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REVIEW

Systematic review and meta-analysis on the association between chronic low back pain and cognitive function

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Abstract

This study aimed to identify and assess the evidence on the association between idiopathic chronic low back pain (LBP) and cognitive function in individuals with LBP. A secondary aim was to explore whether changes in cognitive function are associated with pain characteristics and psychological factors (eg, catastrophizing and fear of movement). Eleven studies were included in this systematic review, and four meta-analyses were conducted. Low to very low-quality evidence suggests impaired cognitive function in individuals with LBP compared to asymptomatic controls for problem solving (k = 5; d = 0.33; CI = 0.16–0.50; z = 3.85 p = 0.0001), speed of information processing (k = 5; d = 0.44; CI = 0.22–0.65; z = 4.02 p<0.0001), working memory (k = 6; d = 0.50; CI = 0.34–0.66; z = 6.09 p<0.0001), and delayed memory (k = 3; d = 0.34; CI = 0.07–0.6, z = 2.49 p = 0.02). The association between LBP intensity and psychological factors and cognitive function was inconclusive. More studies are needed to explore these associations and improve evidence in this field. The results of this study suggest that cognitive aspects should be considered during the rehabilitation process of patients with LBP and raise further questions, including whether individuals with LBP are at a greater risk of developing dementia or whether targeting cognitive function will increase the probability of success of LBP treatment. These questions should, also, be considered in future studies.

KEYWORDS
chronic pain, cognition, cognitive function, cognitive impairment, low back pain

INTRODUCTION

Low back pain (LBP) is the most common pain syndrome in Europe, affecting around 50% of European citizens.[1] Worldwide, its annual prevalence has increased from 1.4% to 15.6% over the last two decades.[2] Furthermore, LBP is one of the main causes of years lived with disability,[3,4] which has increased by 29.8% from 1990 to 2017.[3]

Low back pain has been shown to be multidimensional and associated with negative psychological (eg, fear and emotional distress), social (eg, stress), and lifestyle factors (eg, unhealthy diet and insufficient exercise), as well as biological factors, including a dysfunctional pain processing system (eg, central sensitization).[5] In addition to these associations, previous studies have suggested that individuals with LBP have poorer performance in solving problems,[6,7] poorer working memory,[7,8] and higher difficulty in cognitive tasks[9] than asymptomatic individuals, suggesting an association between LBP and cognitive function. Additionally, in those with pain, higher pain intensity has been shown to be associated with poorer cognitive performance.[7,8]

Potential explanations for the association between pain and cognitive function are that pain uses cognitive resources, alters neural plasticity, and affects the expression and the activity of chemical and cellular neuromodulators, across a complex network of interconnected cognition-related brain regions, potentially resulting in

changes in cognitive functioning.[10] However, existing studies investigating the association between LBP and cognitive function show conflicting results.[7,11] In addition to methodological differences, the fact that studies investigate different cognitive domains (eg, memory, speed of information processing, and problem-solving) might explain the different conclusions.

To our knowledge, studies comparing cognitive function between individuals with LBP and asymptomatic individuals have not been systematically reviewed and synthesized. Therefore, the present study aims to identify and assess existing evidence on the association between pain and cognitive function in individuals with LBP. Studies were sub-grouped considering the cognitive domain assessed (eg, speed of information processing, problem-solving, working memory, and delayed memory) to inform on whether the potential association depends on the domain of cognitive function. A secondary aim was to explore whether changes in cognitive function are associated with pain characteristics and psychosocial factors (eg, catastrophizing and fear of movement).

METHODS

Protocol registration

This review was conducted according to Cochrane Collaboration[12] and Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) Statement guidelines.[13,14] The review protocol was published in PROSPERO (CRD42020218105).

Search strategy, sources, and study eligibility criteria

An electronic search was conducted in Pubmed, Science Direct, Scopus, and Web of Science in January 2021. A combination of words (eg, "low back pain", "cognitive function", and "cognitive impairment"), was used in the search strategy (detailed search available in Appendix S1). Initially, data searches were conducted for publications available from database inception until January 18, 2021. An update was conducted for the period between January 2021 and October 01, 2022, using the same search strategy. To be included in this review, studies had to evaluate cognitive function using any validated instrument in individuals with LBP and asymptomatic controls. For the purpose of this study, LBP was defined as "pain, muscular tension, or stiffness that is localized between the costal margins and the inferior gluteal folds, with or without leg pain (ie, sciatica)".[15,16] Nevertheless, studies not giving a definition for LBP, but stating that participants reported LBP, were also included. Commentaries, editorials, letters to the editor, or review articles were excluded. In addition, studies using mixed samples of participants with pain for which was not possible to retrieve data referring only to participants with LBP were also excluded. Included studies and review articles on cognitive function and pain identified during the search process were hand-searched for citations of interest.

Study selection process

All retrieved references were imported into *EndNote Web (Clarivate Analytics, London)*. One author (EPN) screened the titles and abstracts of all citations against the eligibility criteria. Then, full texts of potentially relevant articles were retrieved and screened independently by two authors (EPN and AGS). If consensus on inclusion or exclusion could not be reached, an independent third author (VTC) was consulted.

Risk of bias assessment

The National Institutes of Health (NHI)—Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies was used.[17] This scale was developed in 2013 by methodologists from the NHI and the Research Triangle Institute International, based on different quality methods and tools (eg, The Cochrane Collaboration, the Agency for Healthcare Research and Quality (AHRQ) Evidence-Based Practice Centers).[17] This tool has 14 questions that are answered 'yes', 'no', 'not applicable', or 'not reported'. Then, each study is given a quality rating of poor, fair, or good, based on the overall rater judgment of the study risk of bias.[17] Two reviewers (EPN and AGS) independently assessed each included manuscript and consensus was reached through discussion.

Data extraction

One author (EPN) used a customized form to extract the following data: (i) participant characteristics: sample size, gender, and age; (ii) LBP characteristics: duration and intensity; (iii) instruments used for cognitive assessment; (iv) mean and standard deviation for cognitive function for both the group of participants with LBP and the asymptomatic group; and (v) conclusion reached by each study authors.

Grading the quality of evidence

The overall quality and strength of evidence per outcome were assessed according to the Grading of Recommendations Assessment, Development, and Evaluation (GRADE).[18] Two authors independently assessed the quality of the evidence considering (i) risk of

bias, (ii) inconsistency of results, (iii) indirectness, (iv) imprecision, and (v) publication bias. Considering the design of included studies (observational), the baseline rating was "low-quality" evidence, which was then upgraded or downgraded based on the authors judgment for the five criteria listed above, and the overall quality rating was classified as high, moderate, low, or very low evidence.[18] A decision was reached by consensus.

Data synthesis and analysis

Measures of cognitive function were categorized according to the cognitive domain targeted into working memory, delayed memory, problem-solving, and speed of information processing, and separated data analysis was performed for each of these domains.

Problem-solving is defined as the process of constructing and applying mental representation of problems and finding a solution.[19] We included in this category of measures the Stroop Test, the Trail Making Test (TMT-part B), and the Iowa Gambling Test.[20]

The speed of information processing is usually measured as the time between a stimulus and an individual response.[21] The measures included in this domain were the Trail Making Test (TMT-part A); the Choice Reaction Time, which is a subtest from the Cambridge Neuropsychological Automated Batteries (CANTAB); and the Controlled Oral Word Association Task (COWAT). These tests are commonly used to assess the speed of information processing.[20]

Working memory or short-term memory is the ability to retain an amount of information during a short period of time.[22] The measures included in this domain were the Digit Span or Repetition series of digits from the Wechsler Adult Intelligence Scale (Wechsler), the Letter-number Sequencing subtest from Wechsler Adult Intelligence Scale (Wechsler), the Immediate Memory Test from the Repeatable Battery for the Assessment of Neuropsychological Status (RBANs), and the Interference Memory Task from the Brown-Peterson task. These tests are commonly used tests to assess working memory.[20]

Delayed memory is the capability to retain information for long periods.[23] The Delayed Memory Test from RBANs, the 12-word text from the Buschke Selective Reminding Test (SRT), and the Hopkins Verbal Learning Test-Revised (HVLT) were included in this category as they are reported in the literature as being commonly used to assess delayed memory.[20]

A meta-analysis using the *R Version 1.4.1106 (R Core Team, Vienna)* was performed with at least three studies categorized as belonging to the same cognitive domain. Effect sizes were determined by the standardized mean difference (SMD) and classified according to Cohen's guidelines as small (0.20), medium (0.50), and large (0.80) effects.[24] For each effect size, 95% confidence intervals (CIs) were calculated. Statistical heterogeneity was inspected using Cochran's Q statistic[25] and the I^2 statistic that ranges from 0% to 100%, with values of 25%, 50%, and 75% reflecting low, moderate, and high statistical heterogeneity, respectively.[26,27] A fixed-effect model was used as the heterogeneity was low, and Forest plots were used to present the SMD and CI for the individual studies of the meta-analysis and for the overall analysis. Most of the included studies assessed cognitive function using more than one instrument/test for each domain. In this situation, the most reliable test considering the Intraclass Correlation Coefficients (ICC) from previous studies was chosen for the meta-analysis. When ICC values were similar, the choice was based on similarity with instruments/tests used in other studies included in the same meta-analysis.

One of the studies[28] presented two groups of individuals with LBP and two groups of asymptomatic individuals, which were combined into only one group of individuals with LBP and one group of asymptomatic individuals following Cochrane guidelines.[12]

The strength of the association between LBP and psychological factors and cognitive function was interpreted as (i) little or no correlation ($r<0.25$), (ii) fair correlation ($0.25>r\leq0.50$), and (iii) moderate to good correlation ($0.50>r\leq0.75$) and good to excellent correlation ($r>0.75$).[29] A significant level of $p<0.05$ was set for all comparisons.

RESULTS

Search results

The results of the study selection are presented in Figure 1. A total of 26,964 articles were identified, being 25,332 articles identified in the first search and 1632 articles in the recent update search. Of these, 9848 were duplicates and were removed and 17,116 were screened by title and abstract. Of these, 57 were identified as potentially relevant, and their full text was retrieved. Finally, a total of 11 articles were included in the present review.

Risk of bias assessment

The methodological quality of the 11 included studies ranged from poor to fair, but only four (36.4%) studies were considered as being of fair quality.[7–9,30] All studies specified the research question (question 1) and utilized valid and reliable exposure measures (question 9). Most of the studies also defined the study population (n = 10 out of 11; 90.9%) (question 2), had clearly defined and valid outcome measures (n = 8 out of 11; 72.7%) (question 11), and reported that all subjects were selected or recruited from the same or similar populations and had similar eligibility criteria (n = 6 out of 11; 54.5%)

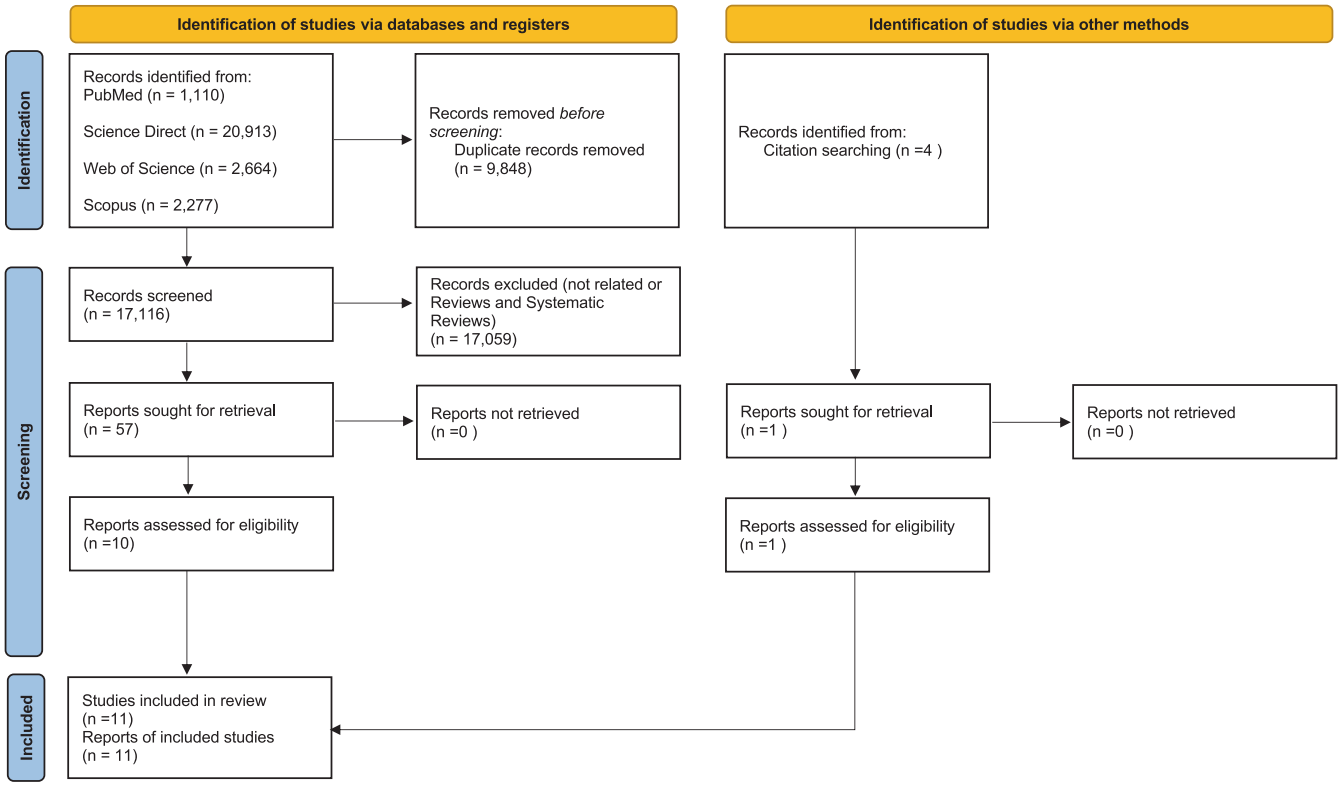

FIGURE 1 Preferred reporting items for systematic reviews and meta-analyses (PRISMA) flow diagram.[13,14]

(question 4). However, only one study justified the sample size (n = 1 out of 11; 9.1%) (question 5), and none of the studies reported using a blind assessor (question 12) or measured the exposure of interest prior to the outcome measurement (question 6). Also, none of the 11 included studies assessed exposure more than once (question 10) or had a sufficient timeframe to explore an association between exposure and outcome (question 7). A detailed summary of the methodological quality can be found in Table S1.

General overview of included studies

A total of 570 individuals with LBP and 475 controls were enrolled in the included studies. Although the search was focused on LBP (including acute and chronic), all studies reported that participants had LBP for at least 3 months, ie, chronic LBP. Most studies (n = 7 out of 11; 63.6%) referred a mean LBP duration that ranged between 6 and 14.2 months. The other four studies (36.4%)[7,8,11,31] only mentioned that duration of LBP was more than 3 months. The definition of LBP also varied among studies, with three (27.3%) studies including individuals with LBP with radicular pain,[11,32,33] two studies (18.2%) included only individuals without radicular pain,[7,31] one study (9.1%)[28] included musculoskeletal pain syndromes in the lumbosacral area, and the remaining studies did not provide an operational definition for LBP.[6,8,9,30,34]

Pain intensity was reported to have been assessed in all studies, and seven (63.6%) studies provided the results. It was assessed using the Visual Analogue Scale (VAS) or the Numerical Rating Scale (NRS) and varied between 4.17±2.45 and 6.62±2.04 out of a maximum score of 10, in the seven studies that provided results.[6–8,11,28,30,33] A summary of the characteristics of included studies can be found in Table S2.

Association between LBP and domains of cognitive function

Problem-solving

Problem-solving was assessed in seven studies (63.6%),[6,7,9,11,28,30,32] with individual studies using more than one test categorized into the same domain of cognitive function. The TMT (part B) was applied in four studies (36.4%)[6,7,9,11] and the Stroop test in five studies (45.4%).[6,11,28,30,32] Besides, the Wisconsin and the Iowa Gambling Tests were also applied in two studies.[6,32] Of the seven studies (63.6%), five (45.4%)[6,7,9,30,32] reported a significant difference between individuals with LBP and asymptomatic controls, consistent with individuals with LBP having lower problem-solving capabilities, and two studies (18.2%)[11,28] reported no between group differences. A summary of the individual study results can be found in Table S3.

A meta-analysis of five studies (45.4%) was performed using the results of the TMT (part B),[6,7,9,11] and of the Stroop test.[30] The other two studies[28,32] were excluded because it was not possible to extract data for the control group even after contacting the authors. There is very low-quality of evidence (Appendix S2) that individuals with LBP have decreased problem-solving abilities compared to asymptomatic individuals and a small-to-moderate effect size (k = 5; d = 0.33; CI = 0.16–0.50; z = 3.85 p = 0.0001) (Figure 2).

Speed of information processing

Speed of information processing was assessed in five studies (45.4%).[6,7,11,30,33] The TMT (part A) was used in three studies (27.3%),[6,7,11] and the Reaction Time Measurement System[33] and the COWAT was used in one study each (9.1%).[30] Besides, the CANTAB was also used by one study,[7] in addition to the TMT (part A). Of these five studies (45.4%), three (27.3%) studies[7,30,33] reported a significant difference between individuals with LBP and asymptomatic controls, and two studies (18.2%)[6,11] reported no between-group significant differences.

The meta-analysis of the five studies (45.4%)[6,7,11,30,33] used the results of TMT (part A) from three studies,[6,7,11] the Reaction Time Test[33] from one study, and COWAT[30] from one study (9.1%). There is very low quality of evidence (Appendix S2) that individuals with LBP have decreased speed of information processing abilities compared to asymptomatic individuals and a small-to-moderate effect size (k = 5; d = 0.44; CI = 0.22–0.65; z = 4.02 p<0.0001) (Figure 3).

Working memory

Working memory was assessed in eight studies (72.7%).[6–9,28,30–32] The Weschler Scale was used in seven studies (63.6%),[6–8,28,30–32] and the RBANs (immediate memory) were used in one study.[9] Besides, the Interference Memory task was also used in one study[6] that also used the Weschler scale. Of the eight (72.7%) studies, five (45.4%)[7–9,28,30] reported a statistically significant difference between participants with LBP and asymptomatic controls, consistent with individuals with LBP having a worst working memory. The other three studies (27.3%)[6,31,32] reported no between-group significant differences.

A meta-analysis was performed with data from six studies (54.5%), using data from the Weschler test,[6–8,28,30] and the RBANs.[9] The other two studies (18.2%)[31,32] were excluded because it was not possible to extract data for the control group even after contacting the authors. There is very low quality of evidence (Appendix S2) that individuals with LBP have decreased working memory abilities compared to asymptomatic individuals and a moderate effect size (k = 6; d = 0.50; CI = 0.34–0.66; z = 6.09 p<0.0001) (Figure 4).

Delayed memory

Three studies (27.3%)[9,28,30] assessed delayed memory using different tests: the RBANS for delayed memory, the 12-word text from the Buschke Selective Reminding Test (SRT), and the HVLT (delayed). There is a low quality of evidence (Appendix S2) that individuals with LBP have decreased delayed memory abilities compared to asymptomatic individuals (k = 3; d = 0.34; CI = 0.07–0.6, z = 2.49 p = 0.02) (Figure 5).

Association between LBP characteristics and cognitive function

The association between pain intensity and cognitive function was explored in seven (63.6%) studies,[6–8,11,28,32,34] but only five (45.4%) provided correlation values. Of these, two (18.2%)[6,32] reported a

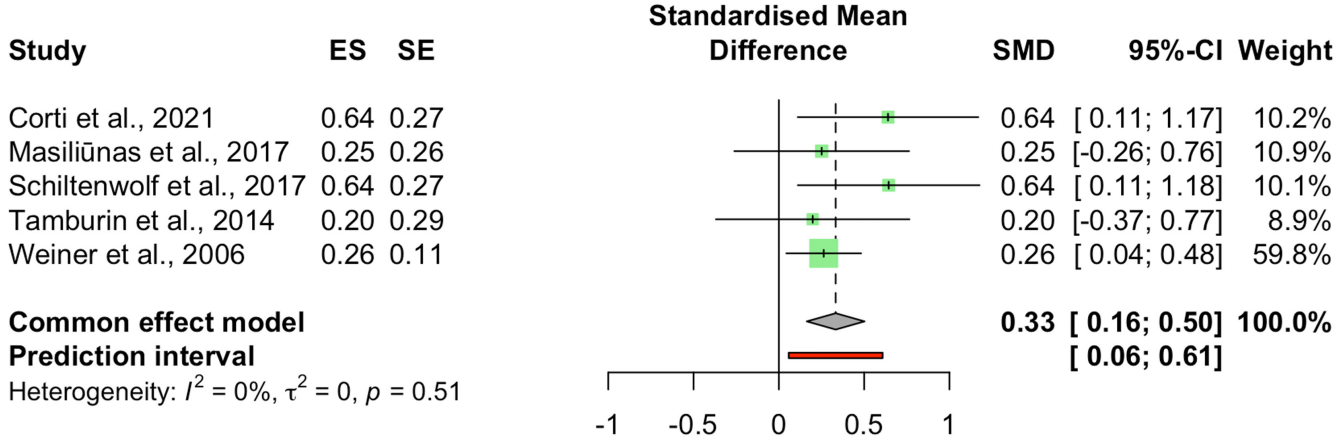

FIGURE 2 Forest plot of problem-solving (TMT- B test and Stroop test) results. ES, estimate effect; SE, standard error.

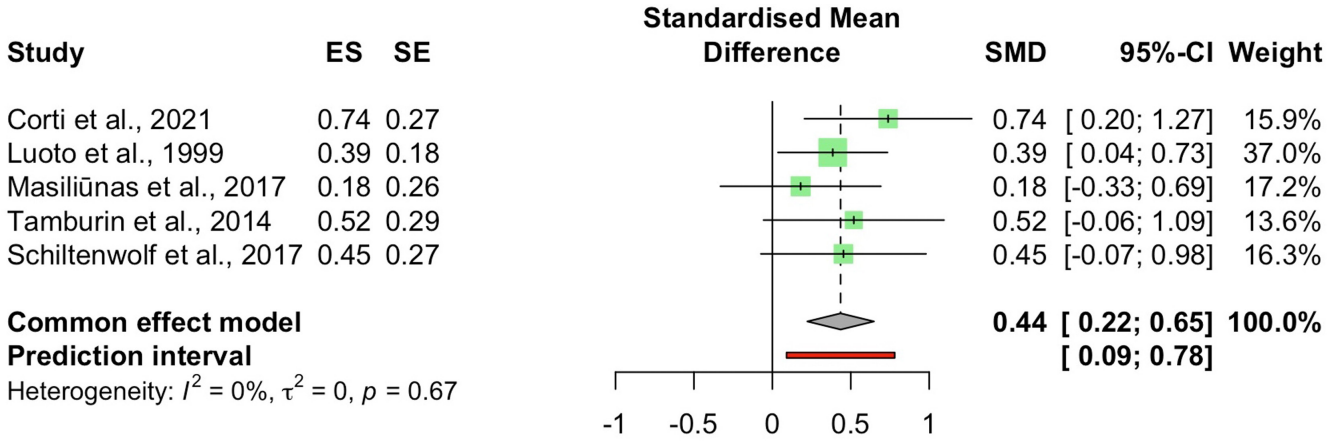

FIGURE 3 Forest plot of speed of information processing (TMT- A test, Reaction Time test, and COWAT) results. ES, estimate effect; SE, standard error.

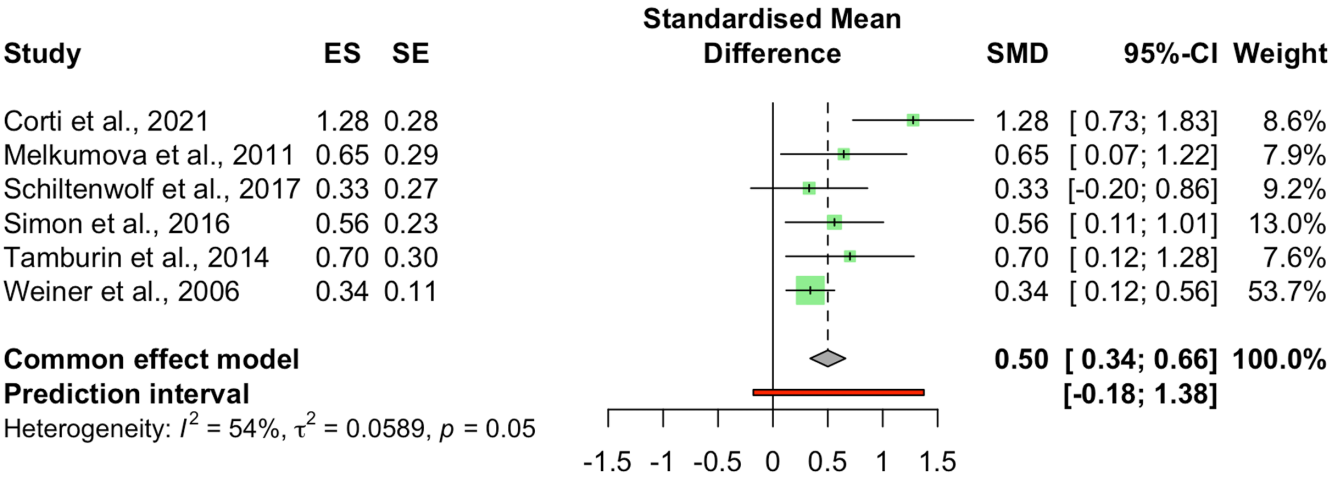

FIGURE 4 Forest plot of working memory (Weschler test and RBANs) results. ES, estimate effect; SE, standard error.

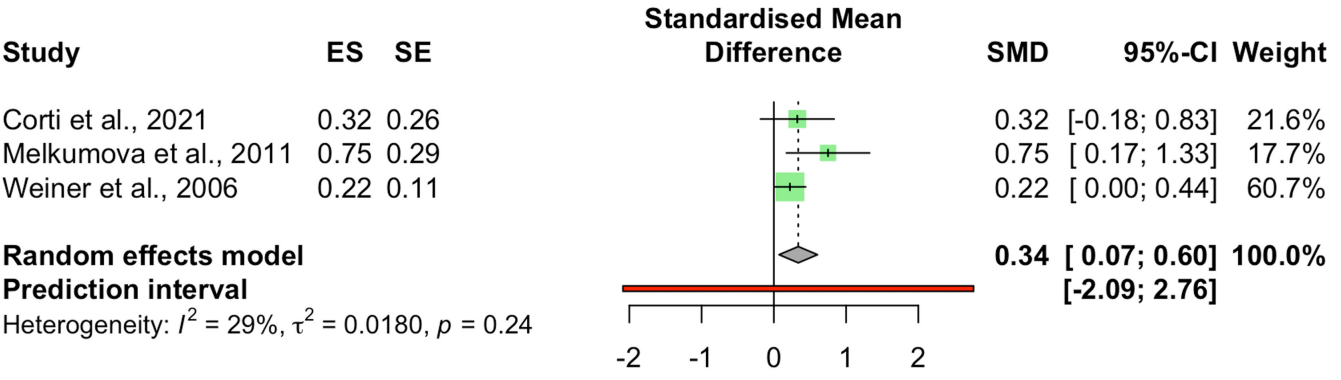

FIGURE 5 Forest plot of delayed memory (SRT, RBANs, and HVLT-delayed) results. ES, estimate effect; SE, standard error.

good-to-excellent correlation between pain intensity and measures of problem-solving (r of −0.75, $p<0.003$ and −0.76, $p = 0.009$)., suggesting that as pain intensity increases, problem-solving capabilities decrease; two (18.2%)[7,8] reported a fair correlation between pain intensity and measures of working memory (r of −0.35, $p = 0.045$ and −0.37, $p<0.001$), suggesting that as pain intensity increases, working memory decreases; and

the other (9.1%)[11] reported little to no correlation between pain intensity and measures of problem-solving (Kendall t = 0.12) and between pain intensity and measures of speed of information processing (Kendall $t = -0.03$). A summary of the associations can be found in Table S4.

Association between psychological factors and cognitive function

The association between anxiety and cognitive function was explored in five (45.4%)[6,7,11,28,32] of the 11 studies included in this review, and the results are conflicting. Of those that provided correlation values (n = 2, 18.2%), one[11] reported little to no correlation (Kendall $t = -0.10$) between anxiety and measures of problem-solving and between anxiety and measures of speed of information processing (Kendall t = −0.04). The other[7] reported a moderate-to-good correlation ($r = 0.57$, $p = 0.028$) between anxiety and measures of problem-solving, suggesting that as anxiety increases, problem-solving capabilities also increase. From the other three studies (27.3%) that did not provide correlation values, two (18.2%)[6,32] reported a non-significant correlation between anxiety and problem-solving, and one (9.1%)[28] reported a negative correlation between anxiety and speed of information processing, suggesting that as anxiety increases, speed of information processing decreases.

The association between depression and cognitive function was explored in four studies (36.4%)[6,7,11,31] also with conflicting results. Of those that provided correlation values (n = 2, 18.2%), one[7] found a moderate-to-good correlation between depression and measures of problem-solving ($r = -0.65$, $p = 0.007$), suggesting that as depression increases, problem-solving abilities decrease. The other study[11] showed little to no correlation between depression and measures of problem-solving (Kendall $t = 0.02$) and between depression and measures of speed of information processing (Kendall t = 0.04). The remaining two studies (18.2%)[6,31] did not provide correlation values but reported a non-significant correlation between depression and problem-solving.

The correlation between catastrophizing and cognitive function was explored in two studies (18.2%),[8,28] but only one study (9.1%)[8] provided the correlation results, indicating a fair correlation (r = 0.31, p<0.16) between catastrophizing and measures of working memory (ie, as catastrophizing increases, working memory also increases).

The correlation between fear of movement and cognitive function was explored in one study (9.1%)[8] that reported a fair correlation ($r = -0.48$, p<0.001) between fear of movement and a measure of working memory (ie, as fear of movement increases, working memory decreases). A summary of the associations can be found in Table S5.

DISCUSSION

This systematic review results suggest that there is very low quality of evidence that individuals with chronic LBP have decreased problem-solving abilities, speed of information processing, and working memory when compared with asymptomatic individuals, and low quality of evidence that individuals with chronic LBP have decreased performance for delayed memory. In addition, this systematic review found conflicting evidence on the association between pain characteristics and cognitive function and between psychological factors and cognitive function.

To the best of our knowledge, this is the first systematic review looking specifically at cognitive changes in individuals with LBP. However, a previous review with heterogeneous samples of patients with pain (including chronic whiplash-associated disorder, fibromyalgia, rheumatoid arthritis, and diabetes) has found a worst performance in tests of working memory, attention, and speed of information processing in these individuals when compared to asymptomatic controls[35] and in individuals with fibromyalgia compared to controls.[36] Similar to the present study, previous systematic reviews[37,38] have also reported an impairment in working memory in individuals with mixed pain syndromes, but, contrasting to our findings, no significant differences for delayed memory were found.[38] Delayed memory results might be conflicting due to the heterogeneous tests used in individual studies, a few including pain-related words, for which individuals with pain presented better memory results than asymptomatic controls.[38] There are three processes in remembering abilities: encoding (process information and creation of a new memory trace), storing (maintaining information), and retrieving (accessing information voluntarily or not).[38] Chronic pain may have an impact on some cognitive processes, eg, those that require attention, such as retaining information in working memory for processing or encoding or retrieving in delayed memory,[38] but not on others.

Different theories have been proposed to explain the association between chronic pain and cognitive function.[10] Chronic pain has an intrinsic connection to the limbic system,[39] which includes the amygdala, hippocampus, and the cingulate gyrus.[40] Those structures have an important function on memory and learning, as well as an association with emotional responses.[41–43] Additionally, individuals with chronic pain present a reduction in the volume of the thalamus, insular cortex, and cingulate cortex,[40,44,45] which are considered as the limbic-related cortex and are areas associated with executive functions, language, memory, and attention.[40,45] In chronic pain conditions, there is also a sensitization of the nervous system and an imbalance between a greater response to ascending stimuli and an inadequate activation of descending inhibitory pathways.[10,44] As chronic pain is associated with a dysregulation of the

modulatory circuits and with an altered chemical function in the central nervous system,[41–43] that competition between resources[39,41] may also aggravate the cognitive functioning.[10]

Concerning the association between pain intensity and cognitive function, previous systematic reviews including samples of heterogeneous chronic pain participants reported that individuals with higher pain intensity presented greater cognitive impairment than individuals with lower pain intensity in terms of attention and speed of information processing[35] and memory.[38] Furthermore, it has been suggested that the association might depend on the complexity of the cognitive task being performed, with more complex and demanding tasks being more competitive for resources.[46] Another explanation may be a slower psychomotor response as a weaker moderator between pain and cognitive function.[47]

Concerning the association between psychological aspects and cognitive function, a previous systematic review[35] reported greater memory impairment in those with higher emotional distress than those with lower scores of distress in a heterogeneous group of individuals with chronic pain (ie, chronic musculoskeletal conditions, including fibromyalgia, rheumatoid arthritis, and low back pain). The different results for the association between cognitive function and pain and psychological factors between previous reviews and the current systematic review might be due to differences in the painful syndromes included,[35,38] which differ in terms of pain characteristics, such as intensity or main mechanisms. Also, it is possible that the association between cognitive functioning and depression and anxiety might be modulated by other factors or be more relevant in the transition from acute to chronic pain. For example, increased vigilance in anxious patients might result in better cognitive performance.[48] A study in individuals with postoperative pain suggested that worse results in cognitive function may increase the odds of pain chronicity[49] and that depressive symptoms also predicted chronic pain at 12-month follow-up in those individuals.[49] Future studies should further explore the association between pain, psychological aspects, and cognitive function in subsamples of individuals with LBP (for example, acute LBP and nociplastic versus nociceptive LBP) as well as explore the importance of these factors in new onset LBP.

Most of the studies included in this systematic review were of high risk of bias due to aspects such as low sample size with no prior sample size calculation or the absence of a longitudinal design. A type 2 error is less likely to occur with adequate sample sizes.[50–52] Additionally, in cross-sectional studies, it is more difficult to evaluate correlations because all measurements, exposure, and outcome are simultaneously assessed.[51,53] Furthermore, most included studies did not account for the medication taken, and some types of medication might impact cognitive function.[10] Future studies should try to overcome these limitations.

Limitations

Only one reviewer screened titles/abstracts for inclusion, and potential studies might have been missed as a previous study[54] has suggested that a small percentage of additional studies might be found when two reviewers are used. Furthermore, the inability to access data for the subgroup of patients with LBP in studies with mixed samples led to a lower number of included studies. The number of studies included in this systematic review was small and all of them with a high risk of bias. Therefore, the overall effect of meta-analyses can be underestimated as a smaller weight is given to smaller studies.[52,55] Additionally, we had no success contacting authors from original studies for data retrieval and clarification, which resulted in fewer studies being included in the meta-analysis. These factors may affect the accuracy of the estimates, suggest caution in their interpretation, and highlight the need for further studies in this area.

Implication for clinical practice and research

These results suggest that problem-solving abilities, speed of information processing, and memory should be considered during the rehabilitation process of patients with LBP. Furthermore, they raise valuable questions, including whether individuals with LBP are at a greater risk of developing dementia or whether targeting cognitive function will increase the probability of success of LBP treatment. These questions should be considered in future research studies.

CONCLUSION

The present systematic review concluded that there is low to very low quality of evidence that individuals with chronic LBP have decreased abilities in problem-solving, speed of information processing, and worst working and delayed memory than asymptomatic controls. Also, it is inconclusive regarding the association between pain characteristics, psychological aspects, and cognitive function in individuals with LBP. The limited number of studies and the high risk of bias suggest caution on the interpretation of results and highlight the importance of further studies aiming to investigate the association between LBP and cognitive function.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.

DATA AVAILABILITY STATEMENT

The authors declare that all data supporting the findings of this study are available within the article and in its Supporting Information files.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

Table S1. Risk of bias assessment.

Table S2. Study characteristics and population.

Table S3. Cognitive function: instruments and mean (standard deviation) values for the group of individuals with low back pain and asymptomatic controls.

Table S4. Association between LBP and cognitive function.

Table S5. Association between psychological factors and cognitive function.

Appendix S1. Search strategy.

Appendix S2. GRADE and summary of findings tables

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