account for this influence by partialing out the effects prior to hypothesis testing. Our

rationale for controlling arthritis severity was due in part to the longitudinal nature of our data whereby participants had differing levels of severity at different times throughout the study. Even though previous research indicated that catastrophizing was not strongly related to severity (Keefe et al. 2000a, b) other research (Maly et al. 2007) suggested that self-reported arthritis severity (i.e., pain and stiffness) negatively correlated with coping self-efficacy. The differential impact of severity on our two primary predictors may have influenced the results of our model and thus we chose to statistically control for severity. What is left after partialing out arthritis severity is the residual effects of coping and catastrophizing that are not affected by the temporal fluctuations of the disease process. We also assessed other mood-relevant covariates (e.g., Negative Affect from the PANAS (Watson et al. 1988)) that may have confounded our hypotheses and found them to be non-significantly related to our primary variables; we omitted these variables to simplify the analysis and explanation.

Data analyses

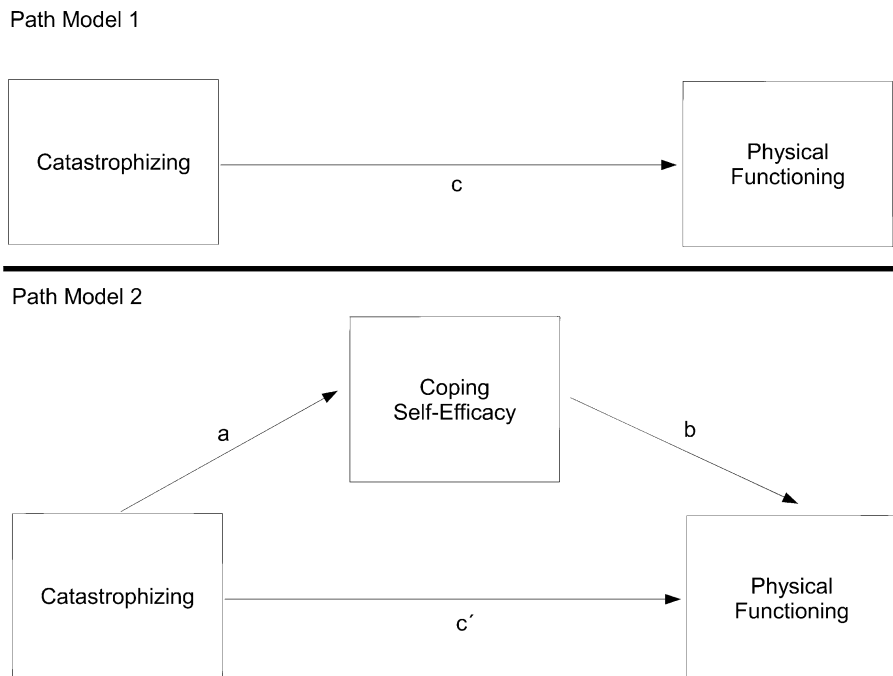
Missing Data Handling Due to the nature of longitudinal data, we expected missing data and planned to treat the missing values by using multiple imputation (Little and Rubin 1987; Rubin 1996; McKnight et al. 2007) The purpose of multiple imputation was both to create complete data and to help estimate the influence missing data have on the parameters of interest. Multiple imputation with chained equations (MICE) offered a flexible solution to condition the imputed values on observed values via

predictive mean matching. All analyses used multiply imputed, complete data from the MICE procedure.

Mediation modeling

The standard approach (Baron and Kenny 1986) for testing mediation required three linear models (see Eqs. 1 through 3) to estimate the parameters depicted in Fig. 1. Linear model 1 (Eq. 1) estimated the relationship between catastrophizing and physical functioning (Fig. 1; Path Model 1, parameter c). Linear model 2 estimated the relationship between self-efficacy and physical functioning (Fig. 1; Path Model 2, parameter b) as well as the residualized effect between catastrophizing and physical functioning (Fig. 1; Path Model 2, parameter c'). Finally, linear model 3 estimated the relationship between catastrophizing and self-efficacy (Fig. 1; Path Model 2, parameter a). All paths must be significantly different from zero for mediation to be possible. Since the data came from a longitudinal, randomized, between-group trial, we needed a more flexible model than mere regression to estimate these parameters (MacKinnon 2008); a linear mixed-effects model provided parameter estimates. Time (b_{1i}) and intercept (b_{0i}) served as a random effects while treatment group (b_2) served as a fixed effect prior to each of the primary, fixed-effect predictors mentioned previously. Thus, the models accounted for the longitudinal data, between group effects and then the mediation effects. The indirect or mediation effect (ab) was estimated first by using the traditional Sobel test whereby the indirect effect is defined as $ab = c - c'$ and computed by multiplying path a with path b ($ab = a \times b$).

Fig. 1 Standard mediation models



The Sobel test tends to be sensitive to sample size so to counter that sensitivity we estimated the indirect effect (ab) via 5000 bootstrap iterations (MacKinnon et al. 2002). We also tested the alternative model specifying catastrophizing as the mediator between self-efficacy and physical functioning to rule out potential model misspecification of the causal order.

$$\text{Physical Functioning} = b_{0i} + b_{1i} * X_{time} + b_2 * X_{Tx} + c * X_{Catastrophizing} \quad (1)$$

$$\text{Physical Functioning} = b_{0i} + b_{1i} * X_{time} + b_2 * X_{Tx} + b * X_{Self-Efficacy} + c' * X_{Catastrophizing} \quad (2)$$

$$\text{Self-Efficacy} = b_{0i} + b_{1i} * X_{time} + b_2 * X_{Tx} + a * X_{Catastrophizing} \quad (3)$$

The R statistical software package (R Development Core Team 2008, version 2.80) produced all estimates. In particular, we used the lme4 (Bates et al. 2008) package for linear mixed-effects models and the mice (Oudshoorn 2007) package for multiple imputation. Bootstrap results were produced by R code available from the primary author.

Moderation modeling

The contrasting model from the mediation model specified the two main effects of catastrophizing and self-efficacy as predictors of physical functioning followed by the interaction of the two main effects. A significant interaction might indicate that the relationship between the predictors does not follow a mediation model but rather a moderation or perhaps a hybrid moderating-mediation model. All parameter estimates came from the same linear mixed-effects model as specified for the mediation model above.

Results

Missing data

Staff recruited 1726 potential participants beginning September, 2003, approximately 21 weeks prior to baseline testing and continued recruitment through December, 2006. A total of 689 (40%) potential participants met initial screening eligibility criteria, agreed to participate (180 refused; 10%), and received knee X-ray exams. Of those, 398 (58%) were judged to have KL grade II radiographically evident tibial-femoral osteoarthritis in either or both knees. Finally, 293 (74%) participants with radiographic evidence of osteoarthritis met inclusionary criteria and

Table 1 Baseline demographic and mediation model variables by group

	Statistic
Study design	
Randomized	$n = 254$
Completed (9 months)	$n = 221$
% Completed	87.0
Demographics	
White (%)	91.6
College educated (%)	61.2
BMI (kg/m ²) (SD)	27.7 (4.2)
Covariates	
Age (SD)	52.5 (7.2)
Female (%)	77.3
Arthritis severity VAS (SD)	23.8 (22.3)
CESD; depression (SD)	8.5 (7.5)
Primary variables	
Catastrophizing (SD)	-.11 (3.44)
Self-efficacy (SD)	-.08 (0.70)
Physical functioning (SD)	71.1 (18.7)

were randomized to one of the three treatment groups. Following randomization, 39 (13%) participants failed to receive the assigned treatment due to lack of interest, non-compliance, health problems, or moving from local area resulting in 254 participants who received treatment. The characteristics of these 254 participants are shown in Table 1.

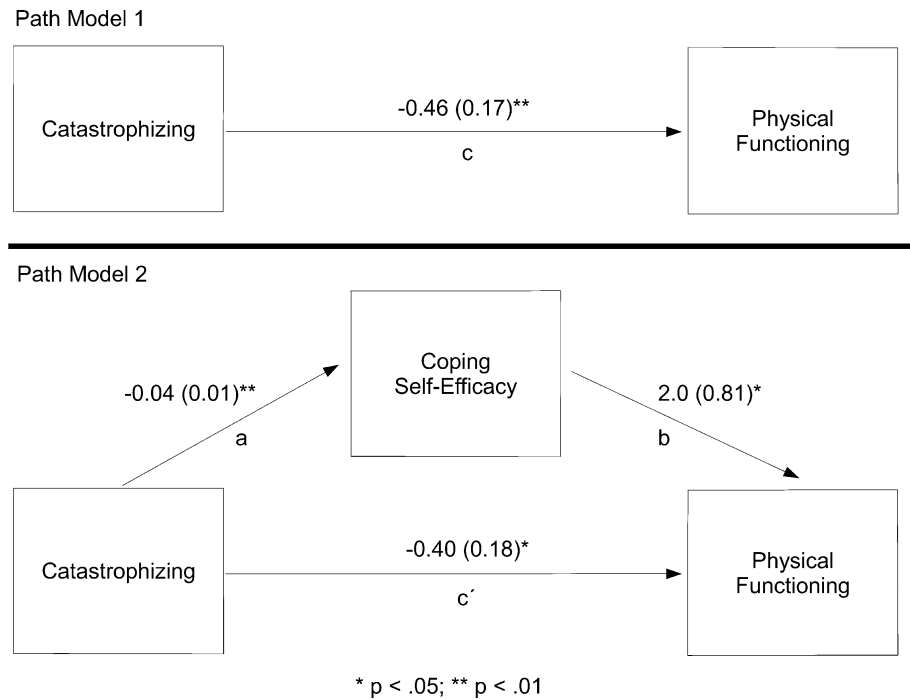
More than 85% of the assigned and treated participants finished the trial after 9-months (221 out of 254 for 87.% 9-month completion rate). Retention among the groups was not substantially nor significantly different ($\chi^2(2) = .26$, $p = .88$). Age, sex, race, arthritis severity, pain, disability, and comorbid medical conditions failed to predict dropout. Furthermore, treatment compliance varied somewhat by treatment (overall compliance was 67.5%) but not significantly. Compliance could be a relevant variable because the data come from a study where the aim was to influence participants coping with osteoarthritis. Since all groups had equivalent treatment compliance, the relevance of that variable diminished.

The multiple imputation procedure produced five complete datasets for each analysis. Amount of missing information was negligible ($\bar{\gamma} < .001$) and parameters were not significantly different ($p > .10$) from each full dataset so we reported a randomly selected result rather than average the parameter estimates.

Mediation models

Figure 2 shows the results of the three models. Each path shows the mean bootstrapped coefficient along with the

Fig. 2 Mediation results



standard error derived through the bootstrap procedure. Specifically, all the paths (*a*, *b*, *c*, and *c'*) were significantly different from zero indicating that the data met all three mediation steps and confirmed our first four hypotheses. While significant, none of the paths indicated very strong relationships

($\beta_a = -0.18, \beta_b = 0.11, \beta_c = -0.12, \beta_{c'} = -0.10$). Also, it is important to note that the direct effect (*c'*) was also significantly different from zero indicating that the results suggest only partial mediation.

The indirect effect (*ab*) estimated through the bootstrap procedure was also significantly different from zero—indicating mediation was likely between the three variables of interest. According to the bootstrap results, the effect was estimated at -0.06 with a bootstrapped standard error estimate of 0.03 ($p < .05$). Figure 3 shows the results from both the bootstrap procedure and the associated confidence intervals (95%: $-0.12, -0.01$) as well as the traditional, non-iterative Sobel test ($b = -0.7, se = 0.03, t = -2.26; p < .05$). The confidence intervals in Fig. 3 do not overlap with zero and thus the effect is said to be significant at the .05 level and, therefore, the mediation effect appears to be defensible on empirical grounds.

Moderation model

The alternative model specifying catastrophizing as a mediator between self-efficacy and physical functioning (hypothesis 5) failed to converge for two of the three linear mixed-effects models so we were unable to estimate either the direct or indirect effects. Additionally, a test of simple

moderation showed that after the main effects of self-efficacy and catastrophizing, the interaction term was non-significant ($p > .25$).

Discussion

The goal of this study was to test the mediated relationship between physical functioning and catastrophizing. Our intent was to assess whether a negative intervention target (catastrophizing) might be related to a positive intervention target (self-efficacy) when predicting a relevant outcome such as physical functioning. If the relationship were fully mediated (i.e., no direct effect between catastrophizing and self-efficacy) then the indirect effect would be far stronger than the direct effect from catastrophizing to physical functioning. While this sounds obvious from a modeling standpoint, it may not be so obvious from a treatment standpoint. Treatments that target mediators in full mediation models will be stronger than those that target the indirect predictor. Our results supported the mediation model, however, full mediation was not supported. Thus, a partially mediated effect exists between catastrophizing and physical functioning; the effect is both direct and indirect. A moderation model, however, failed to get support from the current data so the lack of mediation does not seem to indicate any moderation effects. If we consider both causal pathways and their implications for treatment, it seems logical to conclude that the greatest changes in self-reported physical functioning would occur by targeting both catastrophizing and self-efficacy. Treatments that

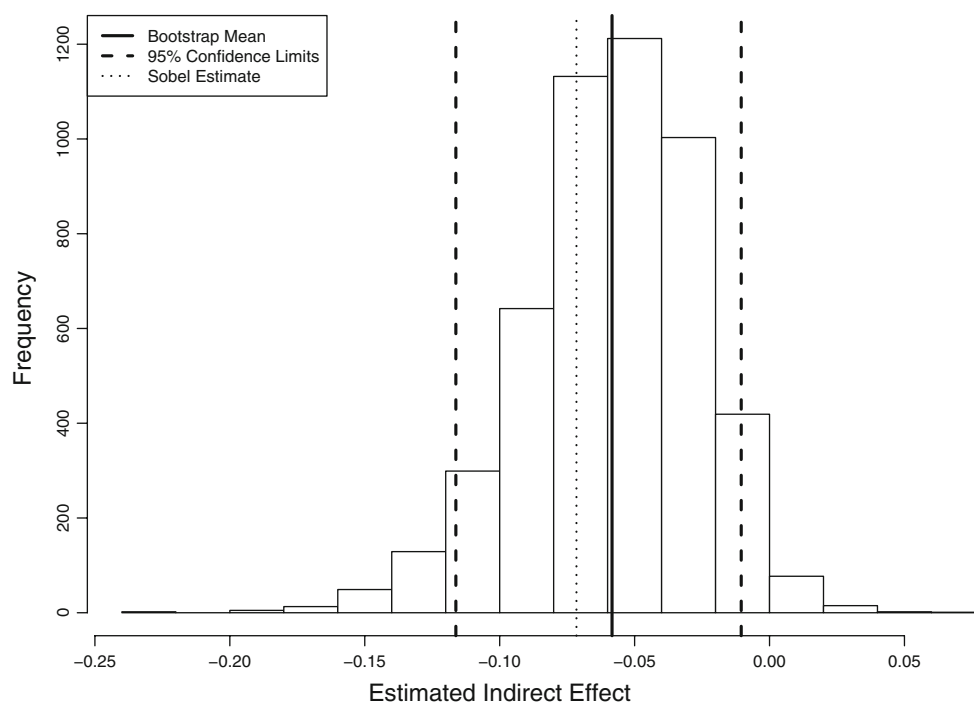


Fig. 3 Indirect effect (ab) bootstrap results

target only one source would lose out on the indirect effect. Furthermore, the direct effects from each offer only a modest relationship with the outcome. The treatment implications, therefore, are that a combined treatment ought to be better than singularly focused, targeted interventions.

Psychosocial treatments favoring cognitive reappraisal (e.g., pain acceptance Kratz et al. 2007) tend to view catastrophizing as a key target. Those treatments offer promising effects with self-reported pain and other relevant outcomes. Other treatments that specifically target self-efficacy (e.g., behavioral self-management programs Lorig and Holman 1993) offer equally promising outcomes (Smarr et al. 1997; Marks et al. 2005). Our findings support the generalizability of previous studies on chronic pain patients (Jensen et al. 2001) where a combination of both approaches produced the greatest changes in outcomes. These effects are now evident with samples that go beyond long-term chronic pain sufferers and include individuals who were otherwise healthy.

The current results also provide new evidence concerning catastrophizing. Some researchers have contended that catastrophizing is an artifact of other psychosocial variables like depression and pain severity (Sullivan and D'Eon 1990; Hirsh et al. 2007). Our results do not support fully this contention; with depression and arthritis severity partialled out, catastrophizing predicted both self-efficacy and physical functioning. We acknowledge the fact that mood in general might affect catastrophizing beyond just

depression; our results were limited by the available mood measures in our secondary data analysis. Failing to account for these other facets of mood may limit the extent we can argue that catastrophizing—as analyzed—exists independent of any mood fluctuations. Regardless, catastrophizing as a significant predictor might be surprising to some given the fact that our sample of young people in good physical condition tend to be low catastrophizers (Watkins et al. 1999).

Our results represent a conservative estimate of the mediation effect due to our covariates selection. We argued that depression, age, sex, and arthritis severity all affected the primary variables (i.e., catastrophizing, self-efficacy, and self-reported physical functioning). The indirect effect was substantially greater without the covariates ($b_{ab} = -0.46$, $SE = 0.07$) compared to the covariate indirect effect ($b_{ab} = -0.06$, $SE = 0.03$) and shows why the influence of the specified covariates might confound and inflate our estimates. Additionally, the paths between the variables were substantially stronger when omitting the covariates. Just as we hypothesized, the covariates inflated the relationship between all three variables. The trouble with covariates, however, lies in interpreting these residual effects. Once the effects are residualized to account for the covariates, we have an effect that must be interpreted with many conditions. For one, covarying out depression from the variables results in a model that is insensitive to fluctuations in mood. We argued that this is a logical covariate but for some participants, mood improvement might serve

to eliminate any benefit from treating either catastrophizing or self-efficacy. Second, and perhaps a more interesting effect comes from controlling for arthritis severity. Our intent was to control for the substantial variation in arthritis severity between participants over time; without statistically controlling for these effects, our results would be confounded by these between-participant fluctuations. Partialing out arthritis severity means that our effects resemble more of the intercept or mean levels of all three variables rather than dynamic, time-dependent variables and their inter-relationships. Thus, our model again serves as an underestimate of the potential effects with truly fluctuating severity (i.e., pain and stiffness) over time as indicated in our results without the covariates.

In summary, the current study focused on a younger sample that received a diagnosis of early osteoarthritis. The sample offers a unique and valuable perspective on the relationship between self-efficacy and catastrophizing. Our analyses provided support for a partial mediation relationship between catastrophizing and physical functioning. However, an alternative model might assume that both self-efficacy and catastrophizing are process components within an overarching sense of helplessness (Keefe et al. 2002) or optimism (Brenes et al. 2002). Patients who feel helpless might report lower levels of self-efficacy and higher levels of catastrophizing. The two treatment targets, however, may work independently since the relationship between the two left a tremendous amount of unaccounted variance. Catastrophizing and self-efficacy may be related but perhaps are different processes. Our initial hypotheses placed the two constructs in a causal relationship, the ordering of which might shift were they examined within an enlarged model. Future work might consider a broader measurement model whereby other constructs that reflect helplessness over time help determine the effects of treatment on a broader, better defined construct. For now, we suspect that when a person first receives a diagnosis of a chronic condition such as osteoarthritis, a likely response is to feel somewhat helpless and then catastrophize about the outcome. Providing a treatment that boosts the patient's self-efficacy and decreases catastrophizing may likely abate the sense of helplessness. Other positive and negative aspects of the psychological milieu may be worth investigating for their distinct influences on outcomes in clinical trials.

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