Cognitive Mediators of Change in Physical Functioning in Response to a Multifaceted Intervention for Managing Osteoarthritis

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Abstract

Purpose—Although non-pharmacological interventions have been shown to improve physical functioning in individuals with osteoarthritis (OA), the mechanisms by which this occurs are often unclear. This study assessed whether changes in arthritis self-efficacy, perceived pain control, and pain catastrophizing mediated changes in physical functioning following an osteoarthritis intervention involving weight management, physical activity, and cognitive-behavioral pain management.

Method—Three hundred Veteran patients of 30 primary care providers with knee and/or hip OA were cluster randomized to an OA intervention group or usual care. The OA intervention included a 12-month phone-based patient behavioral protocol (weight management, physical activity, and cognitive-behavioral pain management) plus patient-specific OA treatment recommendations delivered to primary care providers.

Results—Using linear mixed models adjusted for provider clustering, we observed that baseline to 6-month changes in arthritis self-efficacy and pain control partially mediated baseline to 12-month physical functioning improvements for the intervention group; catastrophizing did not.

Conclusion—Findings of a mediating role of arthritis self-efficacy and pain control in intervention-related functional changes are consistent with hypotheses and align with theoretical assertions of the role of cognitions in cognitive and behavioral interventions for chronic pain. However, contrary to hypotheses, catastrophizing was not found to be a mediator of these changes.

Introduction

Osteoarthritis (OA) is a widespread and debilitating condition, affecting 27 million Americans [1] with prevalence on the rise [2, 3]. Evidence-based guidelines for OA emphasize the importance of combining medical approaches with behavioral interventions, such as cognitive-behavioral therapy (CBT) for pain, weight management, and exercise, all of which have shown efficacy for improving symptom management [4–8]. With an eye toward maximizing benefit for patients, many interventions have combined multiple behavioral strategies. Although these programs have demonstrated improvements in pain and function [7, 9], few studies have explored the mechanisms for these changes. Gaining a better understanding of the processes by which these interventions exert effects can inform the design of more efficient and effective interventions. This study is a secondary mediation analysis exploring mechanisms of change in physical functioning in the context of a randomized controlled trial (RCT) of a multifaceted intervention for Veterans with OA.

Literature examining diverse behavioral interventions for pain suggests that changes in the ways individuals think about their arthritis are one key pathway for gains in functional outcomes [7, 10–12]. Of these behavioral treatments, CBT is unique in targeting cognitive change as the primary intervention strategy, as its underlying theory asserts that changing thoughts about pain can bring about decreased pain and improved function [13]. In CBT for pain, participants work to restructure maladaptive cognitions and patterns of thinking about their pain such as catastrophizing (i.e., magnification of the threat of chronic pain and helplessness in the face of pain) and increase adaptive pain cognitions such as self-efficacy (i.e., confidence in ability to manage chronic pain-related symptoms) and pain control (i.e., perceived ability to exert control over one's pain). Changes in these cognitions have been shown to relate to improvements in functioning through CBT (e.g., [10]). Although cognitions may not be the primary or only intended process of change in other behavioral interventions, research on exercise, physical therapy, and weight loss interventions for chronic pain have also suggested a role for cognitions as mechanisms of change in functional outcomes [7, 11, 12, 14–17]. For example, in a randomized controlled trial, Smeets and colleagues (2006) compared active physical treatment, CBT, active physical treatment + CBT, and wait list control groups of individuals with chronic low back pain. Change in pain catastrophizing mediated improvements in disability for all groups except the wait list group, suggesting that cognitions may act as mechanisms of change even in treatments that do not explicitly target them. Changes in cognitions have also been demonstrated in observational studies of patients undergoing multidisciplinary treatments for chronic pain [18–20], accounting for 26% of the variance in improved physical functioning for participants completing one 4-week multidisciplinary pain program [19].

Despite strong suggestions for a key role of cognitions as mechanisms of change in interventions for chronic pain, the majority of this literature has serious methodological

flaws, either utilizing synchronous (cross) correlations (e.g., [11]) or using lagged correlations without a treatment control group (e.g., [18]) [21]. Mediation analyses allow for the analysis of mechanisms of change over time. Principled methods of mediation (1) demonstrate temporal precedence between the mediator and the outcome and (2) include a control group that allows for causal interpretations about the role of therapy associated with the change in cognitions [22, 23].

The current study is the first to use temporal mediation to explore cognitive processes of change in a multifaceted OA intervention. Specifically, we explored whether changes in arthritis self-efficacy, catastrophizing, and perceived control over pain, measured at the midpoint of the intervention, predicted improvements in physical functioning following the intervention. In order to demonstrate that a factor is a mediator of treatment, the factor must (1) demonstrate change occurring during treatment; (2) be correlated with treatment group, thus potentially being the result of treatment; and (3) have an effect on the outcome [23]. Measuring changes in cognitions 6 months after the randomization and before physical functioning was measured following intervention completion allows for conclusions to be made about whether intervention-related changes in cognitions help to explain intervention-related changes in physical functioning measured 6 months later. Arthritis self-efficacy, catastrophizing, and perceived control over pain were selected because they have been the most commonly analyzed mechanisms of change in behavioral interventions for chronic pain [10–12, 15, 17, 24]. Based on this prior research, we expected that all three cognitions would partially explain treatment-related changes in functioning. Physical function was the outcome of interest because of its important role in OA and because there were significant, yet modest, changes in this outcome following the intervention [25]. The goal of this analysis was to determine whether changes in pain cognitions played a role in this effect, as hypothesized.

Methods

The institutional review board of the Department of Veterans Affairs HealthCare System in Durham, NC (DVAHCS), approved this study. All procedures performed were in accordance with the ethical standards of the institutional research committee and with the 1964 Declaration of Helsinki and its later amendments. Written informed consent was obtained from all participants. Detailed methods and primary study results have been published previously [25, 26].

Study Design

This was a secondary analysis of a cluster-randomized controlled trial, with primary care providers (PCPs) randomized to OA intervention (nPCPs =15; nVeterans = 151) or usual care control (nPCPs = 15; nVeterans = 149). We enrolled approximately 10 patient participants for each PCP. PCPs assigned to the OA intervention received the provider intervention, and their enrolled patient participants received the patient intervention. Patient participants in both arms continued with usual medical care recommended by their providers.

Participants and Recruitment

PCPs in the DVAHCS Ambulatory Care Service with patient panels large enough to likely enroll n =10 participants were invited to participate. Patients were eligible if they had hip OA and/or knee OA, were overweight (body mass index (BMI) 25), and engaged in low physical activity (see Allen et al. [26] for details and exclusion criteria). Potential participants were identified from DVAHCS electronic medical records, mailed an introductory letter, and called for a screening interview. Potential patient participants were blinded to their PCP's randomization until after baseline assessments.

Interventions

Patient Intervention—The intervention lasted 12 months and focused on physical activity, weight management, and cognitive-behavioral pain management strategies [26]. A counselor taught CBT skills alongside general exercise and dietary strategies via telephone. Cognitive restructuring was taught during months 9 and 10. Calls were scheduled twice per month for the first 6 months, then monthly for the last 6 months, and were delivered by one counselor with training in OA, health education, and behavior change, with oversight provided by study co-investigators. Goal setting, action planning, and motivational interviewing strategies were major components of the intervention. Participants were given written patient educational materials, an exercise video for patients with OA, and an audio CD of relaxation exercises.

Provider Intervention—This intervention involved delivery of patient-specific OA treatment recommendations to PCPs via the electronic medical record, based on published treatment guidelines (e.g., refer to physical therapist, recommended topical NSAID, or capsaicin [6, 8, 27]).

Measures

Baseline and 12-month follow-up measures were completed in-person with allowance for telephone-based assessments as needed. Six-month measures of cognitions were assessed via telephone. Outcome assessors were blind to randomization.

Outcome—The primary outcome measure was the physical function subscale of the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), a self-report measure (17 items) referencing difficulty completing everyday physical tasks over the past 2 weeks [28–30]. All items are rated on a five-point Likert scale ranging from "none" to "extreme" (higher scores indicate worse function). The reliability and the validity of the WOMAC subscales have been confirmed [28, 31]. Cronbach's alphas for the physical functioning subscale at both baseline and 12-month time points were 0.96. The outcome was the change score (12-month score minus baseline score); participants with missing baseline or follow-up WOMAC were not included.

Mediators—The Arthritis Self-Efficacy Scale examines perceived ability to cope with arthritis symptoms [32]. This scale includes eight items asking respondents how certain they are that they can perform specific activities or tasks. Items are scored on a Likert scale (1 = very uncertain to 10 = very certain). The scale has shown acceptable construct validity,

internal reliability, and test-retest reliability [32]. Cronbach's alphas for arthritis self-efficacy were 0.91 at baseline, 0.92 at 6 months, and 0.93 at 12 months. The mediator variable was the change score calculated as 6-month score minus baseline score (12-month score minus baseline score for sensitivity analyses); participant with missing baseline or follow-up data were not included.

Pain control was measured using two items from the Coping Strategies Questionnaire [33]. Participants were asked "Based on all the things you did to cope, or deal, with your arthritis pain during the last week, how much control do you feel you had over it?" and "Based on all the things you did to cope, or deal, with your arthritis pain during the last week, how much were you able to decrease it?" They rated responses on a 0–6 scale (0 = no control to 6 = complete control or can decrease it completely). These two items have been previously used as a measure of pain control related to pain intensity and coping [34, 35]. Cronbach's alphas for pain control were 0.77 at baseline and 6 months and 0.81 at 12 months. The mediator variable was the change in score calculated as 6-month score minus baseline score (12-month score minus baseline score for sensitivity analyses); participants with missing baseline or follow-up data were not included.

Catastrophizing was measured using the Pain Catastrophizing Scale (PCS), a 13-item self-report scale asking participants to rate the degree to which they experience specific thoughts and feelings when experiencing pain on a five-point scale (0 = not at all to 4 = all the time). The PCS has demonstrated good reliability and validity [36–38]. Cronbach's alphas for pain catastrophizing were 0.96 at baseline, 6 months, and 12 months. The mediator variable was the change score calculated as 6-month PCS minus baseline PCS (12-month PCS minus baseline PCS for sensitivity analyses); subjects with missing baseline or follow-up data were not included.

Covariates—Mirroring the main study analyses [25], all models included a dichotomous (white vs. non-white) self-reported race variable, which was used as a stratifying variable for randomization.

Data Analyses

First, descriptive analyses including intercorrelations among all mediation analysis variables were performed. Linear mixed models were then fit with WOMAC physical function change from baseline to 12 months as the outcome using the PROC MIXED procedure using SAS software, version 9.4 (SAS Institute Inc., 2014, Cary, NC). A random effect to account for clustering of PCPs was used. The intervention group indicator variable was dummy coded—½ vs. ½, and all mediator variables were centered at 0, in accordance with Kraemer and colleagues' [22, 23] recommendations for assessing mediators of treatment effects in RCTs. All models included the interactions between the intervention group and mediators to account for any interactive effects [22, 23].

Mediation models were first fit separately for each individual mediator then fit together in a single model. We planned a priori to include all hypothesized mediators in the full model, whether or not they demonstrated significant mediation effects in the individual models. This decision was made to clarify the roles of each individual cognition, in the context of

the others, as previous literature has demonstrated that pain cognitions tend to predict other, similar types of pain cognitions [39]. First, we confirmed the effect of the intervention on change in physical functioning outcome from baseline to 12 months (coefficient *c* in Fig. 1). We then tested for differences between the intervention and control groups in baseline to 6-month change in cognitions (catastrophizing, arthritis self-efficacy, and pain control; coefficient *a* in Fig. 1). Next, we examined whether the baseline to 6-month changes in the cognitive mediators predicted the outcome: baseline to 12-month change in physical functioning, with the intervention group variable in the model (coefficient *b* in Fig. 1). We calculated the product of the coefficients ($a \times b$ [40]) and used RMediation [41] to estimate the mediated effects and their associated asymmetric 95% confidence limits.

Finally, analyses were conducted to explore whether the mediation effect remained over and above variables that could also be considered potential mediators: depression (Patient Health Questionnaire-8 [42]) and physical activity (Community Health Activities Model Program for Seniors scale [43, 44]). These variables were *n* measured, only at baseline and 12 months. The same methods described in the primary analysis were used to explore the mediation effect of baseline to 12-month change in catastrophizing, arthritis self-efficacy, and pain control on baseline to 12-month change in physical functioning in the intervention group. Baseline to 12-month change in depression and physical activity were then added to the model to determine whether the mediation effects were maintained when adjusting for these effects.

Results

Descriptive Analysis

Demographic and baseline scores for all variables based on are presented in Table 1. Randomized participants were an average age of 61.1 (±9.2) years, predominantly male (90.7%), and obese (mean BMI 33.8 ± 5.8), most with osteoarthritis of the knee only (79.3%). There were no significant differences in outcome, mediator, or covariate variables between groups. Mean change scores for outcome, mediator, and covariate variables are presented in Table 2. Some data were missing due to participants who were excluded (due to developing a contraindicated/exclusionary health condition), were lost to follow-up (unable to be reached), scoring algorithms, and/or persons who declined to participate. *N* values for the number of participants per group included in the following analyses are presented in Table 2.

Evaluation of intercorrelations among mediation analysis variables revealed that they were all significantly related (*r* values 0.21–0.52, all *ps* 0.001). As expected, baseline to 6-month increases in arthritis self-efficacy and pain control were correlated with baseline to 12-month improvements in physical functioning. Baseline to 6-month changes in catastrophizing were negatively correlated with baseline to 12-month changes in physical functioning.

Mediation Analysis

First, we confirmed using our model that the intervention group showed a significantly greater improvement in physical functioning compared to the control group (c path in Fig. 2; $c = -3.39$, SE $= 1.54$, $p = 0.04$). Next, we determined whether or not intervention group membership predicted differences in baseline to 6-month change in arthritis self-efficacy, catastrophizing, and pain control separately. Findings, depicted as paths a_1 and a_2 in Fig. 2, revealed that individuals in the intervention group showed greater increases in arthritis self-efficacy and pain control from baseline to 6 months than the control group, but not so for catastrophizing. Third, we examined whether increases in arthritis self-efficacy and pain control predicted baseline to 12-month improvements in physical functioning. When assessed in independent models, both arthritis self-efficacy ($a_1b_1 = -0.86$, SE $= 0.41$, 95% CI [-1.75, -0.16]) and pain control ($a_1b_1 = -0.88$, SE $= 0.38$, 95% CI [-1.72, -0.21]) significantly mediated the link between intervention group and baseline to 12-month change in physical functioning. When included together in the model, along with catastrophizing (presented in Fig. 2), baseline to 6-month changes in arthritis self-efficacy (b_1 path) and pain control (b_2 path) were still significantly related to baseline to 12-month changes in physical functioning, beyond the contributions of intervention group and all interaction terms (intervention group $\times$ change in cognitions). The indirect effects for both arthritis self-efficacy and pain control confirmed mediating roles ($a_1b_1 = -0.54$, SE $= 0.33$, 95% CI [-1.30, -0.04]; $a_2b_2 = -0.59$, SE $= 0.33$, 95% CI [-1.33, -0.08]). The direct effect of intervention group on change in physical functioning was no longer significant after including the mediators in the model ($c' = -2.02$, SE $= 1.52$, $p = 0.20$). Thus, the intervention group's increases in arthritis self-efficacy and pain control cognitions (but not catastrophizing) from baseline to the intervention midpoint helped to explain their greater improvement in physical functioning from baseline to intervention completion compared to the control group.

In an additional analysis, we followed the same procedure to determine whether these mediating effects of cognitive changes in response to the intervention remained when controlling for baseline to 12-month intervention-related changes in depressive symptoms and physical activity. Baseline to 12-month change in arthritis self-efficacy was found to be the only significant mediator of the link between intervention group membership and baseline to 12-month change in physical functioning when baseline to 12-month changes in pain control, depression, and physical activity were included in the model (results available upon request). Thus, the intervention group's increased arthritis self-efficacy over the course of the intervention predicted their greater intervention-related improvements in physical functioning compared to the control group. This impact was distinct from that of their intervention-related changes in depressive symptoms and physical activity.

Discussion

This secondary analysis of a large RCT of a multifaceted OA intervention demonstrated that changes in arthritis self-efficacy and perceived control over pain in the first 6 months of treatment partially explained intervention-related improvements in physical functioning. Contrary to our hypothesis, the intervention group did not report decreases

in catastrophizing at the treatment midpoint compared to the control group, and thus, catastrophizing was not found to mediate improved physical functioning for the intervention group. The mediating effect of arthritis self-efficacy persisted even after adjusting for mediation effects of pain control and catastrophizing and intervention-related changes in physical activity and depression symptoms, two alternative potential intervention-related processes of change. These analyses are the first to demonstrate pain cognitions as mechanisms of change in a multifaceted intervention for chronic pain in a controlled design where the mediators precede the outcomes, improving upon past weaknesses in the literature [21]. The results are consistent with cognitive-behavioral and health behavior change theories, building an even stronger case for the importance of cognitions in functional improvements through behavioral means in OA and other chronic pain populations.

These findings reinforce a key role for self-efficacy as a mechanism of change in multifaceted interventions for chronic pain. Self-efficacy is a common thread in multiple health behavior change theories, e.g., social cognitive theory, health locus of control, and theory of self-regulation [16, 45–49]. The multifaceted intervention in the current study specifically targeted self-efficacy, using skills including mastery, modeling, persuasion, and reinterpretation of symptoms (cognitive restructuring). Goal setting was also a primary component of the intervention; counselors helped participants adjust goals when they reported low self-efficacy for accomplishing goals. Our findings are consistent with studies suggesting a mediating role for self-efficacy in predicting physical functioning improvements after exercise [14, 17] and CBT interventions [10].

While pain control/helplessness has been found to mediate change in disability in some trials of CBT and multidisciplinary treatment approaches (e.g., [10, 18, 50]), in at least one study of behavioral interventions for chronic low back pain, perceived pain control did not improve [11]. In Turner and colleagues' [10] temporal mediation analysis of CBT, self-efficacy and pain control were both found to mediate CBT-related disability improvements in individual models. However, when included in the same model, self-efficacy predicted little variance in disability beyond perceived control. The authors interpreted these findings to indicate that these constructs might be related. In our study, both self-efficacy and pain control showed significant improvement, even when included together in the mediation model. However, only self-efficacy remained significant when additional possible mediators were included in the 12 month analysis. These findings suggest the need for further analysis regarding relations between pain cognition processes of change.

Contrary to our hypotheses, there were no differences in catastrophizing between the intervention and control groups at the midpoint or conclusion of the intervention. This finding is inconsistent with a number of past studies suggesting a mediating role for catastrophizing in functional improvements after multiple types of interventions (e.g., [10–12, 24, 51, 52]). Whereas there were a number of planned techniques to increase self-efficacy through the study, cognitive restructuring was the primary mechanism proposed to target catastrophizing, and it was a relatively small part of a large intervention (2/18 sessions) that occurred toward the end of treatment (after the midpoint mediation assessment). Thus, any change from baseline to midpoint was related to the physical activity, weight loss, and behavioral therapy (e.g., relaxation) aspects of the intervention

covered in the first 6 months. However, we also did not find an intervention group difference in catastrophizing from baseline to 12 months. The dose of cognitive restructuring in the intervention may have been too low to effect a significant change in pain catastrophizing. Given past data showing links between decreased catastrophizing and improved pain-related outcomes, the current intervention may have demonstrated more markedly improved outcomes if it had resulted in decreases in catastrophizing. Future trials investigating mediating effects should consider additional measurement points throughout the intervention, at time points relevant to the timing of the intervention mechanism proposing to target the mediator, in order to better capture change in mediators while also maintaining temporal precedence.

Some limitations should be noted regarding these analyses. We focused on cognitions as mechanisms, but there are other potential processes of change for improving functioning in these types of interventions (e.g., increased strength and endurance through exercise, decreased weight resulting in less pain). Future studies should use a theory-based approach to identify and assess other potential mediators in order to understand relative impact on outcomes, in order to improve interventions. Additionally, as the intervention consisted of multiple components (physical activity, cognitive-behavioral), many of which could impact cognitions, it is not possible to determine the relative contribution of each component on the change in cognitions. Subjects with missing follow-up data were case deleted; however, results from our models for the physical function outcome were similar to those presented in the main study paper, which used all available subject data indicating that case deletion was not problematic. Furthermore, we found in the main study paper that results from a sensitivity analysis using multiply imputed data were very similar to the main study results [25]. As shown on the CONSORT diagram in the main outcomes paper [25], the numbers of individuals in the intervention and control groups were similar with respect to missed assessments/lost to follow-up/exclusions and withdrawals at 6 and 12 months. For WOMAC physical function, an additional subject in each arm is missing due to missing items in the survey at 12 months. For the mediators, with respect to missed assessments/lost to follow-up/exclusions and withdrawals at 6 months, 21 observations were missing from the intervention group and 16 observations were missing in usual care. For pain catastrophizing, there were an additional six subjects in control and seven subjects in the intervention group missing due to missing items in the survey at 6 months. Finally, the study population consisted of Veterans (primarily male); thus, these findings may not generalize across different populations.

In summary, this study's temporal, controlled design allowed for a rigorous mediation analysis of intervention-related effects on outcomes in a large sample of patients with OA. This analysis was the first to our knowledge to demonstrate that arthritis self-efficacy and pain control serve as mechanisms of change through a multifaceted intervention for chronic pain involving physical activity, weight loss, and cognitive-behavioral components. These findings add to previous literature asserting the critical role of cognitions in improving functional outcomes for patients with chronic pain. Our findings suggest that behavioral interventions for chronic pain should emphasize techniques shown to increase self-efficacy, such as development and practice to relevant skills, observation of others, and personal accomplishments to increase experiences of mastery [45,46, 53, 54]. Increased focus on

cognitive restructuring, with the goal of increasing adaptive cognitions such as self-efficacy, may promote improved outcomes in future interventions. Ongoing work exploring how and why functional outcomes improve throughout the course of behavioral treatments for chronic pain will help inform the design of more effective and more efficient interventions.

Funding Information

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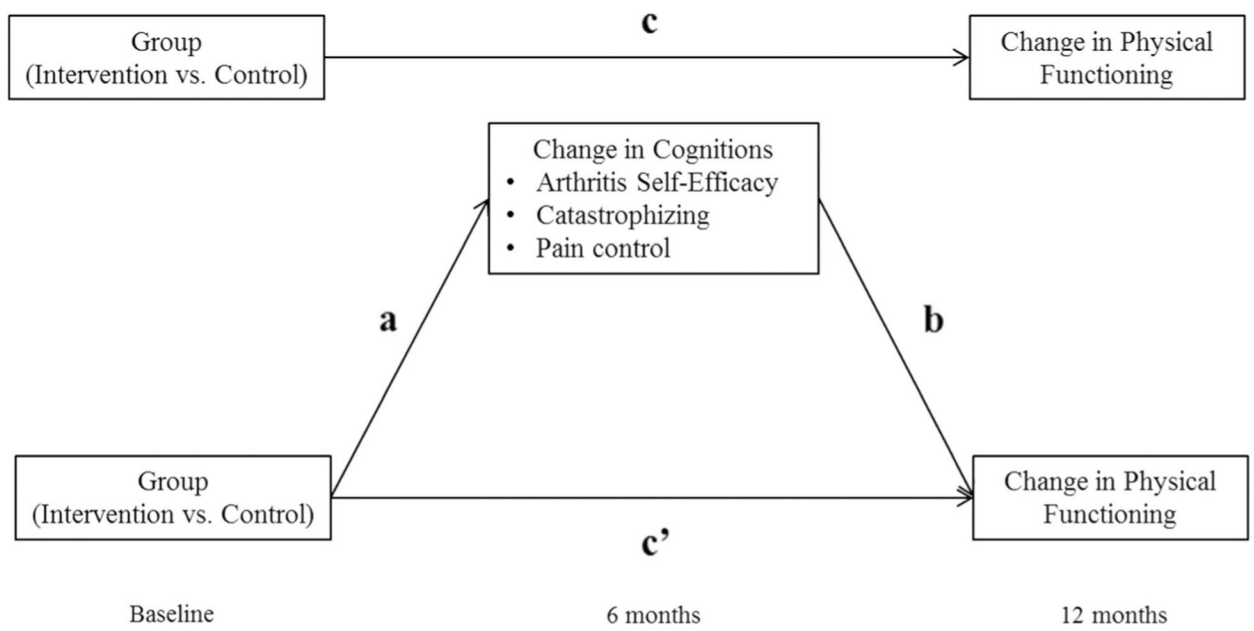
Fig. 1.
Model depicting mediation of the effect of the OA intervention compared to the control group on change in physical functioning over the 12-month intervention by baseline to 6-month changes in cognitions
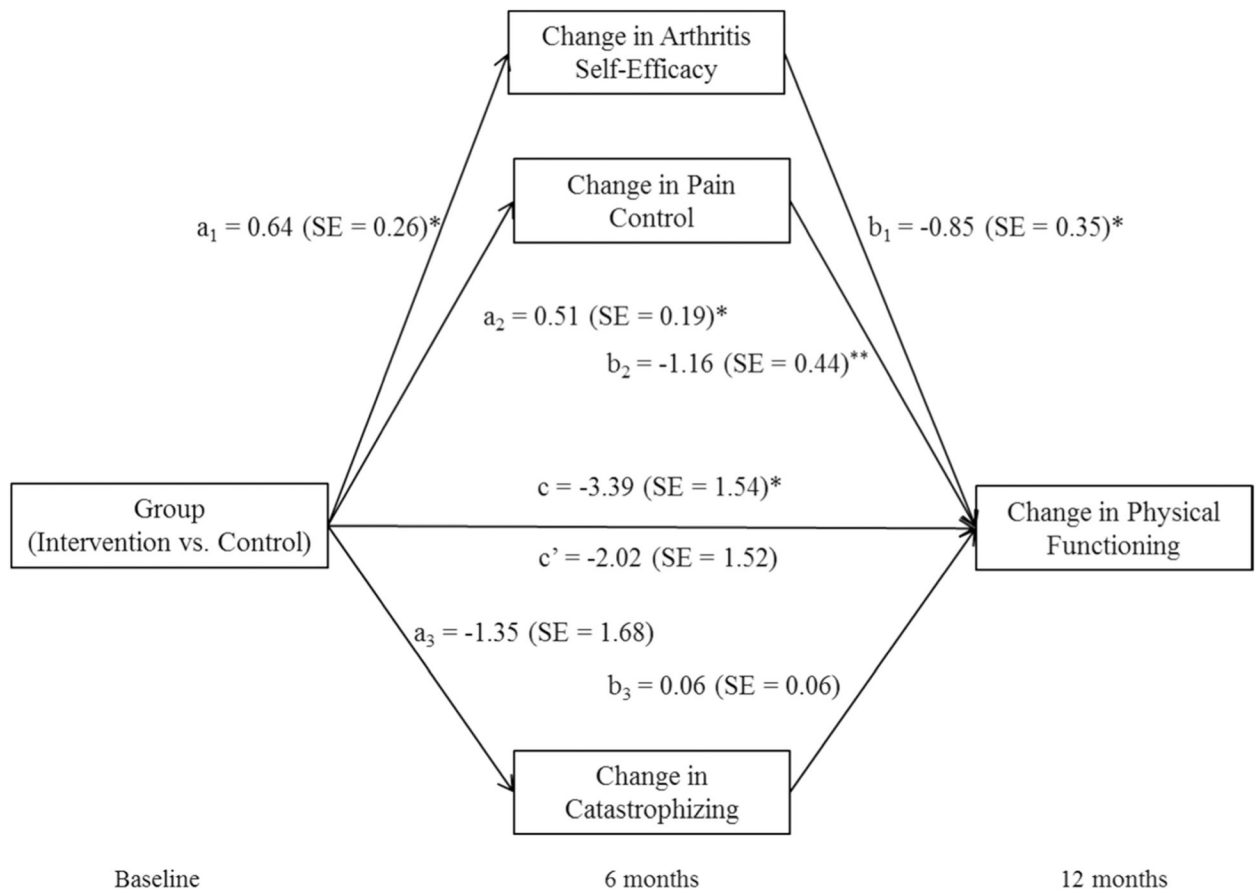
Fig. 2.
Mediation of intervention-related baseline to 12-month change in physical functioning by baseline to 6-month change in cognitions. *$p < 0.05$; **$p < 0.01$

Table 1

Patient participant characteristics at baseline

Characteristic	Overall, $n = 300$	Usual care, $n = 149$	Osteoarthritis intervention, $n = 151$
Mean age (SD) (years)	61.1 (9.2)	61.7 (9.0)	60.4 (9.4)
Men, n (%)	272 (90.7)	141 (94.6)	131 (86.8)
Non-white race, n (%)	150 (50.0)	75 (50.3)	75 (49.7)
Married or living with partner, n (%)	199 (66.3)	106 (71.1)	93 (61.6)
High school education or less, n (%)	81 (27.0)	44 (29.5)	37 (24.5)
Inadequate income, n (%)	103 (34.3)	48 (32.2)	55 (36.4)
Employed or student, n (%)	127 (42.8)	60 (40.5)	67 (45.0)
Disabled, n (%)	98 (33.0)	53 (35.8)	45 (30.2)
Mean BMI (SD) (kg/m^2)	33.8 (5.8)	33.4 (5.7)	34.3 (6.0)
Joints with osteoarthritis, n (%)			
Knee only	238 (79.3)	124 (83.2)	114(75.5)
Hip only	32 (10.7)	14 (9.4)	18(11.9)
Knee and hip	30 (10.0)	11 (7.4)	19(12.6)
Mean WOMAC-PF score (SD) (0–68)	33.8 (13.0)	33.4 (12.9)	34.2 (13.1)
Mean PHQ-8 score (SD) (0–24)	6.8 (5.4)	6.4 (5.1)	7.2 (5.6)
Median CHAMPS all exercise duration (IQR) (h/week)	10.0 (4.8–17.3)	10.8 (5.0–17.9)	9.8 (4.8–16.8)
Mean arthritis self-efficacy (SD) (1–10)	5.1 (2.0)	5.2 (2.0)	5.1 (2.0)
Mean pain control (SD) (0–4)	2.8 (1.5)	2.8 (1.5)	2.7 (1.5)
Mean catastrophizing (SD) (0–52)	18.7 (13.9)	18.4 (13.4)	19.0 (14.4)

Percentages may not sum to 100 due to rounding

BMI body mass index, *CHAMPS* Community Healthy Activities Model Program for Seniors, *IQR* interquartile range, *PHQ-8* Patient Health Questionnaire-8, *WOMAC-PF* Western Ontario and McMaster Universities Osteoarthritis Index-Physical Functioning scale

Table 2

Mean change outcome, mediators, and covariates from baseline to 6 months (intervention midpoint) and 12 months (intervention conclusion)

Variable	Usual care	Osteoarthritis intervention
Outcome		
Mean change in WOMAC-PF score (0–68) (SD)		
Baseline to 12 months	0.7 (10.9) $N = 136$	−2.7 (9.5) $N = 135$
Mediators		
Mean change in arthritis self-efficacy (1 −10) (SD)		
Baseline to 6 months	0.1 (1.9) $N = 133$	0.7 (2.2) $N = 128$
Mean change in pain control (0–4) (SD)		
Baseline to 6 months	0.4 (1.6) $N = 133$	0.9 (1.6) $N = 128$
Mean change in catastrophizing (0–52) (SD)		
Baseline to 6 months	1.5 (11.0) $N = 128$	0.5 (10.4) $N = 123$

WOMAC-PF Western Ontario and McMaster Universities Osteoarthritis Index-Physical Functioning scale