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Are Psychological Factors Associated with Pain Worsening in Individuals with Knee Osteoarthritis? A Systematic Review

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Abstract

Objective: While some reviews have addressed prognostic factors for knee osteoarthritis, relatively few have addressed the worsening of pain with knee osteoarthritis. Moreover, prior reviews have focused on biological and clinical factors and have neglected psychological factors, which have been shown to be associated with pain in other conditions. The aim of this systematic review was to explore the relationship of psychological factors with pain worsening in people with knee osteoarthritis.

Methods: Articles were selected following a comprehensive search of PubMed, the Cochrane Central Register of Controlled Trials, and the Cumulative Index to Nursing and Allied Health Literature from database inception to June 2015. Assessment of the risk of bias was performed using the Risk of Bias Assessment Tool for Nonrandomized Studies. The level of evidence was determined based on the National Health and Medical Research Council of Australia diagnostic levels of evidence.

Results: Twenty articles met the inclusion criteria. Depression, pain catastrophizing, and self-efficacy have been addressed by relatively numerous studies and displayed a significantly positive relationship with pain intensity. However, most studies were cross-sectional studies, and there were only three cohort studies.

Conclusion: There is currently insufficient evidence to support a significant relationship between psychological factors and pain worsening in patients with osteoarthritis of the knee.

Keywords: Knee osteoarthritis; Rheumatoid arthritis; Risk of bias; Arthritis

Introduction

Osteoarthritis (OA) is a joint disorder characterised by progressive degeneration of the articular cartilage resulting in the loss of joint space and a reduction in the formation of new marginal and central bone [1]. Among US adults age 60 years and older, the prevalence of radiographic knee OA and symptomatic radiographic knee OA was 37.4% and 12.1%, respectively [2]. People with symptomatic OA chiefly complain of pain and functional limitation [1,3]. Generally, chronic pain decreases one's quality of life (QOL) [4]. Chronic pain prevention seems essential to avoid the decrease of QOL. Previous research has linked biomechanical [5], demographic [6], and physiological [7] factors to the progression of symptomatic OA. However, no reviews have explored the causal relationship between psychological factors and pain progression in people with knee OA. Psychological factors should also be considered with biological factors as potential factors prognostic for pain, because biopsychosocial perspective is now widely accepted as the most heuristic approach to chronic pain [8]. This relationship has been explored in the context of lower back pain, another chronic musculoskeletal condition [9,10]. In knee OA, there is limited knowledge as to which psychological factors can predict pain worsening. The aim of this systematic review was to explore the causal relationship of psychological factors with pain worsening in people with knee OA.

Methods

Identification and selection of trials

The following electronic databases were searched from the earliest date available until 23 June 2015: PubMed, the Cochrane Library, and the Cumulative Index to Nursing and Allied Health Literature (CINHAL). The search strategy is included in Table 1. The terms used in our search were referred from systematic reviews that investigated psychological factors for chronicity on low back pain [9,10]. Screening of articles using inclusion/exclusion criteria was completed independently by two reviewers (RT and KM), initially focusing on titles and abstracts and then on full-text versions of retrieved articles. Consensus on the final inclusion of articles was reached following discussion; a third reviewer (NK) was available to resolve any outstanding disagreements.

Inclusion criteria

Studies were included if (1) study participants were adults (aged ≥18 years, any sex, any duration of symptoms) with knee OA classified according to clinical or radiographic reference standards (e.g., American College of Rheumatology [ACR] guidelines); (2) outcomes were psychological factors that were measured using questionnaires; (3) study type was non-randomized, such as cohort, case-control, cross-sectional, or case series because these study designs were considered to evaluate evidence level in the National Health and Medical Research Council (NHMRC) evidence hierarchy [11] (Table 2); (4) statistical analysis was performed for the relationship between psychological factors and pain intensity; and (5) the paper was written in English.

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PubMed and the Cochrane library	
#1	Knee osteoarthritis[mesh]
#2	Pain
#3	Adaptation, Psychological[MeSH Terms]
#4	Catastrophization[MeSH Terms]
#5	Fear[MeSH Terms]
#6	Depression[MeSH Terms]
#7	Anxiety[MeSH Terms]
#8	Affect[MeSH Terms]
#9	Somatoform disorders[mesh]
#10	Distress[Title/Abstract]
#11	#1 AND #2 AND (#3 OR #4 OR #5 OR #6 OR #7 OR #8 OR #9 OR #10)
CINAHL	
#1	MH "Osteoarthritis, Knee"
#2	Pain
#3	MH "Adaptation, Psychological"
#4	MH "Catastrophic Illness"
#5	MH "Fear"
#6	MH "Depression"
#7	MH "Anxiety"
#8	MH "Affect"
#9	MH "Somatoform Disorders"
#10	Distress
#11	#1 AND #2 AND (#3 OR #4 OR #5 OR #6 OR #7 OR #8 OR #9 OR #10)

Table 1: Search strategy.

Level	Reseach design
I	A systematic review of level II studies
II	A prospective cohort study
III-1	All or none
III-2	A retrospective cohort study
III-3	A case-control study
IV	A cross-sectional study or case series

Table 2: The National Health and Medical Research Council of Australia diagnostic levels of evidence (NHMRC).

Exclusion criteria

Studies were excluded if (1) study participants had comorbidities such as rheumatoid arthritis, cancer, osteoporosis, or joint infection, or had previous surgery for knee OA; (2) study type was a randomized controlled trial or well-designed quasi-experimental study; (3) duplicate reports from the same study reporting the same data were used; or (4) the study reported exclusively on radiographic variables or laboratory tests with no reference to patient presentation.

Data extraction

Study details and results were extracted by one reviewer from included articles. Results included correlation coefficients, regression coefficients, path coefficients, odds ratios, or P values. Adjusted results were extracted where possible to address the problem of confounding variables [12]. Sources of funding were not noted.

Assessment of the risk of bias

Assessment of the risk of bias was performed using the Risk of Bias Assessment Tool for Nonrandomized Studies (RoBANS) [13]. RoBANS contains six domains: the selection of participants, confounding variables, the measurement of exposure, the blinding of the outcome assessments, incomplete outcome data, and selective outcome reporting (Table 3). RoBANS shows moderate reliability, promising feasibility, and validity [13].

Judge of evidence level

The level of evidence was determined based on the NHMRC evidence hierarchy for aetiology that refers the philosophy or study of causation.

Statistical analyses for synthesis of results

When studies report data as correlations, correlation coefficients per se were synthesized to calculate the square summary correlation. The summary correction was calculated using both fixed-effect and random-effect models. Sample size and correlations in each study were used to perform a meta-analysis using Comprehensive Meta-Analysis Version 2.2.064 software (Biostat, Englewood, USA).

Results

Search results

The combined database search yielded 211 trials. After adjusting for duplicates, 191 trials were considered. Finally, 20 studies fulfilled the inclusion criteria. Consensus was achieved in all cases, thus the third reviewer was not required.

Study details

Details of included studies are available in Table 4. The ACR guidelines were the most frequently used set of guidelines to define OA. Sample sizes ranged from 54 to 1187, studies reported between 44% and 83% women participants, and the mean age ranged from 52.7 to 76.9 years. Most research designs were cross-sectional studies, and there were only three cohort studies

Assessment of the risk of bias

The quality of the included studies is summarized in Figure 1. In appraising the risk of bias as a result of inappropriate participant selection, only three studies were evaluated as 'low risk of bias' on comparability as participants were recruited continuously and prospective data were collected. In confounding variable, nine of all studies were considered to have a 'high risk of bias' because only a univariate analysis was performed. All studies were considered to not have 'low risk of bias' regarding the measurement of exposure, blinding outcome assessment, and incomplete outcome data. Selection of participants could not be assessed because the study protocols could not be obtained.

Judge of evidence level

Table 5 shows a summary of the evidence for the psychological variables related to pain. Psychological variables not examined by more than one included study were not judged for evidence level and are not shown in Table 5. One of the psychological factors assessed by studies in our review was depression. Of eleven studies [14-24], six at least showed a significant relationship between depression and pain, but these findings corresponded to evidence level IV because they were confirmed by cross-sectional studies; the results of the two cohort studies were not consistent. Pain catastrophizing had been measured by ten studies [14,15,19,20,23,25-29] in our review and was shown to be related with pain worsening in six cross-sectional studies as evidence level IV and in one prospective cohort study as evidence level II. Self-efficacy was significantly related to pain worsening in four studies [16,25,27,30], and one study suggesting this finding

Domain	Details	Risk of bias
Selection of participants	Selection bias caused by the inadequate selection of participants	• Low • High • Unclear
Confounding variables	Selection bias caused by the inadequate confirmation and consideration of confounding variable	• Low • High • Unclear
Measurement of exposure	Performance bias caused by the inadequate measurement of exposure	• Low • High • Unclear
Blinding of outcome assessments	Detection bias caused by the inadequate blinding of outcome assessments	• Low • High • Unclear
Incomplete outcome data	Attrition bias caused by the inadequate handling of incomplete outcome data	• Low • High • Unclear
Selective outcome reporting	Reporting bias caused by the selective reporting of outcomes	• Low • High • Unclear

Table 3: Risk of Bias Assessment Tool for Nonrandomized Studies domains (ROBANS).

First author, year	Definition OA for inclusion	Number of patients	Women, %	Age, years	Followup, months	Study design
Conaghan et al. [24]	ACR criteria	1187	68	67.8 ± 9.4	0	Cross-sectional
Finan et al. [14]	ACR criteria	113	66.7	61.1 ± 8.9	0	Cross-sectional
Goodin et al. [15]	ACR criteria	140	74	56.7 ± 7.2	0	Cross-sectional
Keefe et al. [20]	Diagnosed by a rheumatologist	168	57.1	61.1 ± 10.6	0	Cross-sectional
Lim et al. [60]	ACR criteria	90	75.6	70.1 ± 7	0	Cross-sectional
Maly et al. [30]	ACR criteria	54	59.3	68.3 ± 8.7	0	Cross-sectional
Marks [16]	Clinically and radiographically criteria	100	82	69.9 ± 1	0	Cross-sectional
Ozcakir et al. [22]	ACR criteria	100	83	59.5 ± 0.9	0	Cross-sectional
Ozcetin et al. [31]	ACR criteria	81	44.4	52.7 ± 8.6	0	Cross-sectional
Parmelee et al. [17]	Medical record review or direct physician communication	293	65.9	68.5 ± 9.6	12	Prospective cohort
Parmelee et al. [18]	Physician-diagnosed OA of the knee	292	65.8	68.4 ± 9.6	12	Prospective cohort
Pollard et al. [61]	X-rays using KL classification	276	56.7†	69.6 ± 6†	0	Cross-sectional
Rayahin et al. [27]	X-rays using KL classification	212	76.9	64.6 ± 10.1	24	Prospective cohort
Shelby et al. [25]	ACR criteria	192	79	57 ± 10	0	Cross-sectional
Sinikallio et al. [28]	X-rays using KL classification	111	69	63.6 ± 7.2	0	Cross-sectional
Somers et al. [29]	(Not reported in detail)	43	72	57.0 ± 10.0	0	Cross-sectional
Somers et al. [26]	ACR criteria	106	77	58.7 ± 9.2	0	Cross-sectional
Wade et al. [19]	(Not reported in detail)	310	68.7	63.5 ± 9.8	0	Cross-sectional
Wideman et al. [23]	ACR criteria	117	70.1	60.78 ± 10.05	0	Cross-sectional
Wolfe [21]	ACR criteria	564	76.5§	67.8 ± 11.5§	0	Cross-sectional

†: Hip OA or knee OA sample data (n = 763)
§: Including data of hip OA patients (n = 81)

Table 4: Details of included studies.

corresponded to evidence level II. Anxiety was investigated by five studies [14,18,19,28,31], one of these studies was of a prospective cohort design and showed no causal relationship with pain worsening. Three studies [19,26,28] examined the relationship with fear and pain worsening, but statistical significance was not confirmed.

Synthesis of data

Summary correlations of each psychological factor are shown in Figure 2. The data for fear were not synthesized because of a lack of statistical details. There was a significant summary correlation of 0.325 in the fixed-effect model and 0.320 in the random-effect model between depression and knee pain, suggesting that depression only explains about 10% of the pain. The explanatory power of pain catastrophizing and self-efficacy were also similar to that of depression. For anxiety, summary correlations were 0.432 in the fixed-effect model and 0.433 in the random-effect model, thus anxiety explained almost 19% of the pain.

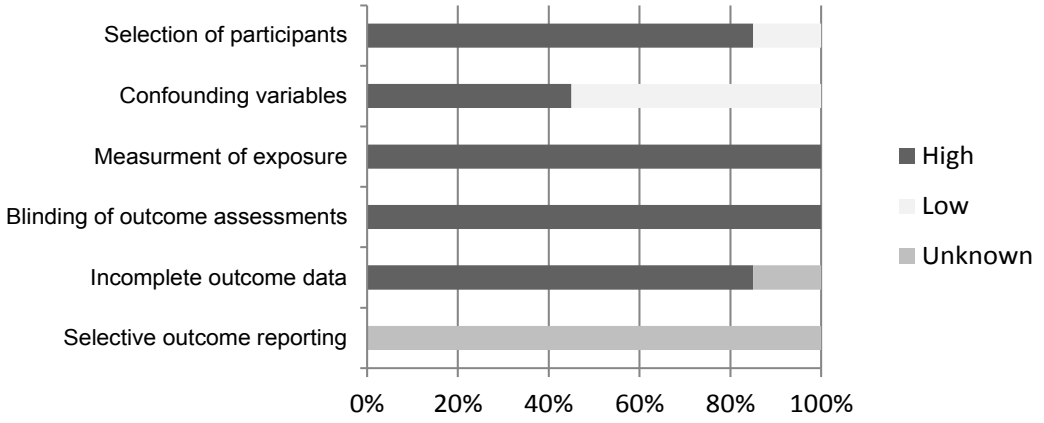
Discussion

Summary of evidence

The current results show that various psychological factors related to pain intensity have been examined. Of these, depression, pain catastrophizing, and self-efficacy have been addressed by numerous studies and displayed a significant positive relationship with pain intensity. However, most studies were cross-sectional studies, and there were only three cohort studies.

Depression

Depression is a mood disorder that causes a persistent feeling



	Selection of participants	Confounding variables	Measurment of exposure	Blinding of outcome assessments	Incompplete outcome data	Selection of participants
Conaghan, 2014	×[a)]	O	×[d)]	×[d)]	Δ [h)]	Δ [i)]
Finan, 2013	×[b)]	×[c)]	×[d)]	×[d)]	×[e)]	Δ [i)]
Goodin, 2013	×[b)]	O	×[d)]	×[d)]	×[e)]	Δ [i)]
Keefe, 2000	×[b)]	O	×[d)]	×[d)]	×[e)]	Δ [i)]
Lim, 2012	×[b)]	×[c)]	×[d)]	×[d)]	×[e)]	Δ [i)]
Maly, 2006	×[b)]	×[c)]	×[d)]	×[d)]	×[e)]	Δ [i)]
Marks, 2007	×[b)]	×[c)]	×[d)]	×[d)]	×[e)]	Δ [i)]
Ozcakir, 2011	×[b)]	×[c)]	×[d)]	×[d)]	×[e)]	Δ [i)]
Ozcetin, 2007	×[b)]	×[c)]	×[d)]	×[d)]	×[e)]	Δ [i)]
Parmelee, 2007	O	O	×[d)]	×[d)]	×[f)]	Δ [i)]
Parmelee, 2013	O	O	×[d)]	×[d)]	Δ [g)]	Δ [i)]
Pollard, 2012	×[b)]	×[c)]	×[d)]	×[d)]	×[e)]	Δ [i)]
Rayahin, 2014	O	O	×[d)]	×[d)]	Δ [g)]	Δ [i)]
Shelby, 2008	×[b)]	O	×[d)]	×[d)]	×[e)]	Δ [i)]
Sinikallio, 2014	×[b)]	O	×[d)]	×[d)]	×[e)]	Δ [i)]
Somers, 2008	×[b)]	×[c)]	×[d)]	×[d)]	×[e)]	Δ [i)]
Somers, 2009	×[b)]	O	×[d)]	×[d)]	×[e)]	Δ [i)]
Wade, 2011	×[b)]	×[c)]	×[d)]	×[d)]	×[e)]	Δ [i)]
Wideman, 2014	×[b)]	O	×[d)]	×[d)]	×[e)]	Δ [i)]
Wolfe, 1999	×[b)]	O	×[d)]	×[d)]	×[e)]	Δ [i)]

O : low risk of bias, ×: high risk of bias, Δ : unclear.

a) Two groups to be compared are not comparable population groups since they differ regarding year since OA diagnosis and clinical diagnosis of OA.

b) There was no group to be compared.

c) Univariate analysis only was performed.

d) Data was obtained by self-reported method.

e) There was no group to be compared for incomplete outcome data.

f) The difference in baseline was observed between completers and dropouts.

g) The dropouts were observed, but no comparison was performed at baseline between completers and dropouts.

h) Participants were not followed longitudinally.

i) Study protocol of original article could not be obtained.

Figure 1: Results of the bias assessment.

of sadness and loss of interest. Also called major depression, major depressive disorder, or clinical depression, it affects how one feels, thinks, and behaves and can lead to a variety of emotional and physical problems. According to the Diagnostic and Statistical Manual of Mental Disorders-IV, diagnosis is confirmed if at least five of the symptoms have been present during the same 2-week period and demonstrate a change from previous functioning. An integral symptom for diagnosis is either a depressed mood or loss of interest or pleasure. These symptoms can be rated by using certain questionnaires. Past studies using questionnaires have tried to establish whether the degree of depressive symptoms was prognostic of future pain in cases such as patients with early inflammatory arthritis, patients following total knee replacement, or post-menopausal women with recurrent pain conditions [32-34]. Additionally, one cohort study that was excluded from our review because it included patients without knee OA, showed that baseline depressive symptoms were the most consistent psychological predictor of annual pain worsening [35]. However, in a 2-year cohort study of lower extremity trauma patients, depression did not predict pain over any period [36]. Moreover, in the current review, there was one study, which design was prospective cohort

Variable	Author	Outcom Measure	Pain scale	Methods of statistical analysis	Statistical value	Dependent Variable †	p Value	NHMRC level of evidence
Depression	Finan et al. [14]	CES-D	Pressure-pain threshold, quadriceps	Correlation analysis	r = -0.3a		p < 0.01	IV
	Goodin et al. [15]	CES-D	WOMAC	Correlation analysis	r = 0.42		p < 0.01	IV
	Marks et al. [16]	CES-D	VAS	Correlation analysis	r = 0.24		p < 0.05	IV
	Parmelee et al. [17]	CES-D	PGC	Correlation analysis	r = 0.248(adjusted)		p < 0.001	II
	Wade et al. [19]	VAS	VAS	Correlation analysis	r = 0.41		(Not reported)	IV
	Keefe et al. [20]	SCL-90R	AIMS	SEM	0.1 (path coefficients)b	Pain-related outcomes	NS	IV
	Wolfe [21]	AIMS	WOMAC	Multiple linear regression	β = 0.16		p < 0.01	IV
	Ozcakir et al. [22]	BDI	WOMAC	Correlation analysis	r = 0.456		p < 0.01	IV
	Wideman et al. [23]	POMS Dep	WOMAC	Correlation analysis	r = 0.124		NS	IV
	Conaghan et al. [24]	(Current or prior depression)	Question 5 of the BPI	Multivariate logistic regression	OR = 1.89 [95%CI 1.41 - 2.54]	IPR or non-IPR	(Not reported)	IV
	Parmelee et al. [18]	A modified SADS	PGC	Multiple linear regression	β = 0.032	Pain	NS	II
Pain catastrophizing	Keefe et al. [20]	CSQ	AIMS	SEM	0.62 (path coefficients)b	Pain-related outcomes	p < 0.05	IV
	Shelby et al. [25]	CSQ	AIMS	Multiple linear regression	β = 0.13	Pain	NS	IV
	Somers et al. [26]	CSQ	AIMS	Multiple linear regression	β = 0.35	Pain	p < 0.01	IV
	Somers et al. [29]	CSQ	VAS	Correlation analysis	r = 0.47		p < 0.001	IV
	Goodin et al. [15]	CSQ	WOMAC	Multiple linear regression	β = 0.40	Pain	p < 0.01	IV
	Wade et al. [19]	PCS	VAS	Correlation analysis	r = 0.4		(Not reported)	IV
	Finan et al. [14]	PCS	Pressure-pain threshold, quadriceps	Correlation analysis	r = -0.19		NS	IV
	Rayahin et al. [27]	PCS	Pain experience stages	Multivariate logistic regression	OR = 0.88 [95% CI 0.83 –0.94]	Good pain outcome or poor pain outcome	(Not reported)	II
	Wideman et al. [23]	PCS	WOMAC	Multiple linear regression	β = 0.196	Pain	p < 0.05	IV
	Sinikallio et al. [28]	PCS	WOMAC	Multivariate logistic regression	OR = 1.03 [95% CI 0.99 -1.08]	Pain (61.4 -100) or not	NS	IV
Self-Efficacy	Rayahin et al. [27]	ASES	Pain experience stages	Multivariate logistic regression	OR = 1.14 [95%] CI 1.04 –1.24]	Good pain outcome or poor pain outcome	(Not reported)	II
	Shelby et al. [25]	ASES	AIMS	Multiple linear regression	β=-0.20c	Pain	p < 0.05	IV
	Maly et al. [30]	ASES	WOMAC	Correlation analysis	r = -0.44		p < 0.01	IV
	Marks [16]	Lorig et al. (1989)	VAS	Correlation analysis	r = -0.286		p < 0.01	IV
	Sinikallio et al. [28]	PSEQ	WOMAC	Multivariate logistic regression	OR = 1.00 [95% CI 0.96 -1.06]	Pain (61.4 -100) or not	NS	IV
Anxiety	Wade et al. [19]	VAS	VAS	Correlation analysis	r = 0.45		(Not reported)	IV
	Finan et al. [14]	STAI	Pressure-pain threshold, quadriceps	Correlation analysis	r = -0.26		p < 0.01	IV
	Parmelee et al. [18]	A modified SADS	PGC	Linear regression	β = -0.009	Pain	NS	II
	Ozcetin et al. [31]	BAI	SF-36	Correlation analysis	r = -0.575		p < 0.001	IV
	Sinikallio et al. [28]	BAI	WOMAC	Multivariate logistic regression	OR = 1.03 [95% CI 0.96 -1.12]	Pain (61.4 -100) or not	NS	IV
Fear	Wade et al. [19]	VAS	VAS	Correlation analysis	r = 0.39		(Not reported)	IV
	Somers et al. [26]	TSK	AIMS	Linear regression	β = 0.05	Pain	NS	IV
	Sinikallio et al. [28]	TSK	WOMAC	Multivariate logistic regression	OR = 0.99[95% CI 0.94 -1.04]	Pain (61.4 -100) or not	NS	IV

AIMS: Arthritis Impact Measurement Scale; ASES: Arthritis Self-Efficacy Scale; BAI: Beck Anxiety Inventory; BDI: Beck Depression Inventory; BPI: Brief Pain Inventory; CES-D: Center for Epidemiologic Studies Depression scale; CI: Confidential Interval; CSQ: Coping Strategies Questionnaire; IPR: Inadequate Pain Relief; OR: Odds ratio; PCS: Pain Catastrophizing Scale; PGC: Philadelphia Geriatric Center Morale Scale; POMS Dep: Profile of Mood States - Depression subscale; SADS: Schedule for Affective Disorders and Schizophrenia; SCL-90R: Symptom Checklist-90-Revised; STAI: State-Trait Anxiety Inventory; TSK: Tampa Scale of Kinesiophobia;VAS: Visual Analogue Scale; WOMAC: Western Ontario and McMaster osteoarthritis index

†: In case of regression analysis, path analysis, or structural equation modelling

a: Value was adapted from the data of pressure-pain threshold (quadriceps) in original paper

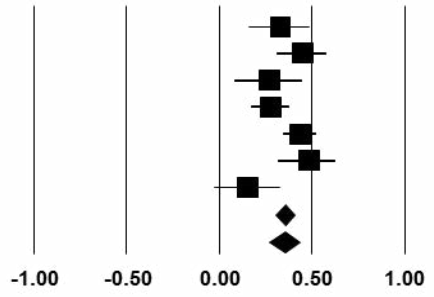
b: Value was adapted from the data of model B in original paper

c: Value was adapted from the data of self-efficacy for pain control in original paper

Table 5: Summary of the evidence for the psychological variables related to pain.

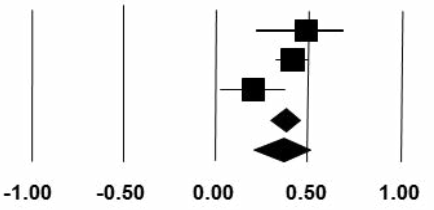
Depression

Model	Study name	Correlation	Lower limit	Upper limit	Z-Value	p-Value	Total
	Finan, 2013	0.300	0.122	0.459	3.246	0.001	113
	Goodin, 2013	0.420	0.273	0.548	5.240	0.000	140
	Marks, 2007	0.240	0.046	0.417	2.411	0.016	100
	Parmelee, 2007	0.248	0.137	0.353	4.313	0.000	293
	Wade, 2011	0.410	0.313	0.499	7.633	0.000	310
	Ozcakir, 2011	0.456	0.285	0.599	4.848	0.000	100
	Wideman, 2014	0.124	-0.059	0.299	1.331	0.183	117
Fixed		0.325	0.272	0.375	11.439	0.000	
Random		0.320	0.232	0.403	6.815	0.000	



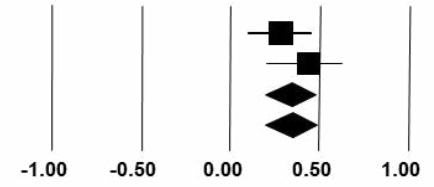
Pain catastrophizing

Model	Study name	Correlation	Lower limit	Upper limit	Z-Value	p-Value	Total
	Somers, 2008	0.470	0.198	0.675	3.226	0.001	43
	Wade, 2011	0.400	0.302	0.490	7.423	0.000	310
	Finan, 2013	0.190	0.005	0.362	2.017	0.044	113
Fixed		0.359	0.276	0.436	8.028	0.000	
Random		0.347	0.185	0.491	4.065	0.000	



Self-efficacy

Model	Study name	Correlation	Lower limit	Upper limit	Z-Value	p-Value	Total
	Marks, 2007	0.286	0.095	0.457	2.898	0.004	100
	Maly, 2006	0.440	0.195	0.633	3.372	0.001	54
Fixed		0.341	0.192	0.475	4.325	0.000	
Random		0.343	0.188	0.481	4.196	0.000	



Anxiety

Model	Study name	Correlation	Lower limit	Upper limit	Z-Value	p-Value	Total
	Wade, 2011	0.450	0.356	0.535	8.493	0.000	310
	Finan, 2013	0.260	0.079	0.424	2.791	0.005	113
	Ozcetin, 2007	0.575	0.408	0.705	5.784	0.000	81
Fixed		0.432	0.358	0.501	10.300	0.000	
Random		0.433	0.269	0.572	4.834	0.000	

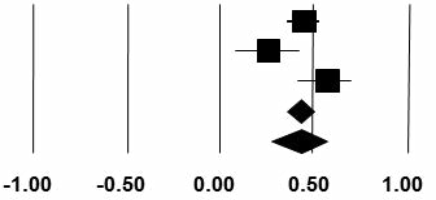


Figure 2: Results of data synthesis (meta-analysis).

study, reporting that depressive symptoms did not significantly predict a worsening of pain [18]. The explanatory power of depression for pain was not large. Thus, depressive symptoms may be a limited prognostic factor for pain with knee OA.

Pain catastrophizing

Pain catastrophizing refers to the tendency to focus on and magnify pain sensations and to feel helpless when experiencing pain [37]. It has been reported that biopsychosocial variables such as catastrophizing play an important role in reported pain severity [38-40]. Studies in which functional magnetic resonance imaging was used showed that pain catastrophizing, independent of the influence of depression, was significantly associated with increased activity in brain areas related to anticipation of pain, attention to pain, and emotional aspects of pain [41]. Recent advances in cognitive-behavioural pain theory and research suggest that the way an individual thinks about and interprets pain may be important in explaining adjustment to pain [42,43]. In the current review, six of ten studies showed a significant positive relationship between pain catastrophizing and pain intensity. One of scales used by most studies was the coping strategies questionnaire [44], for which reliability [45,46] and validity [47,48] have been confirmed. Reliability and validity of another scale, pain catastrophizing scale, has also been confirmed [49]. However, of the ten studies, there was only one cohort study included in our review that reported a causal relationship between pain catastrophizing and pain worsening. Moreover, similar to depression, the explanatory power of pain catastrophizing for pain was not large. Consequently, it cannot be immediately claimed from results of our review that pain catastrophizing can cause pain worsening with knee OA. Nonetheless, these data support additional studies addressing pain catastrophizing as a potential causal agent for future pain.

Self-efficacy

Self-efficacy is commonly defined as the belief in one's capabilities to achieve a goal or an outcome [50]. In the context of rehabilitation, various types of scales for assessing self-efficacy have been developed, (e.g., pain [51], fall [52], or rehabilitation [53]). The arthritis self-efficacy scale (ASES) was developed for assessing self-efficacy in the context of rehabilitation for treating people with arthritis [54] and has been shown to be reliable and valid [55]. All included studies using ASES showed a statistically positive relationship between self-efficacy and pain intensity. However, there was one cohort study reporting the causal relationship between self-efficacy and knee pain, and thus the explanatory power of self-efficacy for pain was not large. Accordingly, self-efficacy should be also addressed as a potential causal agent for future pain.

Strengths of the study

The strength of the current review is that it focuses on psychological factors that have not been evaluated in past systematic reviews for knee OA. Several systematic reviews have evaluated patient characteristics or physical examination variables, but have not included psychological factors [5-7,56]. Moreover, these reviews focus only on knee OA progression, rather than on pain specially; thus, pain from a biopsychosocial model was overlooked. In contrast, the current review concentrated on the causal agents of pain worsening, thus we were able to broaden our outlook for psychological factors. Although this review did not define predictors for knee OA progression, it was able to identify potential psychological factors that might cause pain worsening in people with knee OA. This would impact future studies examining the aetiology of pain worsening with knee OA.

Limitations of the study

However, our study also has several limitations. The first such limitation was due to the methods adopted in studies included in our review. To confirm a causal relationship of psychological factors with pain worsening, the effect of other factors on pain intensity needs to be controlled during statistical analysis. For example, Amin et al. showed in a 30-month, prospective, natural history study that men with knee OA who smoke had more severe knee pain than men who do not smoke [57]. Moreover, Ledingham et al. [58] reported in a prospective observational study of 350 osteoarthritic knees that increased pain score was associated with calcium pyrophosphate crystals, chondrocalcinosis, and severity of knee OA. Statistical analyses performed by most studies included in our review did not control for the effect of these factors on pain intensity. Such confounding factors need to be adequately addressed to determine the causal relationship of psychological factors with pain worsening.

The second limitation was publication bias, which is a bias with regard to what is likely to be published, among what is available to be published. Not all bias is inherently problematic, but one problematic and much-discussed bias is the tendency of researchers, editors, and pharmaceutical companies to handle the reporting of experimental results that are positive (i.e., reporting a significant finding) differently from results that are negative (i.e., supporting the null hypothesis) or inconclusive, leading to a misleading bias in the overall published literature [59]. Whether publication bias existed in the current results was not examined. The reason for the lesser number of studies showing no significant relationship between psychological factors and pain intensity might be the difficulty in getting studies with negative data published. However, significant relationships between pain intensity and depression, pain catastrophizing, and self-efficacy were supported by multiple studies. Thus, we believe that these candidate factors could be potential causal agents for pain worsening.

Conclusions

There is currently insufficient evidence to support a significant relationship between psychological factors and pain worsening in patients with OA of the knee. The low level of evidence in the studies included prevents the authors from making any recommendations regarding guidelines for practice. To help gather more evidence on the relationship of psychological factors to pain intensity in OA of the knee, more prospective cohort studies are needed.

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