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Critical Review

The Role of Self-Efficacy on the Prognosis of Chronic Musculoskeletal Pain: A Systematic Review

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Abstract: **Evidence suggests that self-efficacy can play an essential role as a protective factor as well as a mediator in the relationship between pain and disability in people suffering from chronic musculoskeletal pain. This study systematically reviewed and critically appraised the role of self-efficacy on the prognosis of chronic musculoskeletal pain. Study selection was on the basis of longitudinal studies testing the prognostic value of self-efficacy in chronic musculoskeletal pain. The Newcastle-Ottawa Scale, the Cochrane Collaboration's tool, and the Methodological Index for Non-Randomized Studies checklist were used to evaluate the risk of bias of included studies. A total of 27 articles met the inclusion criteria. Our results suggest that higher self-efficacy levels are associated with greater physical functioning, physical activity participation, health status, work status, satisfaction with the performance, efficacy beliefs, and lower levels of pain intensity, disability, disease activity, depressive symptoms, presence of tender points, fatigue, and presenteeism. Despite the low quality of evidence of included studies, clinicians should be encouraged identify people with chronic musculoskeletal pain who present low self-efficacy levels before prescribing any therapy. It may help clinicians in their clinical decision-making and timely and specific consultations with—or referral to—other health care providers.**

Perspective: ***This article presents promising results about the role of self-efficacy on the prognosis of chronic musculoskeletal pain. However, because of the low quality of evidence of included studies, these findings should be taken with caution, and further research is needed.***

Key words: *Chronic pain, musculoskeletal pain, prognosis, self-efficacy, systematic review.*

Chronic pain is an enormous global health problem. It has been estimated that 1 in 5 adults suffer from pain each year, with 1 in 10 adults developing chronicity.[41] One of the most common forms of chronic pain is chronic musculoskeletal pain (CMP). It is a highly prevalent, disabling, and costly condition, with a substantial socioeconomic burden to individuals, employers, health care systems, and society.[11,57,60,76] The prevalence of CMP ranges from 13.5% to 47% of the general population.[19] People with CMP often show a detrimental effect on their social as well as family environments.[27] This situation is associated with an inability to carry out work, social, recreational, and household tasks,[36,47] negatively affecting their quality of life.[11,73] Despite its worldwide prevalence and the high social and economic weight, a clear understanding of its etiology and pathogenesis remains elusive.

There are several reasons that could explain why there is not a consensus in the treatment of this condition. First, CMP is characterized by the presence of central sensitization,[48,64,81] which makes people with CMP report different levels of pain, although similar radiological or pathological conditions are presented.[55] Second, evidence

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suggests that there is an interplay between central sensitization and the brain regions involved in pain processing.[16,20] Third, a link seems to exist between the alteration of specific regions of the brain such as the corticolimbic circuitry (key system for reward and motivated behavior) and maladaptive behaviors, which could facilitate the perpetuation of symptoms.[15,68] As a result, it seems presumable that chronic pain is not only a mere signal of nociception, but also a biopsychosocial experience. In this context, identification of the tissue damage in a peripheral location is necessary but it is not enough to explain how somatosensory information is transformed into the physiologic, cognitive, affective, and behavioral response, which is labeled as pain.[33]

According to this, evidence suggests that the association between pain and disability is only moderate, with psychological factors playing a crucial role in the processing[33] and modulation of pain[53] and, hence, in the development and maintenance of pain-related problems.[15,77] The fear-avoidance model[45,82,83] highlights the importance of beliefs in the development and perpetuation of chronic pain and disability. This model suggests that catastrophic thoughts lead to an increase in negative beliefs about pain (kinesiophobia, pain-related fear, pain-related anxiety, and hypervigilance), which increases the feeling of threat to an actual and/or a previous painful experience.[84] It gives rise to an intense feeling of avoidance and escape, followed in the long-term by disuse of the affected area, depression, and disability.[45,84] It causes a decrease in rehabilitation efforts, which interferes directly in the patients' recovery, and preserves their negative pain experience.[84] The role played by the psychological factors included in the fear-avoidance model have been analyzed in several systematic reviews.[51,87,88] Despite the findings that propose that people with CMP are prone to have greater levels of catastrophic thoughts, which cause the appearance of negative beliefs such as fear of movement or pain-related fear, certain individuals may achieve higher levels of self-control in everyday situations that minimize the negative effects of these beliefs (fear and avoidance) in their lives.[43] One of the positive beliefs associated with a higher level of self-control is self-efficacy (SE).

SE has a broad definition. Bandura's social cognitive theory refers to SE as the personal confidence to carry out an activity with the aim of successfully achieving a desired outcome.[6] SE is the central motor to developing human motivation, psychosocial well-being, and personal achievement.[6] In the context of pain chronicity and disability, a patient with CMP may have high levels of pain intensity, but they may also show greater self-confidence (eg, high SE) to manage their musculoskeletal pain, and thus, they can successfully execute certain tasks (eg, job, sports, social relationships), attaining the desired result despite the pain.[71]

High SE includes self-confidence, accurate self-evaluation, willingness to take risks, and sense of accomplishment. However, low SE encompasses fear of risks and uncertainty, low aspirations, feelings of fear of failure, and impression management.[58] The existing evidence suggests a potential protective association between SE and disability,[25] pain extent,[30] pain interference,[31] physical and psychological functioning,[31] and quality of life.[2] Moreover, SE seems to play a role as a mediator in the relationship between pain and disability,[21] depression and pain severity,[69] pain-related fear, and pain and disability.[89] A recent review[43] evaluated the role of SE in chronic pain conditions. After analyzing 86 studies, the findings showed SE to be a protective factor related to chronicity (impairment, psychological distress, and pain severity). However, despite these promising results, the prognostic value of SE in CMP is still unclear, because of the cross-sectional nature of most of these studies. Acquiring more information about which factors influence the prognosis of CMP, and how these factors affect chronicity is becoming more important in the treatment of this condition.[22] Knowing and understanding the accurate prognostic value of SE and its association with treatment effects (moderator/mediator role) may help to steer specific treatments. Hence, the aim of this study was to answer the following PECOS (P, participant; E, exposure; C, comparator; O, outcome; S, study design) question through a systematic review of the literature of longitudinal studies (S): How SE (E) influences the prognosis (O) of people suffering from CMP (P), compared with people free of CMP (C)?

Methods

The review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement,[46] and following the Assessing the Methodological Quality of Systematic Reviews (AMSTAR) criteria.[67] The systematic review protocol was registered at the International Prospective Register of Systematic Reviews (PROSPERO: CRD42016042643).

Data Sources and Search Strategy

Two independent investigators (J.M.-C. and C.Z.-C.) searched the following electronic databases from inception to November 2016 using optimized search strategies: MedLine, AMED, CINAHL, PsycINFO, and PubPsych. A manual search of relevant eligible studies was also searched through cross-references identified in the reference lists within original as well as review articles, selecting studies missed by the electronic search. A sensitive search strategy using relevant search terms that were developed from Medical Subject Headings (MeSH), and key words generated from the subject headings, were used: "self-efficacy" [MeSH Terms], "prognosis" [MeSH Terms], "chronic pain" [MeSH Terms], "shoulder pain" [MeSH Terms], "neck pain" [MeSH Terms], "low back pain"[MeSH Terms], "fibromyalgia" [MeSH Terms], "fatigue syndrome, chronic" [MeSH Terms], "osteoarthritis" [MeSH Terms], "musculoskeletal pain" [MeSH Terms], "musculoskeletal diseases" [MeSH Terms], biopsychosocial factors, psychological factors, pain beliefs, whiplash-associated disorder, knee pain, ankle pain, epicondylalgia, and musculoskeletal disorders. The complete search strategy report is shown in Appendix S1. The

gray literature was explored to detect any relevant unpublished work. The following gray literature databases were searched: Research Profiles, JBI COnNECT +, NHS Evidence, New York Academy of Medicine Grey Literature Report, PsycEXTRA, Explore the British Library, TRIP database, National Guideline Clearinghouse, Grey Source, and Open Grey. If it was applicable and necessary to gather any other nonpublished data, researchers were contacted directly. References were exported and duplicates were removed using citation management software (Mendeley Desktop version 1.17.4, Elsevier, London).

Eligibility Criteria

The PECOS framework, as aforementioned, was followed to determine which studies were included in the present systematic review. Each study had to meet the following inclusion criteria: 1) longitudinal studies examining the influence of SE as a prognostic factor in people with CMP. When prospective studies only reported findings regarding the association between SE and the outcome measures at baseline, the studies were considered as a cross-sectional and were excluded, 2) studies whose participants were adults diagnosed with CMP, defined in this review as persistent or episodic pain lasting more than 3 months, around the axial skeleton (neck, low back, and/or pelvic) or peripheral joints (shoulder, elbow, wrist, knee, and/or ankle). In this regard, the Analgesic, Anesthetic, and Addiction Clinical Trial Translations, Innovations, Opportunities, and Networks (ACTTION), American Pain Society (APS) Pain Taxonomy (AAPT) for chronic pain was used,[28] including people with diagnoses of chronic myofascial pain, fibromyalgia, chronic widespread pain (eg, chronic fatigue syndrome), rheumatoid arthritis, spondyloarthropathies, and those with a diagnosis of osteoarthritis. Although the AAPT for chronic pain does not consider spinal pain inside of the musculoskeletal pain group, we decided to include people with chronic axial musculoskeletal low back pain in the present review, 3) studies written in English, 4) no restriction was applied on participants' gender, ethnicity, and follow-up duration, 5) studies recruiting participants from any setting (general population, primary care, or secondary care), 6) studies providing outcome data for at least 1 of the following outcome measures: pain, disability, chronicity (nonrecovery of any pain condition), health-related quality of life, physical activity, sick absence, disease activity, psychological variables (eg, depression), fatigue, and mental health, 7) the prognostic factor of interest was SE assessed at baseline and follow-up. The exclusion criteria were as follows: 1) cross-sectional studies, 2) studies in which the sample did not follow the AAPT for CMP, 3) CMP due to trauma, 4) studies evaluating SE before or after surgery, 5) studies exploring SE as a mediator, 6) studies analyzing SE as an outcome measure, 7) studies aiming to investigate psychometric properties of SE assessment measures.

Study Selection

All studies identified by the search strategy were screened using the eligibility criteria that were specified previously. The first stage of assessment involved the screening of titles and abstracts by 2 reviewers (J.M.-C. and C.Z.-C.). The same reviewers undertook the second stage, screening the full text. In cases of disagreement, a decision was made by consensus or, when necessary, a third reviewer (A.L.-S.) was consulted. A short questionnaire was adapted for the present review and was used to guide the selection of relevant studies (Appendix S2).[1]

Data Extraction

Two independent reviewers (J.M.-C. and C.Z.-C.) extracted the following relevant data from each study: study details (first author, year of publication), characteristics of participants, setting, pain condition, SE-measuring instrument, outcome measures, duration of follow-up, and study design. If there was any discrepancy between reviewers, a third reviewer was consulted (A.L.-S.). When necessary, an e-mail was sent to the original authors to provide further information on participants' data.

Quality Assessment

The risk of bias of each study included in this review was assessed through several methodological tools relying on the study design of the included studies. The Newcastle Ottawa scale (NOS)[86] was used to evaluate the quality of cohort studies. The NOS applies a star rating system to judge methodological quality on the basis of 3 subcategories: selection of groups, comparability, and outcome. This checklist is recommended for cohort studies,[90] and has frequently been used by the Cochrane Collaboration (www.cochrane.org). The Cochrane Collaboration's tool was used to evaluate the risk of bias of randomized controlled trials.[39] Six recommended bias domains, including selection bias (random sequence generation and allocation concealment), performance bias (blinding of participants and personnel), detection bias (blinding of outcome assessments), attrition bias (incomplete outcome data), reporting bias (selective reporting), and other biases, were considered, and each domain was rated as low, high, or unclear according to the level of bias. The Methodological Index for Non-Randomized Studies (MINORS) checklist was used for the quality assessment of nonrandomized trials. It consists of 12 items, 8 items for noncomparative studies and 4 additional items for comparative studies. The items are scored on a 3-point scale; 0 (not reported), 1 (reported, but not adequate), or 2 (reported and adequate).[70] These checklists are recommended for cohort studies, randomized controlled trials, and nonrandomized trials, respectively.[90] Two reviewers (J.M.-C. and C.Z.-C.) carried out this process, and when any disagreement was found a third independent reviewer (A.L.-S.) was consulted.

To assess the overall quality and strength of the evidence per outcome the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system was used.[5] In brief, the GRADE classification was downgraded from high quality by 1 level for each factor that we encountered: 1) risk of bias, 2) inconsistency of results, 3) indirectness, 4) imprecision, and 5) other consider-

ations (eg, reporting bias). Two researchers (A.L.-S. and J.M.-C.) judged whether these factors were present for each outcome. The quality of evidence was defined as: 1) high (further research is unlikely to change our confidence in the estimate of effect and there are no known or suspected reporting biases: all domains are fulfilled), 2) moderate (further research is likely to have an important effect on our confidence in the estimate of effect and might change the estimate: 1 of the domains is not fulfilled), 3) low (further research is likely to have an important effect on our confidence in the estimate of effect and is likely to change the estimate: 2 of the domains are not fulfilled), and 4) very low (we are uncertain about the estimate: 3 of the domains are not fulfilled).[37]

Statistical Analysis

For the primary analysis, studies were grouped per outcomes (pain intensity, pain severity, physical functioning, physical activity participation, disability, activity interference, disease activity, efficacy beliefs, depressive symptoms, anxiety, pain behavior, fatigue, health status, presence of tender points, satisfaction with their performance, presenteeism, work status, and number of days for a total compensation benefits). Because of the absence of a nonexposed group (cohort studies) and control group (clinical studies) in most of the studies potentially eligible for conducting a meta-analysis, and the presence of heterogeneity in terms of population, intervention, and outcome measurement, a meta-analysis could not be carried out. Consequently, a descriptive quantitative analysis (the most relevant summary measure with a precision estimate) for each study was provided. For studies that reported results with several degrees of adjustment for confounders in different models, we extracted the estimate from the model that showed the best adjustment. GRADEpro software,[65] and Review Manager (RevMan) version 5.3 (Copenhagen: The Nordic Cochrane Centre, The Cochrane Collaboration, 2014) software were used in the review to process data.

Results

Study Characteristics

A total of 5,158 citations were identified through electronic databases, with 29 additional studies identified through reference screening. One thousand six hundred seventeen titles and abstracts were screened with 164 full-text articles being evaluated. The number of studies retrieved from each database and the number of studies excluded in each screening phase are shown in Fig 1. The full references of excluded studies in the last screening (n = 137) is reported in Appendix S3. The conflict of interests of included studies are shown in Appendix S4. Of these, 27 longitudinal studies (19 cohort studies[8,10,12,17,18,24,26,29,32,35,38,42,44,49,50,56,63,66,80]; 7 randomized controlled trials[3,4,13,34,52,72,79]; 1 nonrandomized trial[85]) with a total of 11,905 participants (chronic low back pain = 5,048; rheumatoid arthritis = 1,390; spondyloarthropathies = 1,253; fibromyalgia = 1,234; chronic shoulder pain = 1,134; knee osteoarthritis = 540; knee pain = 480; ankylosis spondylitis = 169; chronic whiplash associated disorder = 33; mixed = 624) satisfied our inclusion criteria and were included in this review. Duration of follow-up ranged from 2 weeks to 5 years. Eleven studies evaluated patients of community-based populations,[8,12,24,35,44,50,52,63,66,79,80] whereas 19 studies were conducted in clinical care settings (primary care[4,10,17,18,32,38] and secondary care,[3,10,13,18,26,29,34,35,38,49,56,72,85] and tertiary[42]). Definition of outcomes varied across studies with the most common being disability,[10,13,17,18,24,26,29,32,34,38,44,63,66,72,85] pain intensity,[3,4,12,13,18,26,29,34,44,72,79,80,85] physical functioning,[3,35,63,80,85] depressive symptoms,[35,49,79,80] disease activity,[8,13,35] pain severity,[17] health status,[12,35] physical activity participation,[42,52] fatigue,[12] efficacy beliefs,[35] presenteeism,[38] anxiety,[49] pain behavior,[56] activity interference,[4] presence of tender points,[13] satisfaction with their performance,[85] work status,[29] and number of days for the total compensation benefit.[50] The only psychological prognostic factor included in all studies was SE. The characteristics of the included studies are reported in Table 1.

Methodological Quality

The degree to which studies met the quality criteria varied considerably. The methodological quality assessment of all included studies is presented in Fig 2 (for randomized controlled trials) and Table 2 (for cohort studies). Only 1 study showed a nonrandomized design, as a result the quality of the study was presented descriptively.[85] This study was evaluated with MINORS[85] and showed the following score: a clearly stated aim (2); inclusion and consecutive patients (0); prospective collection of data (0); end points appropriate to the aim of the study (2); unbiased assessment of the study end point (0); follow-up period appropriate to the aim of the study (2); loss of follow-up <5% (1); prospective calculation of the study size (0); an adequate control group (0); contemporary groups (0); baseline equivalence of groups (0); and adequate statistical analyses (0).

Level of Evidence of the Role of SE on the Prognosis of CMP

The evidence of the predictive value of SE on the prognosis of CMP is presented in Table 3. A descriptive of the statistical results is reported in Table 4.

SE and Disability

Disability was evaluated by 15 studies.[10,13,17,18,24,26,29,32,34,38,44,63,66,72,85] A total of 11 studies showed how higher SE levels were significantly associated with lower disability levels.[10,13,18,24,26,29,32,38,63,66,85] Interestingly, 1 study only reported a positive and significant relationship between SE and disability post-intervention.[44] However, there was no statistical relationship identified between SE and disability in 4 studies.[17,34,44,72] The overall quality of evidence was very low.

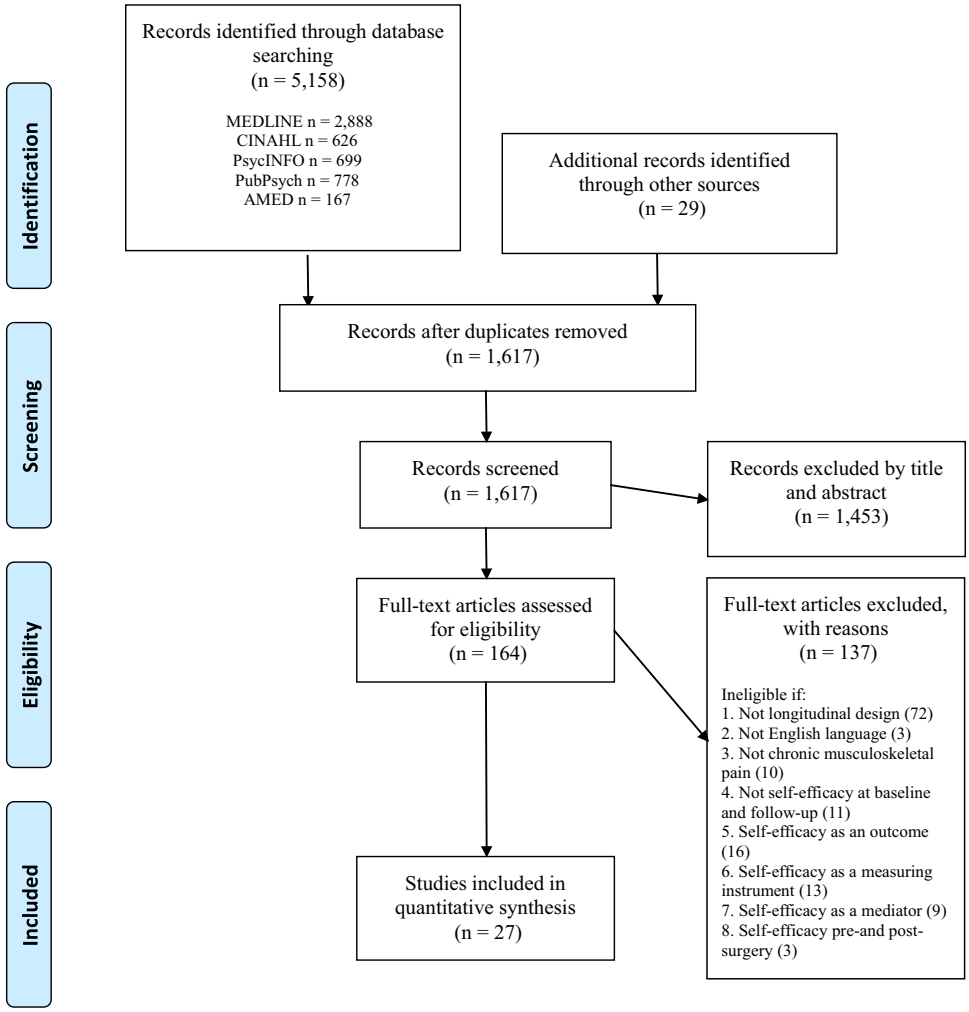

Figure 1. Preferred Reporting Items for Systematic reviews and Meta-Analyses flow diagram of the conducted search.

SE and Pain Intensity

Pain intensity was tested by 13 studies.[3,4,12,13,18,26,29,34,44,72,79,80,85] A total of 9 studies reported that higher SE levels were significantly associated with lower pain intensity levels.[3,12,13,18,26,29,44,79,80] However, there was no statistical relationship identified between SE and pain intensity in 5 studies.[3,4,34,44,72] Moreover, 1 study did not include any data in the predictive model.[24] The overall quality of evidence was very low.

SE and Physical Functioning

Physical functioning was analyzed by 5 studies.[3,35,63,80,85] A total of 5 studies showed that higher SE levels were significantly associated with better physical functioning.[3,35,63,80,85] However, there was no statistical relationship identified between SE and physical functioning in 1 study at discharge.[3] The overall quality of evidence was very low.

SE and Depressive Symptoms

Depressive symptoms were tested by 4 studies.[35,49,79,80] Three studies reported a negative and significant association between SE and depressive symptoms.[35,79,80] However, 1 study showed no statistical relationship between SE and depressive symptoms.[49] The overall quality of evidence was very low.

SE and Disease Activity

Disease activity was explored by 3 studies.[8,13,35] All studies reported a negative and significant association between SE and disease activity.[8,13,35] The overall quality of evidence was very low.

SE and Physical Activity Participation

Physical activity participation was analyzed by 2 studies.[42,52] In 1 study,[52] there was a positive and significant association between SE and physical activity participation. However, the other study[42] did not show a statistical relationship between SE and physical activity participation when variables were included in the multivariable analysis. The overall quality of evidence was very low.

SE and Health Status

Health status was tested by 2 studies.[12,35] Both studies reported a positive and significant association between

Table 1. Characteristics of Included Articles

Reference	Study N	Mean Age, Years	Condition	Setting	SE Measuring Instrument	Outcome Measures	Data Collection	Study Design
Altmaier et al[3]	45	40	CLBP	Clinical care setting (secondary care)	SES	Physical functioning (LBPRS); pain intensity (MPQ): 2 subscales: PPI and PRI	(T1) at baseline; (T2) at discharge; (T3) at 6 months	Longitudinal (randomized controlled trial)
Ang et al[4]	250	55.5	Mixed (back, hip, or knee)	Clinical care setting (primary care)	ASES	Pain intensity and activity interference (GCPS)	(T1) at baseline; (T2) at 3 months	Secondary data analysis (from a randomized controlled trial)
Barlow[8]	169	39.75 (SD 11.16)	Ankylosing spondylitis	Community-based population	Ankylosing Spondylitis Exercise SE Scale	Disease activity (severity) (DSS)	(T1) at baseline; (T2) at 6 months	Longitudinal (cohort study)
Bishop et al[10]	524 (347 completed all follow-ups)	55.0 (SD 15.1)	CLBP	Clinical care setting (primary and secondary care)	Chronic pain SE for pain management subscale	Disability (RMDQ)	(T1) pretreatment; (T2) at 2 weeks; (T3) at 3 months; (T4) at 6 months	Longitudinal (cohort study)
Brekke et al[12]	659 (306 completed the follow-up)	53.3 (SD 11.5)	RA	Community-based population	ASES (PSE, OSE)	Pain intensity and fatigue (VAS); health status (symptoms, pain, vitality; affect; mental health) (AIMS2 and SF-36)	(T1) at baseline; (T2) 5 years	Longitudinal (cohort study)
Buckelew et al[13]	109	44.5 (SD 9.5)	FM	Clinical care setting (secondary care)	ASES	Presence of tender points (tender point index); disease activity (severity) (physician's rating of disease severity); pain intensity (VAS); disability (AIMS)	(T1) at baseline; (T2) after the 6-week intervention	Longitudinal (randomized controlled trial)
Campbell et al[17]	1,591 (488 completed all follow-ups)	47.4 (SD 9.0)	CLBP	Clinical care setting (primary care)	PSEQ	Pain severity and disability (CPG)	(T1) at baseline; (T2) at 6 months; (T3) at 5 years	Longitudinal (cohort study)
Chester et al[18]	1,030 (811 completed all follow-ups)	57 (SD 15)	CSP	Clinical care setting (primary and secondary care)	PSEQ	Pain intensity and disability (SPADI and QuickDASH)	(T1) at baseline; (T2) at 6 weeks; (T3) at 6 months	Longitudinal (cohort study)
Demmelmaier et al[24]	1,024 (379 completed follow-up)	16 to 44	CLBP	Community-based population	SES	Pain intensity and disability (GCPS)	(T1) at baseline; (T2) at 12 months	Longitudinal (cohort study)
Dobkin et al[26]	63 (46 remained)	53.6 (SD 14.5)	FM	Clinical care setting (secondary care)	ASES	Pain intensity (short MPQ); disability (FIQ)	(T1) at baseline; (T2) at the end of treatment; (T3) at 6 months	Secondary data analysis (cohort study)
Engebretsen et al[29]	104 (48 completed all follow-ups)	48 (SD 10.7)	CSP	Clinical care setting (secondary care)	The sum of 4 items obtained from a previous study	Pain intensity and disability (SPADI)	(T1) at baseline; (T2) at 12 months	Secondary data analysis (cohort study)

Table 1. Continued

Reference	Study N	Mean Age, Years	Condition	Setting	SE Measuring Instrument	Outcome Measures	Data Collection	Study Design
Foster et al[32]	1,591 (810 completed follow-up)	43.9 (SD 10.3)	CLBP	Clinical care setting (primary care)	PSEQ	Disability (RMDQ)	(T1) at baseline; (T2) at 6 months	Longitudinal (cohort study)
Gere et al[35]	304 (152 with KOA)	65.8 (SD 10.0)	KOA	Community-based population (advertisement) and clinical care setting (rheumatology clinics)	ASES	Efficacy beliefs (ASES adapted for spouses); disease activity (severity; WOMAC); depressive symptoms (CES-D short form); health status (single item); physical functioning (physical performance; 3 tests: standing balance, gait speed, and chair rises)	(T1) at baseline; (T2) at 6 months; (T3) at 18 months	Longitudinal (cohort)
Haglund et al[38]	1,253	48 (SD 10)	Spondyloarthropathies	Clinical care setting (primary and secondary care)	ASES	Presenteeism (WPAI); disability (WPAI)	(T1) at baseline; (T2) at 2.5 years	Longitudinal (cohort)
Iversen et al[42]	573	61 (SD 12)	RA	Clinical care setting (tertiary care hospital)	ASES	Physical activity participation (meeting ≥150 minutes of moderate intensity or 75 minutes of vigorous physical activity per week (NHSPAQ II)	(T1) at baseline; (T2) at 3 years	Longitudinal (cohort study)
Karlsson et al[44]	57	43 (SD 8.5)	Mixed (chronic neck pain and CSP)	Community-based population	PSEQ; and general SE Scale	Pain intensity (NRS); disability (NDI)	(T1) at baseline; (T2) at 4 to 6 months; (T3) at 1 year	Secondary data analysis (cohort study)
Lötters et al[50]	187	42 (SD 11)	Mixed	Community-based population	Six items adapted from ASES	Cumulative number of calendar days a claimant received total compensation benefits during 1 year	(T1) at baseline; (T2) at 12 months	Longitudinal (cohort study)
Lowe et al[49]	127	Female: 55.2 (SD 11.28); male: 60.0 (SD 12.53)	RA	Clinical care setting (rheumatology clinics)	ASES	Depressive symptoms (HADS-D); anxiety (HADS-A)	(T1) at baseline; (T2) at 8 weeks after intervention	Longitudinal (cohort study)
Gassi Macedo et al[34]	172	49.15 (SD 15); mean values of both groups	CLBP	Clinical care setting (secondary care)	PSEQ	Pain intensity (0–10 scale) and disability (PSFS)	(T1) at baseline; (T2) at 2 months; (T3) at 6 months; (T4) at 12 months	Secondary data analysis (from a randomized controlled trial)

Table 1. Continued

Reference	Study N	Mean Age, Years	Condition	Setting	SE Measuring Instrument	Outcome Measures	Data Collection	Study Design
Mielenz et al[52]	130	71.5 (SD 11.7)	Mixed (RA, KOA, FM)	Community-based population	SEPA and RASE	Physical activity participation (PASE)	(T1) at baseline; (T2) at 3 months	Secondary data analysis (from a randomized controlled trial)
Parker et al[56]	31	61.8 (SD 7.0)	RA	Clinical care setting (hospital)	ASES	Pain behavior (modified pain behavior rating)	(T1) at baseline; (T2) at 6 months	Longitudinal (cohort study)
Rejeski et al[63]	480	71.82 (SD 5.00)	Knee pain	Community based population	SE for stair climb[61]	Physical functioning (stair climb performance, stair climb task); disability (several items provided by a previous study)[62]	(T1) at baseline; (T2) at 15 months; (T3) at 30 months	Longitudinal (cohort study)
Sharma et al[66]	236	68.6 (SD 10.8)	KOA	Community-based population	ASES (function subscale)	Disability (WOMAC physical function scale and rate of chair-stand performance)	(T1) at baseline; (T2) at 3 years	Longitudinal (cohort)
Söderlund et al[72]	33	40.6 (between both groups)	Chronic whiplash-associated disorder	Clinical care setting (secondary care)	SES	Pain intensity (NRS), disability (PDI)	(T1) at baseline; (T2) at post-treatment; (T3) at 3 months	Longitudinal (randomized clinical trial)
Van Liew et al[80]	462	54 (SD 11.1)	FM	Community-based population	ASES	Pain intensity (MPQ), physical functioning (FIQ), depressive symptoms (CES-D)	(T1) at baseline; (T2) at 6 months; (T3) at 1 year	Secondary data analysis (cohort study)
Van Liew et al[79]	600	54 (SD 11)	FM	Community-based population	ASES	Pain intensity (MPQ), depressive symptoms (CES-D)	(T1) at baseline; (T2) at 6 months; (T3) at 1 year	Longitudinal (randomized controlled trial)
Walsh et al[85]	101	46 (SD 24)	CLBP	Clinical care setting (secondary care)	SE questionnaire	Disability (RMDQ), patient-rated performance and satisfaction with their performance (COPM)	(T1) at baseline; (T2) at post-treatment; (T3) at 9 months	Longitudinal (nonrandomized clinical trial)

Abbreviations: CLBP, chronic low back pain; SES, SE Scale; LBPRS, Low Back Pain Rating Scale; MPQ, McGill Pain Questionnaire; PPI, Present Pain Intensity; PRI, Pain Rating Index; T1-T4, time points 1-4; ASES, Arthritis SE Scale; GCPS, Graded Chronic Pain Scale; DSS, Disease Severity Scale; RMDQ, Roland Morris Disability Questionnaire; RA, rheumatoid arthritis; PSE, pain SE; OSE, other symptoms SE; VAS, visual analog scale; AIMS, Arthritis Impact Measurement Scale; SF-36, ; FM, fibromyalgia; PSEQ, Pain SE Questionnaire; CPG, Chronic Pain Grade; CSP, chronic shoulder pain; SPADI, Shoulder Pain and Disability Index; QuickDASH, Quick Disability of the Arm, Shoulder and Hand questionnaire; FIQ, Fibromyalgia Impact Questionnaire; KOA, knee osteoarthritis; WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index; CES-D, Center for Epidemiologic Studies Depression Scale; WPAI, Work Productivity and Activity Impairment; NHSPAQ II, Nurses Health Study II Physical Activity Scale; NRS, Numeric Rating Scale; NDI, Neck Disability Index; PSFS, Patient-Specific Functional Scale; SEPA, SE for Physical Activity scale; RASE, Rheumatoid Arthritis SE scale; PASE, Physical Activity Scale for the Elderly; PDI, Pain Disability Index; COPM, Canadian Occupational Performance Measure; Sf-36, The 36-item Short Form Health Survey; HADS-A, The Hospital Anxiety and Depression Scale-Anxiety Subscale; HADS-D, The Hospital Anxiety and Depression Scale-Depression Subscale.

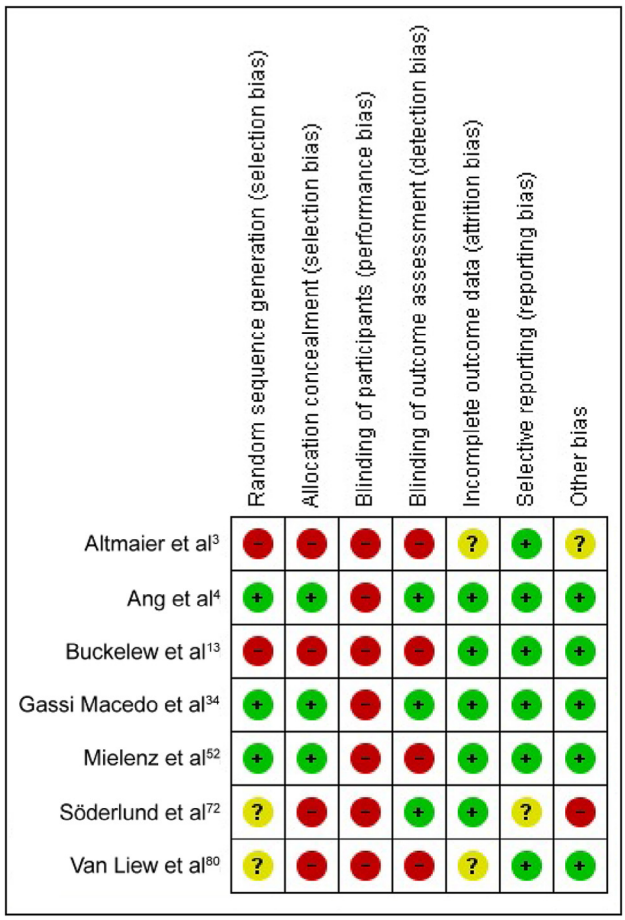

Figure 2. Summary of the methodological quality of included studies (randomized controlled trials).

SE and health status. The overall quality of evidence was very low.

SE and Pain Severity

Pain severity was evaluated by 1 study.[17] There was no statistical relationship between SE and pain severity. The overall quality of evidence was very low.

SE and Anxiety

Anxiety was analyzed by 1 study.[49] There was no statistical relationship identified between SE and anxiety. The overall quality of evidence was very low.

SE and Pain Behavior

Pain behavior was explored by 1 study.[56] There was no statistical relationship identified between SE and pain behavior. The overall quality of evidence was very low.

SE and Efficacy Beliefs

Efficacy beliefs was tested by 1 study,[35] reporting a positive and significant relationship between SE and efficacy beliefs. The overall quality of evidence was very low.

SE and Activity Interference

Activity interference was analyzed by 1 study.[4] There was no statistical relationship identified between SE and activity interference. The overall quality of evidence was very low.

Table 2. Methodological Quality for Cohort Studies (the NOS)

	Selection				Comparability		Outcome				
Reference	1	2	3	4	5	6	7	8	9	Total Score	LOE
Barlow[8]	+	–	–	–	–	–	–	+	+	3 of 9	P
Brekke et al[12]	+	–	–	–	–	–	–	+	–	2 of 9	P
Bishop et al[10]	–	–	–	–	–	–	–	+	–	1 of 9	P
Campbell et al[17]	+	–	+	–	–	–	–	+	+	4 of 9	P
Chester et al[18]	–	–	–	–	–	–	–	+	+	2 of 9	P
Demmelmaier et al[24]	+	+	+	+	+	+	–	+	–	7 of 9	F
Dobkin et al[26]	–	–	–	–	–	–	–	+	–	1 of 9	P
Engebretsen et al[29]	–	–	–	–	–	–	–	+	+	2 of 9	P
Foster et al[32]	+	–	–	–	–	–	–	+	–	2 of 9	P
Gere et al[35]	+	–	+	–	–	–	–	+	+	4 of 9	P
Haglund et al[29]	–	–	+	–	–	–	–	+	–	2 of 9	P
Iversen et al[42]	–	–	+	–	–	–	–	+	+	3 of 9	P
Karlsson et al[44]	+	–	–	–	–	–	–	+	–	2 of 9	P
Lotters[50]	+	–	+	–	–	–	+	+	+	5 of 9	P
Lowe et al[49]	–	–	–	–	–	–	–	–	+	1 of 9	P
Parker et al[56]	–	–	–	–	–	–	+	+	+	3 of 9	P
Rejeski et al[63]	+	–	+	–	–	–	–	+	–	3 of 9	P
Sharma et al[66]	+	–	–	–	–	–	–	+	+	3 of 9	P
Van Liew et al[79]	+	–	–	–	–	–	–	+	–	2 of 9	P

Abbreviations: LOE, level of evidence: F, fair quality; P, poor quality.
NOTE. Newcastle-Ottawa Quality Assessment Scale: cohort studies: 1 = representativeness of the exposed cohort; 2 = selection of the nonexposed cohort; 3 = ascertainment of exposure; 4 = demonstration that outcome of interest was not present at the start of the study; 5 and 6 = comparability of cohorts on the basis of the design or analysis; 7 = assessment of outcome; 8 = was follow-up long enough for outcomes to occur; and 9 = adequacy of follow-up of cohorts.

Table 3. Summary of Findings and Quality of Evidence Assessment

Summary of Findings			Quality of Evidence Assessment (GRADE)					
Outcome	Number of Studies	Number of Participants	Risk of Bias	Inconsistency	Indirectness	Imprecision	Other Considerations	Quality	Importance
SE (prognostic factor)									
Pain intensity	13	4,608	Serious*	No†	Very serious‡	Very serious§	Reporting bias¶	Very low	Critical
Disability	15	8,368	Serious*	No†	Very serious‡	Very serious§	N/A	Very low	Critical
Patient functioning	5	1,392	Serious*	No†	Very serious‡	Very serious§	N/A	Very low	Critical
Depressive symptoms	4	1,493	Very serious*	No†	No‡	Very serious§	N/A	Very low	Critical
Pain severity	1	1,591	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Critical
Presence of tender points	1	109	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Critical
Disease activity	3	582	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Critical
Physical activity participation	2	703	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Important
Activity interference	1	250	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Important
Satisfaction	1	101	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Important
Number of days for a total compensation benefits	1	187	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Critical
Work status	1	104	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Critical
Fatigue	1	659	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Critical
Health status	2	963	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Critical
Efficacy beliefs	1	304	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Critical
Anxiety	1	127	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Critical
Pain behavior	1	31	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Critical
Presenteeism	1	1,253	Very serious*	Very serious†	Very serious‡	Very serious§	N/A	Very low	Important

Abbreviation: OIS, optimal information size; CI, confidence interval.
*Randomized trials (lack of allocation concealment, lack of blinding, incomplete accounting of patients and outcomes events, selective outcome reporting bias, other limitations, observational studies (failure to develop and apply appropriate eligibility criteria, flawed measurement of exposure as well as outcome, failure to adequately control confounding, incomplete follow-up, nonpresence of an unexposed cohort).
†Point estimates vary widely across studies; confidence intervals show minimal or no overlap.
‡Differences in population, differences in intervention, differences in outcome, indirect comparison.
§OIS criterion is not met and the sample size is small; OIS criterion is met but the 95% CI around an effect does not exclude 1.0 (wide CIs); 95% CI is not reported.
¶Outcome data not included in the predictive model.

SE and Presence of Tender Points

The presence of tender points was tested by 1 study,[13] reporting a negative and significant association between SE and the presence of tender points. The overall quality of evidence was very low.

SE and Satisfaction With Their Performance

Satisfaction with their performance was evaluated by 1 study,[85] reporting a positive and significant association between SE and satisfaction with their performance at baseline. However, there was no statistical relationship between SE and satisfaction with the performance after intervention and at 9-month follow-up. The overall quality of evidence was very low.

SE and Number of Days for a Total Compensation Benefits

Number of days for a total compensation benefits was explored by 1 study.[50] There was no statistical relationship between SE and the number of days of total benefits during 12 months of follow-up. The overall quality of evidence was very low.

SE and Work Status

Work status was tested by 1 study,[29] reporting a positive and significant association between SE and work status. The overall quality of evidence was very low.

SE and Presenteeism (Reduced Productivity at Work)

Presenteeism was explored by 1 study,[38] reporting a positive and significant association between SE and presenteeism. The overall quality of evidence was very low.

SE and Fatigue

Fatigue was evaluated by 1 study,[12] reporting a negative and significant association between SE and fatigue. The overall quality of evidence was very low.

Discussion

Statement of Principal Findings

The objective of this review was to evaluate the influence of SE on the prognosis of CMP, on the basis of

Table 4. Summary of the Statistical Results of Each Outcome Measure Included in the Systematic Review; the Role of SE on the Prognosis of CMP

Reference	Pain Intensity	Pain Severity	Disability	Patient Functioning	Physical Activity Participation	Activity Interference	Disease Activity	Fatigue	Health Status
Altmaier et al[3]	Change of SE and pain intensity at discharge in MPQ pain intensity (B = .16, R^2 = .026; **at 6 months in MPQ pain intensity (B = –.41, R^2 = .165, *P* < .01) and MPQ pain rating index (B = –.33, R^2 = .109, *P* < .01);** MPQ pain rating index (B = .06, R^2 = .005)	—	—	Change of SE and patient functioning at discharge (B = .15 R^2 = .019); **at 6-month follow-ups (B = .24, R^2 = .52, *P* < .05)**	—	—	—	—	—
Ang et al[4]	Baseline SE, 3-month GCPS pain intensity: B = –.12 (.58), *P* = .8	—	—	—	—	Baseline SE, 3-month GCPS activity interference: B = –.56 (.81) *P* = .4	—	—	—
Barlow[8]	—	—	—	—	—	—	**Baseline SE, baseline disease activity: *P* < .0001; 6-month SE, 6-month disease activity: *P* < .0001**	—	—
Bishop et al[10]	—	—	**SE disability over time: between-persons effect: B = –.07, *P* < .01 and within-persons effect, B = –.04, *P* < .01**	—	—	—	—	—	—
Brekke et al[12]	**Changes in SE, changes in pain intensity over 5 years: SE pain, pain VAS r = –.24, *P* < .001**	—	—	—	—	—	—	**Changes in SE, changes in fatigue over 5 years: SE symptoms-fatigue VAS r = –.22, *P* < .001**	**Changes in SE, changes in health status over 5 years: SE pain-AIMS2 symptoms r = –.24, *P* < .001; SE pain-SF-36 pain r = .29, *P* < .001; SE symptoms SF-36 vitality r = .25, *P* < .001; SE symptoms- AIMS2 affect r = –.38, *P* < .001; SE symptoms-SF-36 mental health r = 034, *P* < .001**

Table 4. Continued

Reference	Pain Intensity	Pain Severity	Disability	Patient Functioning	Physical Activity Participation	Activity Interference	Disease Activity	Fatigue	Health Status
Buckelew et al[13]	**Baseline SE, baseline pain intensity: r = –.16, *P* = .09; changes in SE, changes pain intensity (6 weeks postintervention): r = –.25, *P* = .01**	—	**Baseline SE, baseline disability: r = –.49, *P* = .0001; changes in SE, changes in disability (6 weeks after intervention): r = –.09, *P* = .33**	—	—	—	**Baseline SE, baseline disease activity: r = –.09, *P* = .35; changes in SE, changes in disease activity (6 weeks after intervention): r = –.24, *P* = .01**	—	—
Campbell et al[17]	—	Baseline SE, low back pain (pain intensity) at 6 months: RR = .990 (95% CI = .98–1.0); baseline SE, low back pain (pain intensity) at 5 years RR = .989 (95% CI = .97–1.0)	Baseline SE, low back pain (disability) at 6 months (RR[95%CI] = .990[.98 to 1.0] baseline SE-low back pain (disability) at five years RR = .989 (95% CI = .97–1.0)	—	—	—	—	—	—
Chester et al[18]	**SE-SPADI (pain intensity) at 6 months: R^2 = –.36 (95% CI = –.50 to –.22), *P* < .001**	—	**SE-SPADI (disability) at 6 months: R^2 = –.36 (95% CI = –.50 to –.22), *P* < .001**	—	—	—	—	—	—
Demmelmaier et al[24]	Data not reported	—	**Baseline SE-, disability at 12 months: B = –.16 (95% CI =–.62 to –.09), *P* < .01**	—	—	—	—	—	—
Dobkin et al[26]	**Change in SE, pain intensity from baseline to 6 months after the end of treatment: B = –1.78 (95% CI = –3.38 to –.18), *P* = .0304**	—	**Change in SE, disability from baseline to 6 months after the end of treatment: B = –11.03 (95% CI = –20.82 to –1.25), *P* = .0281**	—	—	—	—	—	—
Engebretsen et al[29]	**Baseline SE, pain intensity at 12 months: B = 6.0 (95% CI = 2.0–9.9), *P* = .004**	—	**Baseline SE, disability at 12 months: B = 6.0 (95% CI = 2.0–9.9), *P* = .004**	—	—	—	—	—	—
Foster et al[32]	—	—	**Baseline SE, disability at 6 months: B = –.04 (95% CI =–.08 to –.01), *P* < .01**	—	—	—	—	—	—

Table 4. Continued

Reference	Pain Intensity	Pain Severity	Disability	Patient Functioning	Physical Activity Participation	Activity Interference	Disease Activity	Fatigue	Health Status
Gere et al[35]	—	—	—	**Baseline SE, baseline patient functioning r = .35, *P* < .05; changes in SE, changes in patient functioning at 6 months r = .39, *P* < .05; changes in SE, changes in patient functioning at eighteen months r = .39, *P* < .05**	—	—	**Baseline SE, baseline disease activity r = –.38, *P* < .05; changes in SE, changes in disease activity at 6 months r = –.38, *P* < .05; changes in SE, changes in disease activity at 18 months r = –.43, *P* < .05**	—	**Baseline SE, baseline health status r = .41, *P* < .05; changes in SE, changes in health status at 6 months r = .45, *P* < .05; changes in SE, changes in health status at 18 months r = .54, *P* < .05**
Haglund et al[38]	—	—	**SE (ASES pain), disability at 2.5 years: OR = .97(95% CI = .97–.98), *P* < .001; SE (ASES symptom) disability at 2.5 years: OR = .97 (95% CI = .57–.98), *P* < .001**	—	—	—	—	—	—
Iversen et al[42]	—	—	—	—	SE, physical activity participation over 3 years: SE ≥ 80, OR = 1.44 (95% CI = .72–2.84); SE ≥ 50 to 80, OR = 1.28 (95% CI = .66–2.46)	—	—	—	—

Table 4. Continued

Reference	Pain Intensity	Pain Severity	Disability	Patient Functioning	Physical Activity Participation	Activity Interference	Disease Activity	Fatigue	Health Status
Karlsson et al[44]	**Baseline SE, baseline, 1-year after intervention neck pain: r = –.38, *P* = .019;** baseline SE, baseline, 4- to 6-month follow-up, baseline SE, 4 to 6 months after intervention neck pain: r = –.10, *P* = .54; baseline SE, baseline, 1 year postintervention shoulder pain: r = –.20, *P* = .23; baseline SE, baseline, 4 to 6 months after intervention neck and shoulder pain (r = .10, *P* = .54; r = .21, *P* = .18); baseline SE, baseline, 1 year after intervention neck and shoulder pain (r = .14, *P* = .40; r = .19, *P* = .27)	—	**Baseline, 12 months pain SE, baseline, 12 months after intervention disability: B = .236 (95% CI = .018–.454), *P* = .035;** Baseline, 12 months general SE, baseline, 12 months after intervention disability: (B = .010 [95% CI = –.440 to .460], *P* = .96) Baseline, 4 to 6 months after intervention disability: B = –.086 (95% CI = –.541 to .368), *P* = .70; Baseline, 4 to 6 months pain SE, baseline, 4 to 6 months after intervention disability: B = .156 (95% CI = –.038 to .350), *P* = .11	—	—	—	—	—	—
Lötters et al[50]	—	—	—	—	—	—	—	—	—
Lowe et al[49]	—	—	—	—	—	—	—	—	—
Gassi Macedo et al[34]	SE-pain intensity at 2 months (B = .36 [95% CI = –1.12 to 1.85], *P* = .629); SE-pain intensity at 12 months (B = .73 [95% CI = –.92 to 2.37], *P* = .385)	—	SE-disability at 2 months (B = .03 [95% CI = –1.38 to 1.38], *P* = .969); SE-disability at 12 months (B = –.38 [95% CI = –1.78 to 1.02], *P* = .590	—	—	—	—	—	—

Table 4. Continued

Reference	Pain Intensity	Pain Severity	Disability	Patient Functioning	Physical Activity Participation	Activity Interference	Disease Activity	Fatigue	Health Status
Mielenz et al[52]	—	—	—	—	**SE at 3 months measured using RASE and SEPA, 3 months physical activity participation: (high RASE: > 105.85, mean = 79.4 (95% CI = 67.–91.6), low RASE: ≤ 105.85, mean = 65.5 (95% CI = 53.1–77.8), *P* = .117; SEPA: 1, mean = 49.2 (95% CI = 31.1–67.4); 2, mean = 63.7 (95% CI = 53.5–74.0); 3, mean = 78.3 (95% CI = 68.9–87.6); 4, mean = 92.8 (95% CI = 76.0–109.5), *P* = .005**	—	—	—	—
Parker et al[56]	—	—	—	—	—	—	—	—	—
Rejeski et al[63]	—	—	**Baseline SE, disability over time: *P* < .001**	**Baseline SE, patient functioning over time: *P* < .01**	—	—	—	—	—
Sharma et al[66]	—	—	**SE, disability over 3 years: adjusted OR for poor outcome/increment = .80 per 5 points (95% CI = .65 to .98)**	—	—	—	—	—	—
Söderlund et al[72]	Baseline SE, baseline pain intensity (preintervention): mean = 3.0 (SD = 1.4); SE postintervention, pain intensity postintervention: mean = 2.7 (SD = 1.7); SE 3 months, pain intensity 3 months after intervention mean = 2.6 (SD = 1.9)	—	Baseline SE, baseline disability (preintervention): mean = 21.3 (SD = 11.2); postintervention, disability postintervention: mean = 14.7 (SD = 13.0); SE 3 months, disability 3 months after intervention mean = 15.6 (SD =14.4)	—	—	—	—	—	—

Table 4. Continued

Reference	Pain Intensity	Pain Severity	Disability	Patient Functioning	Physical Activity Participation	Activity Interference	Disease Activity	Fatigue	Health Status
Van Liew et al[79]	**Baseline SE, baseline pain intensity: r = –.300, *P* < .001; 6 months SE, 6 months pain intensity: r = –.324, *P* < .001; 12 months SE, 12 months pain intensity: r = –.388, *P* < .001**	—	—	**Baseline SE, baseline patient functioning: r = –.491, *P* < .001; 6 months SE, 6 months patient functioning: r = –.499, *P* < .001; 12 months SE, 12 months patient functioning: r = –.467, *P* < .001**	—	—	—	—	—
Van Liew et al[80]	**Baseline SE, baseline pain intensity: r = –.272, *P* < .001 (depressed group) and r = –.113, *P* < .05 (nondepressed group); 6 months SE, 6 months pain intensity: r = –.375, *P* < .001 (depressed group) and r = –.276, *P* < .001 (nondepressed group); 12 months SE, 12 months pain intensity: r = –.458, *P* < .001 (depressed group) and r = –.236, *P* < .001 (nondepressed group)**	—	—	—	—	—	—	—	—
Walsh et al[85]	—	—	**Baseline SE, baseline disability: r = –.56, *P* < .001; postintervention SE, postintervention disability: r = –.40, *P* < .001; 9 months SE, 9 months disability: r = –.36, *P* < .01**	**Baseline SE, baseline patient functioning: r = .46, *P* < .001; postintervention SE, postintervention patient functioning: r = .25, *P* < .05; 9 months SE, 9 months patient functioning: r = .28, *P* < .05**	—	—	—	—	—

Table 4. Continued

Reference	Depressive Symptoms	Anxiety	Pain Behaviour	Satisfaction	Efficacy Beliefs	Presenteeism	Work Status	Number of Days for a Total Compensation Benefits	Number of Tender Points
Altmaier et al[3]	—	—	—	—	—	—	—	—	—
Ang et al[4]	—	—	—	—	—	—	—	—	—
Barlow[8]	—	—	—	—	—	—	—	—	—
Bishop et al[10]	—	—	—	—	—	—	—	—	—
Brekke et al[12]	—	—	—	—	—	—	—	—	—
Buckelew et al[13]	—	—	—	—	—	—	—	—	**Baseline SE, baseline number of tender points: r = –.15, *P* = .133; changes in SE, changes in number of tender points (6 weeks after intervention): r = –.24, *P* = .01**
Campbell et al[17]	—	—	—	—	—	—	—	—	—
Chester et al[18]	—	—	—	—	—	—	—	—	—
Demmelmaier et al[24]	—	—	—	—	—	—	—	—	—
Dobkin et al[26]	—	—	—	—	—	—	—	—	—
Engebretsen et al[29]	—	—	—	—	—	—	**Baseline SE, work status at 12 months: OR = .67 (95% CI = .5–1.0), *P* = .0052**	—	—
Foster et al[32]	—	—	—	—	—	—	—	—	—
Gere et al[35]	**Baseline SE, baseline depressive symptoms r = –.50, *P* < .05; changes in SE, changes in depressive symptoms at 6 months r = –.49, *P* < .05; changes in SE, changes in depressive symptoms at 18 months r = –.60, *P* < .05**	—	—	—	**Baseline SE, baseline efficacy beliefs r = .28, *P* < .05; changes in SE, changes in efficacy beliefs at 6 months r = .34, *P* < .05; changes in SE, changes in efficacy beliefs at 18 months r = .34, *P* < .05**	—	—	—	—

Table 4. Continued

Reference	Depressive Symptoms	Anxiety	Pain Behaviour	Satisfaction	Efficacy Beliefs	Presenteeism	Work Status	Number of Days for a Total Compensation Benefits	Number of Tender Points
Haglund et al[38]	—	—	—	—	—	**SE (ASES pain), presenteeism at 2.5 years: OR = .97 (95% CI = .97–.98), *P* < .001; SE (ASES symptom), presenteeism at 2.5 years: OR = .97 (95% CI = .96–.98), *P* < .001**	—	—	—
Iversen et al[42]	—	—	—	—	—	—	—	—	—
Karlsson et al[44]	—	—	—	—	—	—	—	—	—
Lötters et al[50]	—	—	—	—	—	—	—	SE, number of days for a total compensation benefits at 12-month follow-up: HR = .96 (95% CI (.90–1.01), *P* = .12	—
Lowe et al[49]	Baseline, 8 weeks after intervention SE (ASES pain), baseline, 8 weeks after intervention depressive symptoms: B = .02; baseline, 8 weeks after intervention SE (ASES symptoms), baseline, 8 weeks after intervention depressive symptoms: B = –.18 (adjusted R^2= .30)	Baseline, 8 weeks after intervention SE (ASES pain), baseline, 8 weeks after intervention anxiety: B = .02; baseline, 8 weeks after intervention SE (ASES symptoms), baseline, 8 weeks after intervention anxiety: B = –.10 (adjusted R^2= .13)	—	—	—	—	—	—	—
Gassi Macedo et al[34]	—	—	—	—	—	—	—	—	—
Mielenz et al[52]	—	—	—	—	—	—	—	—	—

Table 4. Continued

Reference	Depressive Symptoms	Anxiety	Pain Behaviour	Satisfaction	Efficacy Beliefs	Presenteeism	Work Status	Number of Days for a Total Compensation Benefits	Number of Tender Points
Parker et al[56]	—	—	Baseline pain SE, baseline pain behavior: r = –.32, *P* = .08; baseline function SE, baseline pain behavior: r = –.52, *P* = .002; baseline other SE, baseline pain behavior: r = –.34, *P* = .06; 6-month pain SE, 6-month pain behavior: r = –.20, *P* = .27; 6-month function SE, 6-month pain behavior: r = –.41, *P* = .02; 6-month other SE, 6-month pain behavior: r = –.37, *P* = .04	—	—	—	—	—	—
Rejeski et al[63]	—	—	—	—	—	—	—	—	—
Sharma et al[66]	—	—	—	—	—	—	—	—	—
Söderlund et al[72]	—	—	—	—	—	—	—	—	—
Van Liew et al[79]	**Baseline SE, baseline depressive symptoms: r = -.482, *P* < .001; 6 months SE, 6 months depressive symptoms: r = -.543, *P* < .001; 12 months SE: 12 months depressive symptoms r = –.555, *P* < .001**	—	—	—	—	—	—	—	—

Table 4. Continued

Reference	Depressive Symptoms	Anxiety	Pain Behaviour	Satisfaction	Efficacy Beliefs	Presenteeism	Work Status	Number of Days for a Total Compensation Benefits	Number of Tender Points
Van Liew et al[80]	**Baseline SE, baseline depressive symptoms: r = –.318, *P* < .001 (depressed group); r = –.221, *P* < .001 (nondepressed group); 6 months SE, 6 months depressive symptoms: r = –.510, *P* < .001 (depressed group); r = –.453, *P* < .001 (nondepressed group); 12 months SE: 12 months depressive symptoms r = –.478, *P* < .001 (depressed group); r = –.547, *P* < .001 (nondepressed group)**	—	—	—	—	—	—	—	—
Walsh et al[85]	—	—	—	**Baseline SE, baseline satisfaction with their performance r = .30, *P* < .01** postintervention SE, postintervention satisfaction with their performance: r = .20, *P* > .05; 9 months SE, 9 months satisfaction with their performance: r = .24, *P* > .05	—	—	—	—	—

Abbreviations: MPQ, McGill Pain Questionnaire; GCPS, Graded Chronic Pain Scale; VAS, visual analog scale; AIMS, Arthritis Impact Measurement Scale; RR, Relative Risk; CI, confidence interval; SPADI, Shoulder Pain and Disability Index; ASES, Arthritis SE Scale; OR, odds ratio; RASE, Rheumatoid Arthritis SE scale; SEPA, SE for Physical Activity scale.
NOTE. Significant results are shown in bold.

the analysis of longitudinal studies. Our results suggest that higher levels of SE could be significantly associated with a better prognosis (greater physical functioning, physical activity participation, health status, work status, satisfaction with the performance, efficacy beliefs, and lower pain intensity, disability, disease activity, depressive symptoms, presence of tender points, fatigue, and presenteeism) in CMP. Nevertheless, the quality of evidence was very low, the risk of bias was substantial, and the results were only on the basis of limited studies for most outcomes.

Comparison With Other Studies

To our knowledge, this is the first analysis of the evidence that shows the role of SE on the prognosis of CMP. Our findings are strongly in accordance with the social cognitive theory proposed by Bandura.[6] In this model, it is hypothesised that SE usually appears when an actual or perceived threat to deal with an unfavorable issue comes into play.[7] In this sense, increases in SE levels could help people with CMP to develop the ability to manage an adverse well known situation, even to carry out stressful tasks that individuals have never done. This model has been supported by previous reviews that have investigated the influence of SE in different chronic pain conditions.[9,14,59,75] Rajati et al[59] assessed the SE strategies to improve exercise in people with heart failure, showing the existence of a relationship between SE and the beginning and maintenance of exercise in this population, especially in the short-term. Thompson et al[75] explored how pain beliefs could predict treatment adherence in chronic pain. After analyzing 10 studies, evidence showed that pain-SE influences treatment adherence behaviors. Burke et al[14] investigated the most common psychological problems associated with chronic pain. In this review, SE appeared to be lower in chronic pain suffers. Furthermore, this review indicated how people with low SE showed more self-confidence to execute the demands associated with their life in general, than to control pain itself and hence, their ability to function in its presence, which is in accordance with the results of this study. Bérubé et al[9] tested the potential risk and protective factors in the development of chronic pain after extremity trauma. Pain-SE was shown as a protective factor in avoiding the development of chronic pain. Therefore, SE seems to play an essential role as a protective factor, which facilitates treatment adherence. This reduces the likelihood of developing or maintaining pain chronicity. In this context, a recent study put into practice the importance of SE to adherence rehabilitation through a model for clinicians to help individuals to carry out home exercise programs.[58] This model highlights the importance of the increase of SE levels to achieve the desired effects of home exercise. Both models, as well as our results and previous reviews underline the importance of SE not only as a predictor, but also as an outcome measure in people with chronic pain. High levels of SE can be achieved by the development of greater levels of mastery experiences, vicarious experiences, and verbal or social persuasion.[6] With this premise, a start point for clinicians could be the execution of psychological interventions to create and to strengthen SE beliefs.[23,54,78] However, despite these promising results, the conclusions should be taken with caution, because of the following: first, the presence of methodological inconsistencies (heterogeneity in the results and risk of bias) found in this and previous reviews[59,75]; and, second, the robust application of the GRADE approach in this review means that, although there is some promising results supporting the potential influence of the SE in the prognosis of CMP, the quality of the found evidence was very low.

Noteworthy, an interesting finding was obtained in this review. Karlsson et al[44] reported how higher pain-SE levels were significantly associated with greater disability at the end of their study. One possible explanation could be that the well defined, designed, and structured intervention that was carried out in this study could serve as an effective support for the participants, which means that people with low SE might have compensated for their uncertainty of self-confidence when moving, causing an uncommon association between both variables (SE and disability). Nevertheless, the relationship between SE and changes in outcome measures such as pain intensity, disability, pain severity, and others, as a result of a specific treatment, have been rarely investigated until now, and further research is required.

Strengths and Weaknesses of the Study

This study has a number of strengths, which include the use of a prespecified protocol registered on PROSPERO, the PRISMA checklist, the AMSTAR criteria, the GRADE system to evaluate the overall quality of the evidence, and the NOS checklist, the Cochrane collaboration tool, and MINORS checklist to determine the quality of each study. All of the procedures used for conducting this review were in accordance with current guidelines.[74] However, the limitations that are associated with this study must be acknowledged when the results are interpreted. First, although a long variety of MeSH terms, gray literature, and a manual search were carried out, it is still possible that not all studies were identified. Second, outcome measures were very diverse and in some cases authors used different self-reported questionnaires to measure the same outcome, which limits the opportunity to establish comparisons between the included studies. Another limitation of this study was that the results were only on the basis of 1 or 2 studies for most outcomes, thus, firm conclusions could not be drawn for these outcomes. Finally, mediation analysis should be primarily carried out with the aim of identifying causal mechanisms, to avoid possible inflation of the results.[40] Nevertheless, none of the included studies specifically evaluated the possible mediator effect of SE in CMP, and confounding variables were not always explored in all included studies. Therefore, results of this systematic review should be taken with caution.

Clinical Implications of Study Findings

SE levels can be improved by increasing the number of experiences (mastery of experience, vicarious

experience, physiological state, and verbal persuasion),[6] which are modifiable. As aforementioned, these experiences can be changed by clinicians with the aim of increasing SE levels. This can decrease pain, improve function, and ameliorate depressive symptoms. Along this line, the early identification of people with CMP who present low SE levels before the prescription of any therapy, may assist in clinical decision-making and timely and specific consultations with—or referral to—other health care providers. Furthermore, because SE is a common barrier to rehabilitation adherence, clinicians should be encouraged to assess SE levels of patients suffering from CMP to facilitate the implementation of individualized care.

Future Research

The present systematic review has concluded that there is low evidence of the influence of SE on the prognosis of CMP, mainly on the basis of the flaws found in the design of the analyzed studies. There is a clear gap in the literature that should be filled, mainly related to the absence of longitudinal studies that can solve these flaws. Hence, there are some recommendations to guide future research. Further studies prospectively analyzing the influence of SE (at baseline and follow-up) on the prognosis of CMP, standardizing self-reported measuring instruments to assess SE and outcome measures, are needed. Moreover, further experimental studies evaluating the effectiveness of therapeutic strategies (eg, pain self-management,[23] cognitive-behavioral therapy,[54] or mindfulness[78]) that appear to enhance SE levels, are required. Because CMP is a complex multifactorial condition, future investigations should consider the combination and interaction of a cluster of factors to increase their predictive value, and to determine the importance of each factor. Despite the promising influence of SE on the prognosis of CMP, further research that evaluates its role as a prognostic factor, even as a mediator of this condition, as well as its clinical usefulness, are needed. Last, conducting cohort studies with exposed and unexposed groups will not only permit the comparison of results, but also it will also allow carrying out a meta-analysis about the influence of SE on the different outcome measures.

Conclusions

This systematic review provided information about the role of SE on the prognosis of CMP. Our results suggest that higher SE levels are associated with greater physical functioning, physical activity participation, health status, work status, satisfaction with the performance, efficacy beliefs, and lower pain intensity, disability, disease activity, depressive symptoms, presence of tender points, fatigue, and presenteeism. Despite these promising results, further research is needed.

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Supplementary Data

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