Asia-Pacific Journal of Public Health
2015, Vol. 27(2) NP382–NP391

DOI: 10.1177/1010539512443974
aph.sagepub.com
SAGE

Does Functional Disability Mediate the Pain–Depression Relationship in Older Adults With Osteoarthritis? A Longitudinal Study in China

Qian Wang, MPH, DrPH[1], Rohan Jayasuriya, MBBS, MD[2], Wing Young Nicola Man, BVSc[2], and Hua Fu, MB, PhD[3]

Abstract

Older adults with osteoarthritis have been found to be impaired in physical functioning and report higher levels of depression. This study examined the relationships between pain, functional disability, and depression to test the activity restriction model in a cohort of 176 older adults in China. This model states that disability is a mediator for the relationship between pain and depression. Other investigators have found that pain and disability were two independent correlates of depression. In both cross-sectional and longitudinal analyses, the authors found that disability is a mediator, using commonly accepted methods (indirect effect 44%, Sobel $Z = 4.07$, $P < .001$; 41%, Sobel $Z = 3.45$, $P < .001$, respectively). However, this mediation effect was not seen when the outcome was residualized with the baseline value. When the baseline level of depression is residualized, the effect size of the relationship is reduced, requiring larger sample size to test its effect.

Keywords

activity restriction model, depression, functional disability, mediation, moderation, osteoarthritis (OA), pain

Introduction

Osteoarthritis (OA) is one of the commonest types of arthritis among older adults. According to recent data from China, about 20% of elderly people (defined as age 50 years and older) suffer from radiographic OA.[1] The prevalence is similar to that reported in the United States,[2] and the numbers of OA sufferers are growing steadily.

[1]Turning Point Alcohol and Drug Centre, Fitzroy, Victoria, Australia
[2]University of New South Wales, Sydney, New South Wales, Australia
[3]Fudan University, Shanghai, China

Corresponding Author:
Qian Wang, Population Health Research, Turning Point Alcohol and Drug Centre, 54-62 Gertrude Street, Fitzroy, Victoria 3065, Australia.
Email: kenw@turningpoint.org.au

The management of OA requires attention to functional disability and pain, which often follow progressive change in the joints. Studies have found that pain is a better predictor of disability than radiographic change.[3] Although some studies have found a relationship between OA disability (measured by level of function) and psychological factors in older adults,[3-5] the role of psychological factors in OA is insufficiently understood.[6]

Older adults with OA have been found to be impaired in physical functioning and report higher levels of depression.[7] Older adults with chronic illness such as OA are more likely to suffer from depressive symptoms when compared with their healthy counterparts.[8] A more recent study on successful aging echoes this finding by demonstrating that years of having arthritis increase the incidence of depression and functional impairment among the elderly.[9] The existence of linkages among pain, depression, and functioning is supported by evidence from a number of key studies.[10-12]

As OA results in physical limitations and social and occupational disability, patients may experience psychological impacts.[10] Williamson and Schulz[11] and Williamson[12] reasoned that pain would prevent sufferers from carrying out daily activities and that such functional constraints could then result in psychological distress such as depression and anxiety. They proposed an activity restriction model, which states that pain increases depressive affect and also restricts physical function, which in turn contributes to depression. To test their hypothesis, they carried out a mediation analysis, which revealed that functional disability mediates the relationship between pain and depression.[12] Parmelee et al[13] found that pain and disability were two independent correlates of depression in institutionalized elderly. These studies were cross-sectional, and to address this shortcoming, a study was conducted[14] to examine the contribution of pain and disability to depression in people with OA of the knee, using both cross-sectional and longitudinal analyses. In the cross-sectional results, they found that pain mediated the effects of disability on depression. In the longitudinal analysis, when the effects of baseline level of depression were residualized, neither baseline pain nor baseline functional disability was associated with change in depression over a one-year period. Another longitudinal study of elderly community residents did not find support for the hypothesis that the pain–depression relationship was mediated by disability.[15] Parmelee et al[14(p458)] concluded that "although the basic inter-relationship of depression, pain and functional disability is almost indisputable, mechanisms underlying it remain in question."

There is increasing evidence as to the role of self-efficacy as an important psychological variable related to outcomes in arthritis management. In a longitudinal study of men and women with OA of the knee, high baseline self-efficacy decreased the odds of self-reported poor physical functioning 3 years later.[16] Studies using the arthritis self-management (ASM) model[17,18] have consistently shown that clinical improvement in symptoms (eg, pain, depression, and functional disability) was linked to baseline and improved self-efficacy in coping.

The aim of the present study is to further our understanding of the temporal relationships between pain, functional disability, and depression in OA. First, we hypothesize that OA-related pain, self-efficacy for pain, and functional disability at baseline are significant predictors of subsequent depression. Second, given the equivocal findings of the role of functional disability as a mediator of the relationship between pain and depression in older adults, we hypothesize that functional disability mediates the relationship between pain and depression in cross-sectional analysis and in longitudinal analysis as postulated by Williamson and Schulz[19] in the activity restriction model.

Methods

Participants

Participants were drawn from an existing urban cohort of elderly community residents in Shanghai, China, to whom an ASM intervention had been delivered 1½ years before the present

study commenced in 2006. To be eligible for the present study, at the time of recruitment participants had to be community-dwelling residents aged 50 years or older, with OA as a primary medical condition. We successfully approached 176 eligible older adults who agreed to participate. They were given an appointment at a time of their convenience to attend a local seniors' center. Participants who turned up on the day were invited to complete a questionnaire written in Chinese after oral consent was obtained prior to its administration. Each participant was also required to complete the same questionnaire 6 months after the baseline. Participants' height and weight were measured by trained health workers at follow-up, from which body mass index in kg/m^2 was calculated. Each participant was given a gift valued equivalent to AU$2 to compensate for their time in the study. The study was approved by the Human Research Ethics Committee of the University of Wollongong, Australia, and the institutional review board of the School of Public Health, Fudan University, China.

Measures

Depression. Depression was assessed using the 15-item Geriatric Depression Scale. This scale has been used widely in older populations. Scale items included "Are you basically satisfied with your life?" and "Have you dropped many of your activities and interests?" with yes/no response options. Each answer was assigned a score of 0 or 1 depending on the answers given, resulting in a maximum score of 15. A score higher than 5 is generally considered indicative of a depressed mood. Cronbach's α for the Geriatric Depression Scale has been reported to be higher than .80 in past research[20] and has been tested in Chinese populations.[21]

Pain and physical function. The pain and physical function scales were derived from the Western Ontario and McMaster University Osteoarthritis index. For pain, each item is preceded by the stem question: "How painful do you feel . . .", whereas for functioning, the stem question is "What degree of difficulty do you have with . . .". Participants were asked to rate arthritic pain experienced or their difficulty with activities of daily living during the past month on a visual analogue scale from 0 to 100, with higher scores indicating greater pain or difficulty. The mean of the responses to the subscale questions was taken as the overall pain or physical function score. Reliabilities of the measures (Cronbach's αs) have been reported as .70 to .81 for pain and .89 to .91 for function.[22]

Arthritis self-efficacy for pain. A 5-item pain subscale of the Arthritis Self-efficacy Scale was used to measure participants' perceived ability to control their arthritis-related pain. Items included the following: "How certain are you that you can decrease your pain quite a bit?" and "How certain are you that you can continue most of your daily activity?". Each question was answered on an 11-point Likert-type scale, ranging from 0 (*very uncertain*) to 10 (*very certain*), and the total score was derived from the mean of the responses. In other words, higher scores on the scale indicated greater confidence about ability to control OA-related pain. The reliability of the Arthritis Self-efficacy Scale relating to pain, in terms of Cronbach's α, was previously reported as .75.[23]

Statistical Analysis

Raw data were first screened for normality, skewness, kurtosis, and univariate or multivariate outliers. The Shapiro–Francia test of normality showed that the main variables of interest (ie, pain, functional disability, depression, and self-efficacy) had highly significant departures from normality ($P < .001$). Visual examination of histogram plots also indicated that pain, depression, and functional disability were positively skewed and self-efficacy was negatively skewed. Hence, an origin-shifted logarithmic, $\log_e(\pm x + k)$, transformation[24] was used for transformation of the scores, where x represents measures of pain, depression, functional disability,

Table 1. Correlations of Time 1 and Time 2 Study Variables.[a]

	Pain1	Func1	Dep1	Sep1	Pain2	Func2	Dep2	Sep2
T1 Pain (Pain1)	**.91**							
T1 Functional disability (Func1)	.79[b]	**.96**						
T1 Depression (Dep1)	.28[c]	.39[b]	**.75**					
T1 Self-efficacy for Pain (Sep1)	−.28[b]	−.33[b]	−.35[b]	**.85**				
T2 Pain (Pain2)	.46[b]	.34[b]	.17	−.21[d]	**.85**			
T2 Functional disability (Func2)	.35[b]	.37[b]	.28[c]	−.23[c]	.77[b]	**.97**		
T2 Depression (Dep2)	.15	.30[b]	.54[b]	−.40[b]	.15	.22[d]	**.75**	
T2 Self-efficacy for Pain (Sep2)	−.13	−.17	−.28[c]	.31[b]	−.27[c]	−.41[b]	−.26[c]	**.91**

[a]Cronbach's αs are shown in boldface on the diagonal.
[b]$P < .001$.
[c]$P < .01$.
[d]$P < .05$.

or self-efficacy and k is a constant that was derived so that there would be zero skewness in the transformed variable.[25] Three cases were identified as being both univariate and multivariate outliers; therefore, they were excluded before transformation. All subsequent (correlation and regression) analyses were performed on the transformed variables, controlling for age, gender, education, marital status, body mass index, and number of self-reported comorbidities.

Pearson's correlation coefficients were calculated to examine associations among all variables of interest. Sequential multiple linear regression analyses were then carried out with depression at follow-up (T2) as the dependent variable to test the first hypothesis. Interactions between pain and functional disability as well as pain and self-efficacy were also fitted in the final steps of the sequential linear regression analysis to test for moderation.

Further regression analyses were undertaken to test for mediation using steps developed by Baron and Kenny[26] and further refined by Preacher and Hayes.[27] A series of Sobel tests was used to assess the significance of the mediated effect.[28] As the Sobel test is performed using the product of coefficients, computation of the standard error of the resulting product coefficient using Taylor series approximation works well only with a reasonably large sample size. Hence, the bootstrapping procedure with 10 000 resamples was applied to obtain more robust results. The authors modified the program *sgmediation*[29] in Stata to produce Sobel test and bootstrap intervals for the standardized coefficients. The statistical software, SPSS version 18 for Windows (SPSS, Chicago, IL) and/or Stata, version 11.2 (StataCorp LP, College Station, TX) were used in the data analyses.

Results

The baseline sample comprised 173 people with OA, 136 of whom were available at 6-month follow-up after exclusion of the 3 participants with multivariate outliers from the data set. No statistically significant differences were found in terms of sociodemographic characteristics and other variables between those who remained and those who dropped out at follow-up (data not shown). At baseline, the mean age of the participants was 62 years and the sample consisted of 83% females. Participants had an average of 8.7 years of education, and more than 80% were married.

The internal reliability of the measures was examined by means of Cronbach's α. As shown on the diagonal from Table 1, Cronbach's αs for each of the scales at both T1 and T2 ranged from .75 to .97, suggesting good internal consistency of the scales across collections.

Table 2. Hierarchical Linear Regression Analyses for Change in Depression.

Step	Variables	Statistics by Step			Statistics by Variable		
		Total R^2	R^2 Change	Standardized β	Final Standardized β	*t*	*P*
Outcome: Depression at follow-up							
1.	Depression (T1)	.30	.30	0.55	0.42	5.00	.00
2.	Age (in years)			−0.03	−0.05	−0.53	.60
	Female (vs male)			−0.09	−0.04	−0.45	.66
	Education (in years)			−0.12	−0.11	−1.24	.22
	Currently married (vs currently not married)	.33	.03	−0.05	−0.06	−0.79	.43
3.	Body mass index (T2)			−0.02	−0.04	−0.54	.59
	Number of comorbidity (T1)	.33	.00	0.04	>−0.001	−0.01	.99
4.	Pain (T1)	.33	.00	0.05	−0.18	−1.35	.18
5.	Functional disability (T1)	.35	.02	0.27	0.27	1.84	.07
6.	Self-efficacy for pain (T1)	.38	.03	−0.18	−0.19	−2.25	.03
7.	Pain × functional disability (T1)	.40	.02	−0.04	>−0.01	−0.06	.96
8.	Pain × self-efficacy for pain (T1)	.40	.00	0.14	0.14	1.86	.07

Correlations between the key variables are also displayed in Table 1. Pain, functional disability, and depression were significantly correlated at both time points. Pain and functional disability were highly correlated at T1 and T2. The variable most strongly correlated with functional disability (at T2) was OA-related pain (at T2) ($r = .77$, $P < .001$), whereas the variable most strongly correlated with depression (at T2) was depression (at T1) ($r = .54$, $P < .001$).

Table 2 shows the results of sequential regression analysis of pain and functional disability on depression at follow-up. Depression at follow-up was first regressed on baseline depression (at step 1), which explained 30% of variance and made a significant contribution to the equation (F for change in $R^2 = 53.79$, $P < .001$). Together, the demographic variables contributed 3% of the unexplained variance (F for change in $R^2 = 1.10$, $P = .36$), and no individual demographic factor significantly predicted depression at T2 after controlling for depression at T1.

Pain at T1 in step 4 did not account for any of the unexplained variation in depression at T2 (F for change in $R^2 = 0.43$, $P = .52$). Functional disability and self-efficacy at T1 were included at steps 5 and 6 and accounted for a further 2% (F for change in $R^2 = 3.58$, $P = .06$) and 3% (F for change in $R^2 = 4.84$, $P < .05$) of the variance, respectively. No interaction terms at baseline (ie, between pain and functional disability or between pain and self-efficacy) were found to be significant (F for change in $R^2 = 0.20$, $P = .65$; F for change in $R^2 = 3.46$, $P = .07$, respectively). In other words, there was no significant moderation of the effect of pain on depression by either functional disability or self-efficacy.

According to Baron and Kenny,[26] criteria for testing a mediation effect include the following: (*a*) the predictor must be significantly associated with the outcome variable, (*b*) the predictor must be significantly associated with the hypothesized mediator, (*c*) the mediator must be significantly associated with the outcome variable, and (*d*) the effect of the predictor on the outcome variable must be smaller when both predictor and mediator are included than when the predictor

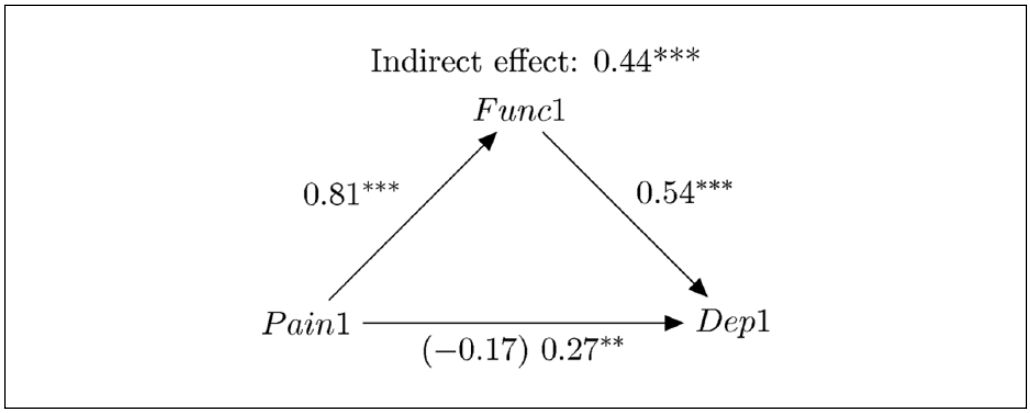

Figure 1. Cross-sectional mediational analysis for pain, depression, and functional disability[a]
Abbreviations: Pain1, pain at T1; Func1, functional disability at T1; Dep1, depression at T1.
[a]Value (β) within parenthesis represents the direct effect of the predictor on the residualized outcome variable controlling for other covariables (sociodemographics, body mass index, and number of comorbidities). Value outside the parentheses represents the total effect of the predictor on the residualized outcome variable when both predictor and mediator have been entered into the regression equation simultaneously while controlling for other covariables. *$P < .05$; **$P < .01$; ***$P < .001$.

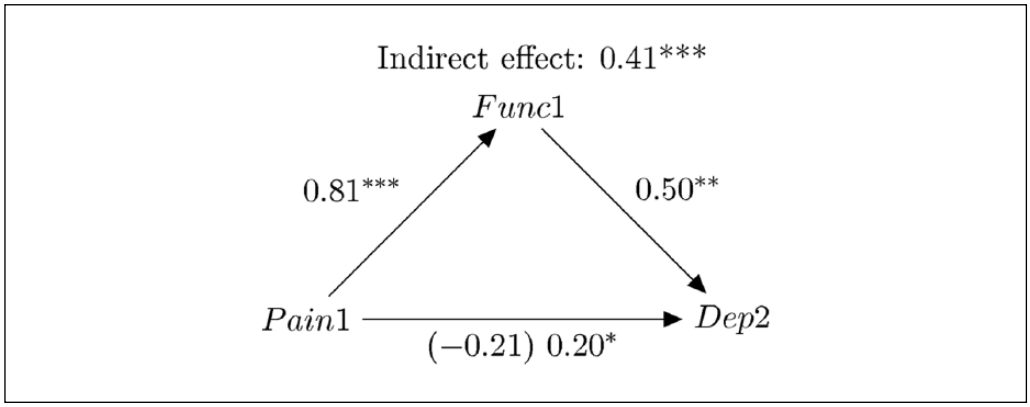

Figure 2. Longitudinal mediational analysis for pain, depression, and functional disability[a]
Abbreviations: Pain1, pain at T1; Func1, functional disability at T1; Dep2, depression at T2.
[a]Value (β) within parenthesis represents the direct effect of the predictor on the outcome variable controlling for other covariables (sociodemographics, body mass index, and number of comorbidities). Value outside the parentheses represents the total effect of the predictor on the outcome variable when both predictor and mediator have been entered into the regression equation simultaneously while controlling for other covariables. *$P < .05$; **$P < .01$; ***$P < .001$.

alone predicts the outcome variable. However, when the first criterion is not satisfied, indirect effect can still exist as explained by Preacher and Hayes.[27]

In the mediational models shown in Figures 1 and 2, the relationships between pain, depression, and functional disability were examined in cross-sectional and longitudinal models, respectively. At baseline, pain significantly predicted functional disability ($\beta = 0.81$, $P < .001$) in the two models in Figures 1 and 2.

Figure 1 shows the cross-sectional mediational model in which functional disability at T1 was hypothesized to be the mediator when pain at T1 was the main predictor and depression at T1 the outcome variable. When pain and disability were entered into the linear regression simultaneously to predict depression, the effect of pain on depression was reduced by 0.44 (the indirect

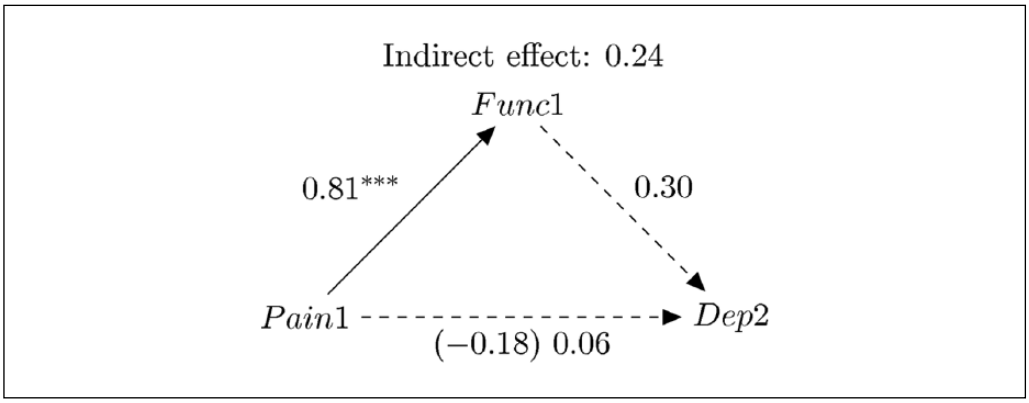

Figure 3. Residualized mediational analysis for pain, depression, and functional disability[a]
Abbreviations: Pain1, pain at T1; Func1, functional disability at T1; Dep2, depression at T2.
[a]Value (β) within parenthesis represents the direct effect of the predictor on the outcome variable controlling for other covariables (sociodemographics, body mass index, and number of comorbidities). Value outside the parentheses represents the total effect of the predictor on the outcome variable when both predictor and mediator have been entered into the regression equation simultaneously while controlling for other covariables. *$P < .05$; **$P < .01$; ***$P < .001$.

effect, Sobel $Z = 4.07$, $P < .001$), from β = 0.27 (the total effect, $P = .003$) to β = −0.17 (the direct effect, $P = .22$). The direct effect of pain becoming negative and not significantly different from zero suggests that disability completely mediated the relation between pain and depression at baseline. The bias-corrected 95% confidence interval (CI) (0.23-0.67) from bootstrapping of the product coefficient (estimating the indirect effect) did not contain zero, which further confirmed the significance of the mediated effect.

Figure 2 displays the longitudinal mediational model in which functional disability at T1 was hypothesized to be the mediator when pain at T1 was the predictor and depression at T2 the outcome variable. The β for indirect effect of pain via functional disability was 0.41 (Sobel $Z = 3.45$, $P < .001$), as β decreased from 0.20 ($P = .03$) for the total effect to −0.21 ($P = .18$) for the direct effect when functional disability at T1 was added to the equation. The bias-corrected 95% CI (0.16-0.68) of the indirect effect did not contain zero, which further confirmed the significance of the mediated effect.

Figure 3 presents the longitudinal (residualized outcome) mediational model in which functional disability at T1 was hypothesized to be the mediator when pain at T1 was the predictor and residualized depression the outcome variable. Neither the total effect (β = 0.06, $P = .50$) nor the direct effect (β = −0.18, $P = .32$) of pain at T1 on residualized depression was statistically significant. In other words, the first condition for establishing mediation was not met. Further analysis revealed that functional disability at T1 did not significantly affect residualized depression (β = 0.30, $P = .07$) after controlling for pain at T1. The indirect effect of pain via functional disability at T1 on residualized depression was not significant either (β = 0.24, bias-corrected 95% CI = −0.05-0.54, Sobel $Z = 1.64$, $P = .10$), that is, there was no evidence of significant indirect effect.

Discussion

In this study, we found that all four variables, pain, functional disability, self-efficacy for pain, and depression, were correlated in a sample of community-dwelling older adults in China. The main factors that predicted depression at follow-up after the effects of baseline levels of depression were removed (residualized) were functional disability at baseline and self-efficacy for pain at baseline. This provides additional evidence for the value of increasing coping skills for older

adults with pain due to OA as those with higher levels of self-efficacy for controlling pain were less depressed. This cohort had received the ASM intervention in the past, and this may be one explanation for the lower levels of depression in this group of community living older adults than those investigated in other studies.[14,19]

Theorists who advocate the activity restriction model argue that pain contributes to depression through its impact on functional disability.[11,12,19] Those who favor the common cause model argue that pain is a mediator between depression and disability among OA sufferers.[13,14] In this study, we addressed this question by testing for the mediation effect of functional disability. Using cross-sectional data, mediational analysis showed that functional disability mediated the relationship between pain and depression. The same relationships were observed in longitudinal analysis, where functional disability at baseline mediated the relationship between pain at baseline and depression at follow-up. Therefore, when using the same analysis as Williamson and Schulz[11] the findings of our study support the activity restriction model.[19]

Parmelee et al[14] found no support for the mediation effect of functional disability in longitudinal analysis. In their study, Parmelee et al[14] used a measure of depression at follow-up after taking into account the effect of depression at baseline by residualizing the baseline effect. When we used a similar analysis, we found similar results, and the indirect effect of pain via functional disability was not statistically significant.

In summary, this study provides additional evidence in favor of the activity restriction model in a sample of community-living older adults from a non-Western cultural background. In our study, we found that one of the contributory reasons for the equivocal results of the mediation effect of functional disability on the relationship of pain and depression in patients with OA could be the methods of analysis used.

There is evidence that past depression affects current depression.[14] To account for this, residualization of follow-up depression for baseline depression and/or fitting baseline depression as a predictor should be performed. When the baseline level of an outcome (in this case depression) is residualized or adjusted for when testing for mediation, the effect size of the relationship between the antecedent (in this case pain) and outcome (depression) decreases and larger samples are required to detect correlations.[30] This may explain the findings of Parmelee et al[14] and our findings, when we used residualized outcome variables for the regression models.

Other explanations for the findings are that participants who had received the ASM program, in which they were taught to reduce worry because of pain, were able to reduce the effect of pain on depressive thoughts. One indication of this effect is seen as self-efficacy for pain at baseline was a significant predictor of depression at follow-up. Moreover, there could be cultural reasons that among Chinese people, pain is more acceptable in older age, as compared with people from Western cultures.[15] It appears that the role of functional disability as a mediator is complex and varies with the level of depression and characteristics of the patients. Further study is required to tease out these relationships.

There are some limitations for generalizing the findings of our study. First, the sample comprised OA sufferers from an urban community in China and the findings cannot be generalized to other populations in China. Another limitation was that because of constraints of time and resources, the follow-up study was carried out within 6 months. This may not have been a sufficient period to observe changes in some of the factors of interest, particularly long-term changes in pain, functional disability, and depression.

Unlike pain and functional disability, depression is a relatively dynamic variable with large variation over short periods (weekly) or even within the day, which contributes to the weaker associations observed between depression and its predictors, compared with functional disability and its predictors. Further longitudinal research with more follow-up time points are indicated. Such methods can also be used for determining the optimal lag period between the outcome (ie,

depression) and its predictors for measurements to be taken, which would be useful for the planning of future studies.

Conclusion

To our knowledge, this is the first study conducted in a non-Western cultural setting to test the activity restriction model, where disability is a mediator for the relationship between pain and depression on older adults. Our findings indicate that functional disability is a mediator, using commonly accepted methods for mediation analysis. However, similar to findings of Parmelee et al,[14] this mediation effect was not seen when the outcome was residualized with the baseline value. The equivocal findings of others may be explained by the type of analysis for mediation. When the baseline level of depression is residualized, the effect size of the relationship is reduced, requiring larger sample size to test its effect.

In addition, the findings also support the value of interventions that increase the capacity of people with arthritis to deal with pain, especially by increasing their self-efficacy for controlling pain. In translation of research to practice, these results add to the value of self-management interventions for persons with arthritis and specifically OA. The findings have direct application to low- to middle-income countries such as China, where joint replacement surgery is not a viable option for many people with OA.

Acknowledgments

We would like to acknowledge instrumental support provided by Dr Zhu YongYuan at Lin Fen Community Health Service Centre in Shanghai, China. We would like to thank health workers and medical students who involved in the data collection and residents from Lin Fen Community who participated in this study.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

References

1. Kang X, Fransen M, Zhang Y, et al. The high prevalence of knee osteoarthritis in a rural Chinese population: the Wuchuan osteoarthritis study. *Arthritis Rheum*. 2009;61:641-647.
2. Lawrence RC, Felson DT, Helmick CG, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II. *Arthritis Rheum*. 2008;58:26-35.
3. Creamer P. Osteoarthritis pain and its treatment. *Curr Opin Rheumatol*. 2000;12:450-455.
4. Salaffi F, Cavalieri F, Nolli M, Ferraccioli G. Analysis of disability in knee osteoarthritis. relationship with age and psychological variables but not with radiographic score. *J Rheumatol*. 1991;18: 1581-1586.
5. Keefe FJ, Lefebvre JC, Maixner W, Salley AN, Caldwell DS. Self-efficacy for arthritis pain: relationship to perception of thermal laboratory pain stimuli. *Arthritis Care Res*. 1997;10:177-184.
6. Peters TJ, Sanders C, Dieppe P, Donovan J. Factors associated with change in pain and disability over time: a community-based prospective observational study of hip and knee osteoarthritis. *Br J Gen Pract*. 2005;55:205-211.
7. Zautra AJ, Smith BW. Depression and reactivity to stress in older women with rheumatoid arthritis and osteoarthritis. *Psychosom Med*. 2001;63:687-696.

8. Sherina MS, Rampal L, Mustaqim A. Factors associated with chronic illness among the elderly in a rural community in Malaysia. *Asia Pac J Public Health*. 2004;16:109-114.
9. Hsu H-C. Impact of morbidity and life events on successful aging. *Asia Pac J Public Health*. 2011;23:458-469.
10. Hurley MV, Mitchell HL, Walsh N. In osteoarthritis, the psychosocial benefits of exercise are as important as physiological improvements. *Exerc Sport Sci Rev*. 2003;31:138-143.
11. Williamson GM, Schulz R. Activity restriction mediates the association between pain and depressed affect: a study of younger and older adult cancer patients. *Psychol Aging*. 1995;10:369-378.
12. Williamson GM. Extending the activity restriction model of depressed affect: evidence from a sample of breast cancer patients. *Health Psychol*. 2000;19:339-347.
13. Parmelee PA, Katz IR, Lawton MP. The relation of pain to depression among institutionalized aged. *J Gerontol*. 1991;46:15-21.
14. Parmelee PA, Harralson TL, Smith LA, Schumacher HR. Necessary and discretionary activities in knee osteoarthritis: do they mediate the pain-depression relationship? *Pain Med*. 2007;8:449-461.
15. Geerlings SW, Twisk JWR, Beekman ATF, Deeg DJH, van Tilburg W. Longitudinal relationship between pain and depression in older adults: sex, age and physical disability. *Soc Psychiatry Psychiatr Epidemiol*. 2002;37:23-30.
16. Sharma L, Cahue S, Song J, Hayes K, Pai Y-C, Dunlop D. Physical functioning over three years in knee osteoarthritis: role of psychosocial, local mechanical, and neuromuscular factors. *Arthritis Rheum*. 2003;48:3359-3370.
17. Lorig K, Holman HR. Long-term outcomes of an arthritis self-management study: effects of reinforcement efforts. *Soc Sci Med*. 1989;29:221-224.
18. Fu D, Fu H, McGowan P, et al. Implementation and quantitative evaluation of chronic disease self-management programme in Shanghai, China: randomized controlled trial. *Bull World Health Organ*. 2003;81:174-182.
19. Williamson GM, Schulz R. Pain, activity restriction, and symptoms of depression among community-residing elderly adults. *J Gerontol*. 1992;47:367-372.
20. Yesavage JA, Brink TL, Rose TL, et al. Development and validation of a geriatric depression screening scale: a preliminary report. *J Psychiatr Res*. 1982;17:37-49.
21. Chau J, Martin CR, Thompson DR, Chang AM, Woo J. Factor structure of the Chinese version of the Geriatric Depression Scale. *Psychol Health Med*. 2006;11:48-59.
22. Bellamy N, Buchanan WW, Goldsmith CH, Campbell J, Stitt LW. Validation study of WOMAC: a health status instrument for measuring clinically important patient relevant outcomes to antirheumatic drug therapy in patients with osteoarthritis of the hip or knee. *J Rheumatol*. 1988;15:1833-1840.
23. Lorig K, Chastain RL, Ung E, Shoor S, Holman HR. Development and evaluation of a scale to measure perceived self-efficacy in people with arthritis. *Arthritis Rheum*. 1989;32:37-44.
24. Griffiths DA. Interval estimation for the three-parameter lognormal distribution via the likelihood function. *Appl Stat*. 1980;29:58-68.
25. StataCorp. Stata Statistical Software: Release 11. College Station, TX: StataCorp LP; 2009.
26. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *J Pers Soc Psychol*. 1986;51:1173-1182.
27. Preacher K, Hayes A. SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behav Res Methods*. 2004;36:717-731.
28. Sobel ME. Asymptotic confidence intervals for indirect effects in structural equation models. *Sociol Methodol*. 1982;13:290-312.
29. UCLA: Academic Technology Services Statistical Consulting Group. How to perform Sobel-Goodman mediation tests in Stata? http://www.ats.ucla.edu/stat/stata/faq/sgmediation.htm. Accessed October 24, 2011.
30. Shrout PE, Bolger N. Mediation in experimental and nonexperimental studies: new procedures and recommendations. *Psychol Methods*. 2002;7:422-445.