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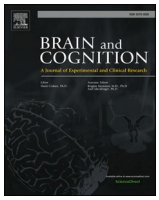

The effect of chronic pain on memory: A systematic review and meta-analysis exploring the impact of nociceptive, neuropathic and nociplastic pain

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ABSTRACT

Chronic pain is becoming increasingly prevalent in modern society. Much research to date has focused on the physical symptoms of pain associated with various conditions, yet living with chronic pain is also known to impact an individual's cognition. Within cognition, memory is particularly vulnerable to outside factors, yet our understanding of the impact of chronic pain on memory is inconclusive. This systematic review and *meta*-analysis examined the association between chronic pain type and memory performance. Chronic pain samples were classified as nociceptive, neuropathic or nociplastic and were compared to healthy controls. Studies were sourced from Embase, Web of Science, MEDLINE, PubMed, PsycINFO, Scopus and CINAHL databases between December 2023 and July 2024. A total of 15 good – strong studies with 1865 participants were included (106 who experienced chronic nociceptive pain, 315 who experienced chronic neuropathic pain, 589 who experienced chronic nociplastic pain and 855 healthy controls). Results indicated that individuals with nociceptive and nociplastic pain had impaired short-term and long-term memory performance compared to healthy controls. The same was not true for individuals with neuropathic pain. These findings demonstrate that the type of pain one experiences impacts memory performance. This has profound implications both clinically and with regard to research and offers a new lens for how we can consider chronic pain when trying to understand the impact on cognition.

1. Introduction

Chronic pain is a common and complex condition characterised by persistent pain experienced on most days of the week, for a period of more than 3 months. Almost 1 in 5 Australians experience chronic pain and this number is increasing. While chronic pain is often associated with the elderly, 68 % of people with chronic pain are of working age and 40 % of early retirement in Australia is due to chronic pain. Chronic pain limits daily activity and subsequently results in significant productivity loss (Duenas et al., 2016). When we think of chronic pain we tend to think of the physical experience of pain, however, living with pain can also have a profound effect on one's psychological wellbeing, cognition, and social relationships. In terms of cognition, chronic pain has been associated with broad impairments in cognitive function (Cherup et al., 2023; Coats et al., 2020; Dick et al., 2008) but there is a lack of consensus on how and why different aspects of cognition are impaired by different conditions and in different people.

Experiencing and managing pain draws on cognitive resources, limiting how they can be used for other processes (Phelps et al., 2021). In particular, memory systems are vulnerable to impact, with research demonstrating that memory is susceptible to pain, fatigue, medications, lack of sleep, and chronic health problems (Anggraini, 2023; Segura-Jimenez et al., 2016). Maintaining information in the conscious mind and rehearsing it in a way that allows for effective storage is a resource-intensive task as is actively retrieving it from these storage systems. When resources are limited due to other factors such as pain, our memory systems are unable to work as effectively as they would otherwise. Interestingly, most research to date investigating pain and

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cognition has focused on acute pain, which has been found to have varied cognitive impact (Moore et al., 2017). The little research on chronic pain has also produced inconclusive results e.g (Albrecht et al., 2016; Canaipa et al., 2022; Moriarty et al., 2017). Given the lack of understanding on how pain impacts cognition, in particular memory systems which are known to impact quality of life when impaired (Alfeo et al., 2022), and the increasing prevalence of chronic pain, understanding the impact of chronic pain on memory is crucial.

Research to date has approached the study of pain in humans as pain associated with specific conditions and/or diseases. However, research investigating the experience of pain highlights that not all pain is the same (Nijs et al., 2023). Broadly pain can be categorised into 3 types: nociceptive, neuropathic and nociplastic. Nociceptive pain is associated with tissue damage, neuropathic pain is associated with dysfunction or lesions on the nervous system and nociplastic pain describes pain that is associated with altered nociception despite there being no evidence of actual tissue damage which would cause the activation of pain pathways. These umbrella terms are not specific diagnoses but rather descriptors of the pain being experienced. The present review posits that the type of pain one experiences may be what influences variation in impact on cognition and, in particular, memory. Hence, this review aimed to systematically investigate current literature examining the association between pain type and memory performance, drawing on past research that has primarily focused on different conditions or populations whereby the presence of chronic pain is a shared experience, to better inform understanding of this association.

1.1. Understanding types of pain

Pain can broadly be described as a subjective, unpleasant sensory and emotional experience (Scherder et al., 2017). Pain has evolutionary purposes, alerting individuals when faced with harmful stimuli and/or harmful changes in the body (Garland, 2012). However, pain does not always have a clearly defined cause and for some, pain can persist, as a result of continual damage or disease or even after the original source has healed. Chronic pain is an often misunderstood condition that can be heavily stigmatised (Perugino et al., 2022). Yet, with the incidence of chronic pain increasing, developing a comprehensive understanding of chronic pain and associated conditions is paramount to effective management and support. Chronic pain is a broad umbrella term that encompasses any form of persistent pain, yet there is significant nuance to consider (13, 15). Like acute pain, chronic pain can be categorised based on the description of the pain experience with the same 3 broad classifications (nociceptive, neuropathic and nociplastic). The International Association for the Study of Pain (IASP) is in the process of developing clinical criteria and a grading system for all pain types. While this classification is not yet developed enough to be considered a ‘gold standard’ it is currently our most comprehensive set of descriptors to date and provides clinicians and researchers with a clear understanding of each pain type. This section will articulate the differences between each type of pain.

1.1.1. Nociceptive pain

Nociceptive pain results from tissue damage caused by trauma or inflammatory processes (Smith, 2018). It is the primary type of pain associated with musculoskeletal disorders and rheumatic diseases where the underlying pathology is structural in nature (Puntillo et al., 2021). Nociceptors are specialised nerve cells that detect harmful stimuli that could damage the body, such as thermal stimuli, mechanical stimuli or chemical stimuli (Kendroud et al., 2022). When nociceptors detect these harmful stimuli (e.g. extreme heat, pinching, inflammation respectively) these cells pass information from their location in the peripheral nervous to the spinal column within the central nervous system, this information then ascends to the brain for processing and response. Nociceptors can be found in most soft tissue, the skin and within internal organs (Garland, 2012). This form of pain is generally localized and can reduce as the impacted body part heals. However, in incurable conditions such as osteoarthritis, the pain is ongoing and often fluctuates. Patients experiencing nociceptive pain often describe it as an aching or throbbing pain (Puntillo et al., 2021).

1.1.2. Neuropathic pain

Neuropathic pain is nerve-based pain that can occur due to a malfunction along the peripheral or central nervous system (Pickering et al., 2013). Chronic neuropathic pain is usually the result of chronic and progressive nerve-based disease, but research also shows that it can be a biproduct of the healing process associated with tissue damage or infection (Costigan et al., 2009). Common neuropathic pain conditions include diabetic neuropathy, trigeminal neuralgia and multiple sclerosis. Chronic neuropathic pain is not typically triggered by a specific event, rather it reflects both peripheral and central sensitization mechanisms, that is, this pain can generally be better described as a dysfunctional communication mechanism. Hyperalgesia is the term used to describe an increased sensitivity to pain (Jensen and Finnerup, 2014) and is most commonly associated with neuropathic pain, as the sensitivity to pain one feels is the result of over-responsive nerves. In these cases, the pain one feels is real and debilitating, even if disproportionate to the stimuli. Patients experiencing neuropathic pain typically describe it as a burning or pinching sensation (Costigan et al., 2009).

1.1.3. Nociplastic pain

Nociplastic pain is used to describe chronic pain conditions such as fibromyalgia, where the pain an individual experiences is real despite having no discernible physical origin (Cohen, 2021). The term nociplastic pain was only established in 2016 and thus our understanding of it is limited (Nijs et al., 2023). That being said, nociplastic pain is mechanistically different from nociceptive and neuropathic pain (Bułdyś et al., 2023). Specifically, it alters the function of sensory pathways associated with pain in both the peripheral and central nervous and therefore is often more diffuse than the other pain types. The diagnosis of nociplastic pain (and associated conditions) is one based on exclusion (Nijs et al., 2023). To date laboratory markers are unable to distinguish between nociplastic pain and other pain types.

1.2. Processing pain

All pain is processed in the brain and imaging research has identified distributed networks that are responsible for the encoding of the sensory experience (Martucci and Mackey, 2018). Moreover, there is overlap between these regions and those responsible for affective processing (e.g. the amygdala in the limbic system) and cognitive processing (e.g. the prefrontal cortex). This research aids our understanding of the complexities of pain and explains why cognitive impairment and/or emotional dysregulation are often present in those with chronic pain. The core regions involved in the processing of pain are the primary/secondary somatosensory cortices, the anterior cingulate gyrus, the insula, the thalamus and the prefrontal cortex (Ong et al., 2019). Functional magnetic resonance imaging studies have shown that in chronic pain patients more widespread contralateral activation is observed as opposed to acute pain that is more localised and ipsilateral (Martucci and Mackey, 2018). These findings, however, explore chronic pain without consideration for differences between pain types.

Research attempting to make direct comparisons between types of pain is limited and thus many of the inferences made are based on the findings of separate studies. Early work attempted to understand the differences between nociceptive pain and neuropathic pain, particularly as a common belief at the time was that chronic pain associated with an original nociceptive injury after healing was the result of dysfunction in the nervous system and thus neuropathic in nature. In terms of physiology, neuropathic pain can originate from lesions in the nervous system (Costigan et al., 2009). Anatomical research shows that the lesion must directly involve the nociceptive pathways to result in nerve-based pain

(Boivie et al., 1989). Lesions of the nervous system but on non-nociceptive pathways do not induce nerve-based pain, or often any pain at all (Campbell and Meyer, 2006). This indicates that the pain pathways for nociceptive pain and neuropathic pain are in many ways shared. While it is not yet understood how nociceptive pathways are able to process both nociceptive and neuropathic pain, these findings highlight the commonalities while also acknowledging that there are different mechanisms at play.

Research investigating chronic neuropathic and nociplastic pain is more prolific than that investigating chronic nociceptive pain (especially without a disease-driven lens). This research arose during the development and classification of nociplastic pain as a separate pain type. Research has identified three categories for currently known mechanisms: supraspinal, spinal and peripheral (Bułdyś et al., 2023). The supraspinal mechanisms explain experiences such as hyper-responsiveness to pain stimuli, hyperactivity and connectivity between the parts of the brain responsible for pain perception observed in both types of pain. Decreased activity is also observed in the parts of the brain responsible for pain inhibition, in this case the medial prefrontal cortex, the anterior cingulate gyrus and the insula. Furthermore, connectivity between these regions is also often diminished. Interestingly, these three regions also have critical roles in cognition, with the medial prefrontal cortex being involved in attention, working memory and decision making (Jobson et al., 2021), the anterior cingulate gyrus being involved in cognitive flexibility, conflict monitoring (of cognitive resources) and motivation (Stevens et al., 2011), and the insula being involved with decision making, language, working memory and interoception (Zhang et al., 2024). Research has shown that variation in neurotransmitters is also present between individuals with neuropathic pain, nociplastic pain and healthy individuals (Fitzcharles et al., 2021). Specifically, research indicates that there are increased concentrations of glutamine in the cerebral spinal fluid, increased concentration of substance P and inhibition of GABA in individuals with pain, with those with nociplastic pain showing the greatest increase and disinhibition. Imaging studies indicate variation in the size and shape of grey and white matter in the aforementioned regions associated with pain and this can vary based on pain type (Alshelh et al., 2022). In terms of spinal mechanisms, dysfunction in the convergence of signals from pain loci are observed, as well as diminished pain inhibition with the spine and increased immune system activation, again with individuals with nociplastic pain showing the greatest changes (Popkirov et al., 2020). Finally, in terms of peripheral mechanisms, variation in the proliferation of sodium channels and sympatho-afferent coupling is observed (Bułdyś et al., 2023). While to date most of these findings are derived from animal-based research, they suggest that there may be anatomical and physiological variations based on pain type in humans. In terms of cognition, findings related to variations in volume and shape of grey and white matter and neurotransmitters are of particular interest. Given their roles in learning and cognition, this gives rise to the suggestion that cognitive symptoms associated with pain may differ based on pain type.

1.3. The secondary impacts of chronic pain: pain and cognition

Memory function is susceptible to impact from a range of factors associated with chronic health conditions (Anggraini, 2023). Memory is also fundamental to the human experience, broadly referring to the continued process of information retention and retrieval over time. While when we think of memory and recall we often do so in reference to the acquisition and consolidation of new information, memory is also critical to day-to-day life, as individuals encode and recall their routines, where key objects are located, and information related to work and maintaining social relationships (Zlotnik and Vansintjan, 2019). Contemporary memory models are well established and include 3 core systems that are supported by our attention and sensory systems initially. Short term memory is a conscious and active component of memory. Limited by both duration and capacity, information must be actively rehearsed to be maintained in the conscious mind. Neuroimaging studies show that short term memory primarily activates our temporal lobes and hippocampus. Working memory is a frontal lobe function that describes the active manipulation of information that is being held in short term memory. Subsequently the capacity of working memory is smaller than that of short-term memory and more resource intensive as individuals draw on information from long term storage to aid in decision making or manipulation. Finally, long term memory is said to be limitless in capacity and duration but is not a conscious storage. Assessment of long-term memory focuses on understanding how information is stored and retrieved.

It is well established that the presence of pain has a profound impact on an individual's quality of life and their capacity to complete daily tasks (Duenas et al., 2016). Research investigating this predominantly focusses on the impact of the physical aspects, but it is established that individuals with pain also often have diminished cognitive capacity and impaired social relationships (Segura-Jimenez et al., 2016). Given the push for a holistic, biopsychosocial approach to pain management further work to better understand the 'psychosocial' component is warranted. Research highlights that individuals with chronic pain report subjective memory complaints, particularly around recall and concentration (McGuire, 2013). Further research on objective measures of memory shows that implicit memory remains largely intact (Grisart and Van der Linden, 2001) giving further evidence that the cognitive resources pain utilises is a driver for impaired cognition, as cognitive resources are more predominantly used for explicit tasks. A recent review highlighted that it is still unknown whether the presence and/or intensity of chronic pain impacts on immediate memory, delayed memory, recognition memory or verbal and visual memory (Khera and Rangasamy, 2021). However, individual findings on acute pain suggest that where impairment is present it is exacerbated by the intensity of pain (Attridge, Noonan, Eccleston, & Keogh, 2015). Cognitive impairments or dysfunction have been identified in individuals with fibromyalgia (Albrecht et al., 2016), migraine (Tomes-Pires and Miro, 2014), chronic back pain (Karimi et al., 2016), diabetes neuropathy (Croosu et al., 2022), trigeminal neuralgia (Coats et al., 2020) and multiple sclerosis (Scherder et al., 2017) to name a few, however, few studies have focused specifically on memory in the context of the type of chronic pain one is experiencing (Jacobsen et al., 2023).

1.4. The current review

The aim of this systematic review was to evaluate the literature examining the association between different pain types and memory systems. Little research to date has investigated cognition through the perspective of type of pain. For this reason, we chose to include studies where pain was a primary symptom of the condition being examined and then categorised them during data extraction accordingly. As a result, it was not possible to compare individuals with different pain types to one another, instead the present review compares each pain population to healthy controls with the intent of identifying whether deficits are present in the different memory subsystems (short-term, working and long-term memory) in the pain population. While this will not allow us to draw inferences between the pain types, we will be able to infer whether memory is impacted as a result of each type of pain which will provide a sound basis for understanding.

2. Method

This *meta*-analysis was conducted in line with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) Guidelines (Page et al., 2021). PRISMA is a 27-item checklist used to improve transparency and quality of reporting in *meta*-analyses. The *meta*-analysis was registered with Open Science Framework on the 30th of June 2024.

2.1. Search strategy

The following databases were comprehensively searched; Embase, Web of Science, MEDLINE, PubMed, PsycINFO, Scopus and CINAHL. These databases were chosen based on the recommendations outlined in Bramer and colleagues (Bramer et al., 2017). The final search terms are shown in Table 1. Database searches were conducted on the 17th of November 2023 and the 1st of July 2024. The approximately 7.5-month break between searches aimed to encapsulate new and current research published in this period.

2.2. Study selection

Studies had to meet eligibility criteria for inclusion in the *meta*-analysis (see Table 2). The criteria are listed in a hierarchical order of priority. A pilot screening was done on 5 randomly selected studies to ensure consistency between reviewers.

Study screening was conducted independently by 3 reviewers in Covidence (Veritas Health Innovation, 2022), which is a primary screening tool used to manage and streamline the *meta*-analysis screening process. Duplicate studies were identified both by Covidence (n = 2457) and the reviewers (n = 11). Titles and abstracts were screened independently by two reviewers against the eligibility criteria. Studies were either excluded or retained for full text screening. The full texts of the included studies were assessed against the same criteria. The studies that did not meet the full criteria were excluded. There was approximately a 10 % discrepancy between reviewers in both rounds of screening. All discrepancies were resolved between reviewers by consensus.

2.3. Data extraction and synthesis

Data from the eligible studies were extracted using a template in Microsoft Excel. A summary of extracted variables is provided in Table 3. Any discrepancies were discussed by the reviewers and resolved by reviewing the relevant paper and arriving at consensus.

2.4. Quality assessment and risk of bias

The risk of bias and study quality were evaluated using the Standard Quality Assessment Criteria for Evaluating Primary Research Papers from a Variety of Fields (Kmet et al., 2004). This 14-item checklist allows for the evaluation of both qualitative and quantitative research papers using a 3-item scale. Reviewers determine if each item is answered as 'yes' (worth 2 points), 'partial' (worth 1 point) or 'no' (worth 0 points). Items that are not applicable are marked as so and excluded from the overall summary score. The summary score is determined by summing the total score of all relevant items and then dividing this value by the total possible score. Papers that score greater than 80 % are considered strong, 70–79 are considered good, 50–69 are considered adequate and scores less than 50 are considered to have limited quality. For the purpose of this study items 5–7 were removed as they focused on interventional designs. Therefore, the adapted scale consisted of 11 items, and the maximum score was 22. Each study was reviewed by two independent reviewers. Any studies that showed adequate quality or below (<70 %) were excluded from the *meta*-analysis. Any conflicting responses were resolved utilising a conservative approach by selecting the lower score.

2.5. Data analysis

All data were analysed using the JASP *meta*-analysis function (JASP Team (2024), 2024) using the random effects model. The random effects model was used as it acknowledges the heterogeneity and variance in effect sizes between studies. Meta analyses were performed for each domain of memory (STM, WM, LTM) and then for each pain group (Nociceptive pain/Controls, Neuropathic pain/Controls, Nociplastic pain/Controls). To be eligible for *meta*-analysis there needed to be a minimum of 3 studies. Due to limited studies 3 groups were excluded (Working memory – Nociceptive/Controls, Long term memory – Neuropathic/Controls and Nociplastic/Controls). All other groupings met the minimum criteria for *meta*-analysis. Prior to running the analyses, Hedges' g effect sizes and standard errors were calculated using MAVIS (Hamilton et al., 2015). Hedges' g was selected as it is less susceptible to the effects of small samples (Hedges and Olkin, 1985). Effect sizes were interpreted using Cohen's classifications of small (0.20), medium (0.5) and large (0.80). Where studies included multiple groups or conditions these were treated as separate studies and separate effect sizes and standard errors were calculated. All effect sizes were visualized using forest plots.

Heterogeneity was measured using an I^2 statistic with a 95 % confidence interval. This statistic identifies what proportion of variance is not just random error (Khan, 2020). A value of < 25 % would be considered low, between 26 and 74 % would be considered moderate and > 75 % would be considered high. Where heterogeneity is significant ($p < 0.05$) the risk of publication bias was also statistically assessed using Egger's regression test (Egger et al., 1997). Publication bias was also visually presented with a funnel plot. Where significant results were present Rosenthal's Failsafe *N* (Rosenthal, 1979) was also calculated to determine the number of unpublished or non-significant studies required to refute the *meta*-analysis results.

3. Results

3.1. Study selection

A total of 5671 studies were initially imported following the search of the databases. Following the removal of 2468 duplicates, the remaining 3203 studies underwent title and abstract screening against the eligibility criteria. At the end of this process 3114 studies were considered irrelevant and 88 papers were considered eligible for the full text screening phase. During this process 73 studies were excluded for various reasons, with the most common being 'No clearly defined pain group' (n = 31) meaning that the sample in the study could not be

Table 1
Meta-Analysis Search Terms.

Search Terms
1. 'Nocicept*' OR 'Nociceptive Pain' OR 'Tissue Damage'
2. 'Neuropath*' OR 'Neuropathic Pain'
3. 'Nociplastic' OR 'Nociplastic Pain' OR 'Functional Pain'
4. 'Memory' OR 'Short Term Memory' OR 'Working Memory' OR 'Long Term Memory' OR STM OR WM OR LTM
5. Pain
6. 1 AND 4
7. 2 AND 4
8. 3 AND 4
9. 5 AND 4

Note. * = truncation symbol, mild adjustments were made to terms to suit the interfaces of each database.

Table 2
Meta-Analysis Eligibility Criteria.

Inclusion Criteria
1. The study is in English
2. The full text is available
3. The study is an original, experimental, and peer reviewed research paper
4. The study contains a clearly defined pain group
5. The study contains a control group consisting of healthy individuals
6. There is a standardized, objective, cognitive measure of memory
7. Data is available for specific pain groups
8. Means and standard deviations are available for performance outcome (memory tests)

Table 3
Variables Extracted from the Studies Included in the Meta-Analysis.

Category	Data Extracted
Study Details	Study authors, year, title, aims
Participant Demographics	Sample size, sex (n of males and females), age (M, SD), diagnosis (for pain samples) for each sample (nociceptive, neuropathic, nociplastic, controls)
Task Information	Type of memory being evaluated (STM, WM, LTM), task name, modality of information (verbal, visual)
Performance outcome measures on each memory task	Performance outcomes (M, SD) for each memory task for the pain group and control group

Note. N = sample size, M = mean, SD = standard deviation, STM = short term memory, WM = working memory, LTM = long term memory.

clearly delineated as nociceptive or neuropathic or nociplastic pain. 15 studies met the full inclusion criteria and thus, were included for data extraction and included in the *meta*-analysis. Fig. 1 presents a PRISMA flow diagram demonstrating the study search and selection process (Page et al., 2021).

3.2. Study characteristics

The total number of participants in the *meta*-analysis was 1865, including 106 who experienced chronic nociceptive pain, 315 who experienced chronic neuropathic pain, 589 who experienced chronic nociplastic pain and 855 healthy controls. Sample sizes ranged from 23 to 673 (M = 121.33, SD = 158.79). Characteristics for each included study are displayed in Tables 4, 5 and 6.

3.3. Quality assessment and risk of bias

To determine the potential risk of bias, a quality assessment of the 15 papers included in the *meta*-analysis was performed using the Standard Quality Assessment Criteria for Evaluating Primary Research Papers from a Variety of Fields (Kmet et al., 2004). All received a score of 16 or above (M = 20.43, SD = 1.50). When converted to percentages, scores ranged from 73 % – 100 % (M = 92.50, SD = 6.81). Of the 15 studies, 14 studies were of strong quality and 1 was considered good quality. As no studies were considered adequate or of limited quality all were retained for the purpose of this research. Table 7 presents the results from the quality assessment.

3.4. Meta-analysis of memory performance based on pain type

The following section presents the *meta*-analyses conducted. To be eligible for inclusion a *meta*-analysis had to have at least three data points that would contribute to the analysis (Khan, 2020). Unless otherwise specified studies including verbal and visual measures were combined. Table 8 provides a summary of the variables inspected and analyses run.

As depicted in Table 8, the present study includes 6 *meta*-analyses that are detailed below.

3.5. Meta-analysis of short-term memory performance in individuals with nociceptive pain compared to healthy controls

A random effects (RE) analysis was performed on studies assessing short term memory performance in individuals with chronic nociceptive pain compared to healthy controls. As shown in Fig. 2a, individuals with nociceptive pain displayed significant deficits in short term memory when compared to healthy controls, with a large effect size (g = -0.95, 95 % CI = −2.26 to −0.06, p = 0.04). Heterogeneity was considered high (I^2 = 99.54 %). The Egger's test revealed the presence of a publication bias (p = 0.03) with a funnel plot displayed in Fig. 2b suggesting there may be a lack of studies on the left side of the plot. While Egger's test is recommended in the Cochrane Handbook for Systematic Reviews, recent research suggests that Egger tests may inflate type 1 errors. Thus, we supplemented our Egger's regression test with Rosenthal's Fail-Safe N test. Rosenthal's Fail-Safe N indicated 964 unpublished or nonsignificant studies would be required to render the findings non-significant.

3.6. Meta-analysis of short-term memory performance in individuals with neuropathic pain compared to healthy controls

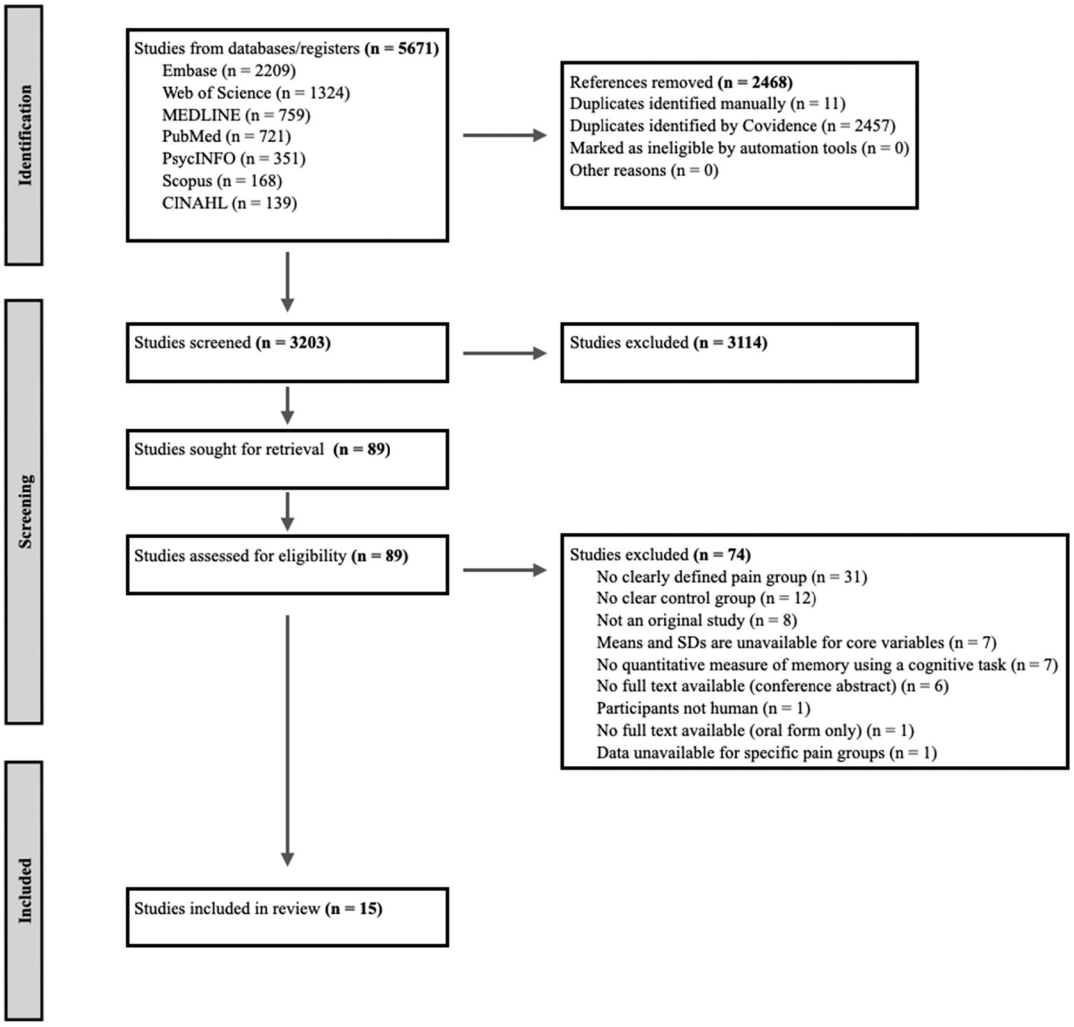
A random effects (RE) analysis was performed on studies assessing short term memory performance in individuals with chronic neuropathic pain compared to healthy controls. As shown in Fig. 3a individuals with neuropathic pain displayed no significant deficits in short term memory when compared to healthy controls, with a moderate effect size (g = -0.41, 95 % CI = −0.83 to −0.00, p = 0.06). Heterogeneity was considered high (I^2 = 99.23 %). The Egger's test revealed no publication bias (p = 0.28) with a funnel plot displayed in Fig. 3b. Due to the nonsignificant findings Rosenthal's Fail-Safe N was not calculated.

3.7. Meta-analysis of short-term memory performance in individuals with nociplastic pain compared to healthy controls

A random effects (RE) analysis was performed on studies assessing short term memory performance in individuals with chronic nociplastic pain compared to healthy controls. As shown in Fig. 4a, individuals with nociplastic pain displayed significant deficits in short term memory when compared to healthy controls, with a large effect size (g = -0.80, 95 % CI = −1.40 to −0.20, p = 0.01). Heterogeneity was considered high (I^2 = 99.40 %). The Egger's test revealed no publication bias (p = 0.92) with a funnel plot displayed in Fig. 4b. Rosenthal's Fail-Safe N indicated 14,951 unpublished or nonsignificant studies would be required to render the findings non-significant.

3.8. Meta-analysis of working memory performance in individuals with neuropathic pain compared to healthy controls

A random effects (RE) analysis was performed on studies assessing working memory performance in individuals with chronic neuropathic pain compared to healthy controls. As shown in Fig. 5a, individuals with neuropathic pain displayed no significant deficits in working memory when compared to healthy controls, with a moderate effect size (g = —0.15, 95 % CI = −0.40 to 0.10, p = 0.23). Heterogeneity was



Note. N = number of studies. Diagram adapted from PRISMA [38]

Fig. 1. PRISMA Flowchart of the Study Search and Selection Process Note. N = number of studies. Diagram adapted from PRISMA [38].

considered high ($I^2 = 97.20$ %). The Egger's test revealed no publication bias ($p = 0.61$) with a funnel plot displayed in Fig. 5b. Due to the nonsignificant findings Rosenthal's Fail-Safe N was not calculated.

3.9. Meta-analysis of working memory performance in individuals with nociplastic pain compared to healthy controls

A random effects (RE) analysis was performed on studies assessing working memory performance in individuals with chronic nociplastic pain compared to healthy controls. As shown in Fig. 6a, individuals with nociplastic pain displayed significant deficits in working memory when compared to healthy controls, with a moderate effect size (g = -0.46, 95 % CI = −0.88 to −0.05, $p = 0.03$). Heterogeneity was considered high ($I^2 = 99.14$ %). The Egger's test revealed no publication bias ($p = 0.85$) with a funnel plot displayed in Fig. 6b. Rosenthal's Fail-Safe N indicated 7353 unpublished or nonsignificant studies would be required to render the findings non-significant.

3.10. Meta-analysis of long-term memory in individuals with nociceptive pain compared to healthy control

A random effects (RE) analysis was performed on studies assessing nociceptive pain versus healthy controls on long-term memory. As shown in Fig. 7a, individuals with nociceptive pain displayed significant deficits in verbal long-term memory compared to healthy controls, with a moderate effect size (g = -0.48, 95 % CI = −0.69 to −0.27, $p < 0.001$). Heterogeneity was considered high ($I^2 = 80.35$ %). The Egger's test revealed no publication bias ($p = 0.36$) with a funnel plot displayed in Fig. 7b. Rosenthal's Fail-Safe N indicated 110 unpublished or nonsignificant studies would be required to render the findings non-significant.

4. Discussion

4.1. Findings of the meta-analyses

This systematic review and *meta*-analysis aimed to assess the literature exploring the impact that different types of pain have on memory. Although much research has been done exploring the impact that pain associated with specific chronic health conditions has on memory as well as cognition more broadly [e.g. 10, 20, 36, 54, 56], research investigating specific types of pain has been less overt and statements about the impact of chronic pain often group all types together (Cohen, 2021; Fitzcharles et al., 2021; McGuire, 2013). This has impacted our understanding of the effect that pain has on memory and cognition more broadly. Hence, this *meta*-analysis aimed to consider the type of pain (i.e., nociceptive, neuropathic or nociplastic) regardless of the condition it is associated with to glean an understanding of the effect that pain type has on memory performance. A total of 15 studies of good to high quality

Table 4
Characteristics for the Studies Included in the Meta Analysis – Short Term Memory.

Study – Authors and Year of Publication	Characteristics of the Pain Group					Characteristics of the Control Group			Short Term Memory Task
	Sample Size	Sex Split – M:F	Age	Diagnosis	Pain Category	Sample Size	Sex Split – M:F	Age	
(Albrecht et al., 2016) Albrecht et al 2016	12	0:12	28.30 (6.20)	Fibromyalgia	Nociplastic	11	0:11	28.40 (7.30)	Digit Span Forwards
(Canaipa et al., 2022) Canaipa et al 2022	29	0:29	50.41 (10.34)	Fibromyalgia	Nociplastic	122	47:74	24.33 (3.37)	Digit Span Forwards
(Cherup et al., 2023) Cherup et al 2023	26	14:12	34.35 (11.13)	Traumatic brain injury	Nociceptive	37	18:19	28.54 (7.63)	Brief Visual Memory Test – Revised Hopkins Verbal Learning Test
(Croosu et al., 2022) Croosu et al 2022	20	10:10	50.50 (NR)	Diabetic peripheral neuropathy with pain	Neuropathic	20	10:10	50.50 (NR)	N-Back (0-back)
(Jacobsen, Stiles, Stubhaug, Landro, & Hansson, 2021) Jacobsen et al 2021	29 45	11:17 5:40	45.60 (12.30) 48.50 (10.90)	Peripheral neuropathic pain Fibromyalgia	Neuropathic Nociplastic	20	NR	NR	Paired Associates Learning (CANTAB)
(Jongsma et al., 2011) Jongsma et al 2011	16	10:6	49.50 (11.90)	Chronic pancreatitis	Nociceptive	16	10:6	48.00 (11.30)	Digit Span Forwards Visual Span
(Karimi et al., 2016) Karimi et al 2016	31	12:19	43.30 (13.30)	Chronic lower back pain	Nociceptive	31	12:19	43.00 (13.00)	Free Recall
(Moriarty et al., 2017) Moriarty et al 2017	38	16:22	45.60 (9.90)	Chronic neuropathic pain	Neuropathic	38	16:22	44.20 (10.40)	Logical Memory 1 Spatial Span Forwards
(Palomo-Osuna et al., 2022) Palomo-Osuna et al 2022	71	36:35	71.68 (9.00)	Diabetic peripheral neuropathy with pain	Neuropathic	78	31:47	71.68 (9.00)	Test Your Memory – Anterograde Memory
(Pickering et al., 2013) Pickering et al 2010	42	20:22	72.00 (8.00)	Post herpetic pain	Neuropathic	42	20:22	72.00 (8.00)	Graded Naming Test
(Scherder et al., 2017) Scherder et al 2017	91	49:42	51.24 (9.71)	Multiple sclerosis	Neuropathic	80	36:44	48.79 (10.49)	Eight Words Immediate Recall
(Schiltenwolf et al., 2017) Schiltenwolf et al 2017	33	8:25	49.82 (10.23)	Lower back pain	Nociceptive	25	15:10	45.88 (9.24)	Spatial Span
(Segura-Jimenez et al., 2016) Segura-Jimenez et al 2016	388	21:367	47.95 (7.20)		Fibromyalgia	285	53:232	49.05 (9.35)	Rey Auditory Verbal Learning Task – Immediate Recall

Table 5
Characteristics for the Studies Included in the Meta Analysis – Working Memory.

Study – Authors and Year of Publication	Characteristics of the Pain Group					Characteristics of the Control Group			Working Term Memory Task
	Sample Size	Sex Split – M:F	Age	Diagnosis	Pain Category	Sample Size	Sex Split – M:F	Age	
(Albrecht et al., 2016) Albrecht et al 2016	12	0:12	28.30 (6.20)	Fibromyalgia	Nociplastic	11	0:11	28.40 (7.30)	Arithmetic
(Canaipa et al., 2022) Canaipa et al 2022	29	0:29	50.41 (10.34)	Fibromyalgia	Nociplastic	122	47:74	24.33 (3.37)	Digit Span Backwards
(Croosu et al., 2022) Croosu et al 2022	20	10:10	50.50 (NR)	Diabetic peripheral neuropathy with pain	Neuropathic	20	10:10	50.50 (NR)	N-Back (2-back)
(Dick et al., 2008) Dick et al 2008	30	0:30	49.60 (12.54)	Fibromyalgia	Nociplastic	30	0:30	46.56 (10.00)	Reading Span Test Teach – Auditory Working Memory Score
(Jacobsen, Stiles, Stubhaug, Landro, & Hansson, 2021) Jacobsen et al 2021	29 45	11:17 5:40	45.60 (12.30) 48.50 (10.90)	Peripheral neuropathic pain Fibromyalgia	Neuropathic Nociplastic	20	NR	NR	Spatial Working Memory
(Jacobsen et al., 2023) Jacobsen et al 2023									Spatial Working Memory
(Jongsma et al., 2011) Jongsma et al 2011	16	10:6	49.50 (11.90)	Chronic pancreatitis	Nociceptive	16	10:6	48.00 (11.30)	Digit Span Backwards
(Moriarty et al., 2017) Moriarty et al 2017	38	16:22	45.60 (9.90)	Chronic neuropathic pain	Neuropathic	38	16:22	44.20 (10.40)	Spatial Span Backwards
(Pickering et al., 2013) Pickering et al 2010	42	20:22	72.00 (8.00)	Post herpetic pain	Neuropathic	42	20:22	72.00 (8.00)	Stockings of Cambridge
(Scherder et al., 2017) Scherder et al 2017	91	49:42	51.24 (9.71)	Multiple sclerosis	Neuropathic	80	36:44	48.79 (10.49)	Digit Span Backwards
(Segura-Jimenez et al., 2016) Segura-Jimenez et al 2016	388	21:367	47.95 (7.20)		Fibromyalgia	285	53:232	49.05 (9.35)	PASAT

Table 6
Characteristics for the Studies Included in the Meta Analysis – Long Term Memory.

Study – Authors and Year of Publication	Characteristics of the Pain Group					Characteristics of the Control Group			Long Term Memory Task
	Sample Size	Sex Split – M:F	Age	Diagnosis	Pain Category	Sample Size	Sex Split – M:F	Age	
(Cherup et al., 2023) Cherup et al 2023	26	14:12	34.35 (11.13)	Traumatic brain injury	Nociceptive	37	18:19	28.54 (7.63)	Brief Visual Memory Test Revised – Delayed Hopkins Verbal Learning Test – Delayed
(Jongsma et al., 2011) Jongsma et al 2011	16	10:6	49.50 (11.90)	Chronic pancreatitis	Nociceptive	16	10:6	48.00 (11.30)	Verbal Word Learning Delayed
(Moriarty et al., 2017) Moriarty et al 2017	38	16:22	45.60 (9.90)	Chronic neuropathic pain	Neuropathic	38	16:22	44.20 (10.40)	Logical Memory II
(Scherder et al., 2017) Scherder et al 2017	91	49:42	51.24 (9.71)	Multiple sclerosis	Neuropathic	80	36:44	48.79 (10.49)	Eight Words Delayed Recall
(Segura-Jimenez et al., 2016) Segura-Jimenez et al 2016	388	21:367	47.95 (7.20)		Fibromyalgia	285	53:232	49.05 (9.35)	Rey Auditory Verbal Learning Task – Delayed Recall

Table 7
Quality Assessment Outcomes.

Study	Questions											Score	%	Rating
	1	2	3	4	5	6	7	8	9	10	11			
(Albrecht et al., 2016) Albrecht et al (2016)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Partial	Yes	Yes	Partial	21	95	Strong
(Cherup et al., 2023) Cherup et al (2023)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	22	100	Strong
(Canaipa et al., 2022) Canaipa et al (2022)	Partial	Partial	Partial	Yes	Yes	Partial	Yes	Yes	Yes	Partial	Partial	16	73	Good
(Croosu et al., 2022) Croosu et al (2023)	Yes	Yes	Yes	Yes	Yes	Partial	Yes	Yes	Partial	Yes	Yes	29	90	Strong
(Dick et al., 2008) Dick et al (2008)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	22	100	Strong
(Jacobsen, Stiles, Stubhaug, Landro, & Hansson, 2021) Jacobsen et al (2021)	Yes	Yes	Yes	Yes	Partial	Yes	Yes	Yes	Yes	Partial	Partial	20	90	Strong
(Jacobsen et al., 2023) Jacobsen et al (2023)	Yes	Yes	Partial	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	21	95	Strong
(Jongsma et al., 2011) Jongsma et al (2011)	Yes	Yes	Partial	Partial	Yes	Partial	Yes	Yes	Yes	Yes	Yes	21	95	Strong
(Karimi et al., 2016) Karimi et al (2016)	Partial	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Partial	Yes	Yes	20	90	Strong
(Moriarty et al., 2017) Moriarty et al (2017)	Yes	Yes	Yes	Partial	Yes	Partial	Yes	Partial	Yes	Yes	Yes	21	95	Strong
(Palomo-Osuna et al., 2022) Palomo-Osuna et al (2022)	Yes	Yes	Yes	Yes	Partial	Yes	Yes	Yes	No	Partial	Yes	19	86	Strong
(Pickering et al., 2013) Pickering et al (2010)	Partial	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Partial	Yes	Partial	19	86	Strong
(Scherder et al., 2017) Scherder et al (2017)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	22	100	Strong
(Schiltenwolf et al., 2017) Schiltenwolf et al (2017)	Yes	Yes	Partial	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	21	95	Strong
(Segura-Jimenez et al., 2016) Segura-Jimenez et al (2016)	Yes	Yes	Yes	Yes	Yes	Partial	Yes	Yes	Partial	Yes	Yes	21	95	Strong

Note Yes Partial No.

met the inclusion criteria and contributed to 6 separate *meta*-analyses.

Overall, our results suggest that individuals diagnosed with chronic pain are impacted differently based on the type of pain they experience. Specifically, our *meta*-analyses revealed that individuals experiencing nociceptive and nociplastic pain performed worse than healthy controls on short term and working memory tasks, whereas individuals with neuropathic pain showed no deficits. This highlights that pain type may be a factor that explains the variable findings that exist in the literature to date regarding how chronic pain impacts memory. As chronic pain is often long term and/or permanent, gaining an understanding of the secondary impacts of pain (e.g., psychological) can allow healthcare providers to better support individuals (Segura-Jimenez et al., 2016).

Table 8
Summary of Meta-Analyses.

	Nociceptive Pain*	Neuropathic Pain*	Nociplastic Pain*
Short Term Memory	N = 6	N = 8	N = 4**
Working Memory	Ineligible for inclusion	N = 7	N = 6
Long Term Memory	N = 3**	Ineligible for inclusion	Ineligible for inclusion

* All pain types were compared to healthy controls.
** All memory tests were verbal in nature.

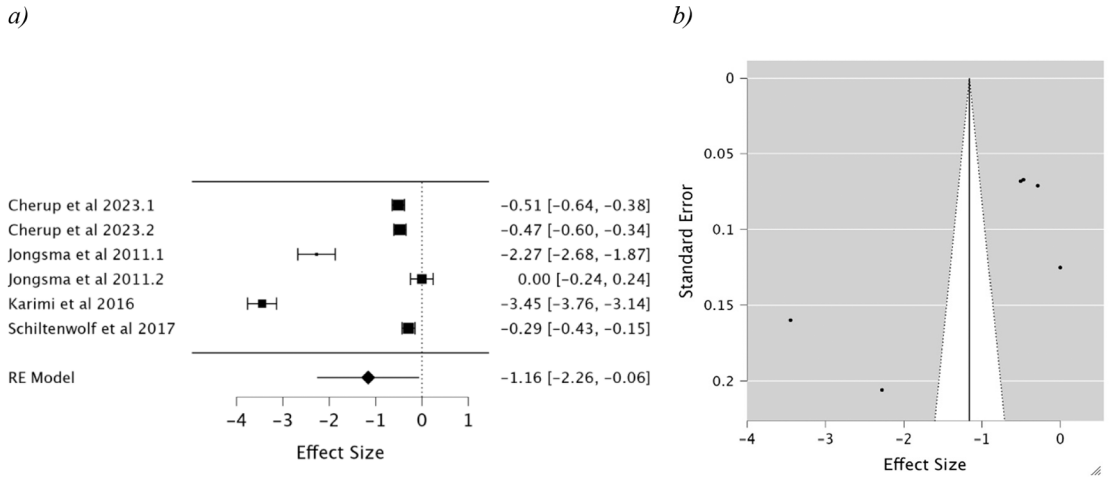
This research also highlights how understanding the type of pain a patient has may elucidate a more accurate understanding of how their pain impacts their memory, and possibly subsequent cognition, more so than simply considering the pain as chronic or a symptom of a chronic condition. Moreover, much research exploring the impact of chronic pain does so through the lens of neuropathic pain (Moore et al., 2017; Jensen and Finnerup, 2014; Cohen, 2021), but the findings of the present review indicate that we cannot assume that the findings of these studies are applicable to all chronic pain patients and highlight the importance of further investigation to better understand the impact of pain type.

4.2. The connection between cognition and physiology

The present research focuses on the cognitive impact of chronic pain and provides encouraging support that there are differences between pain types. While the present review did not investigate anatomical or physiological mechanisms, given the known correlation between cognition and anatomy and physiology we can posit some potential avenues that warrant further investigation. Research to date is mostly animal based and shows that there are minor differences associated with pain type in aspects such as supraspinal mechanisms (Bułdyś et al., 2023), concentration of grey and white matter (Alshelh et al., 2022) and activation of neurotransmitters (Fitzcharles et al., 2021). While these differences are minor, we know that the presence of grey and white matter is correlated with cognition and cognitive impairment, as too are the presence and concentrations of different neurotransmitters (Teleanu et al., 2022). The findings of the present review do not allow for a causal relationship to be inferred but they may provide evidence and a different lens for identifying physical differences. If an average comprehensive cognitive profile is able to be developed for each pain type, then researchers may be able to use these profiles to identify which regions of the brain warrant further interrogation to determine whether there are anatomical or physiological differences. Alternatively, this review suggests that while some aspects of pain are able to be observed physically, perhaps not all aspects of pain are (like aspects of memory consolidation). Thus, understanding the cognitive and behavioural profiles associated with pain type may be able to be used in conjunction with our physical knowledge to build a comprehensive understanding of each pain type.

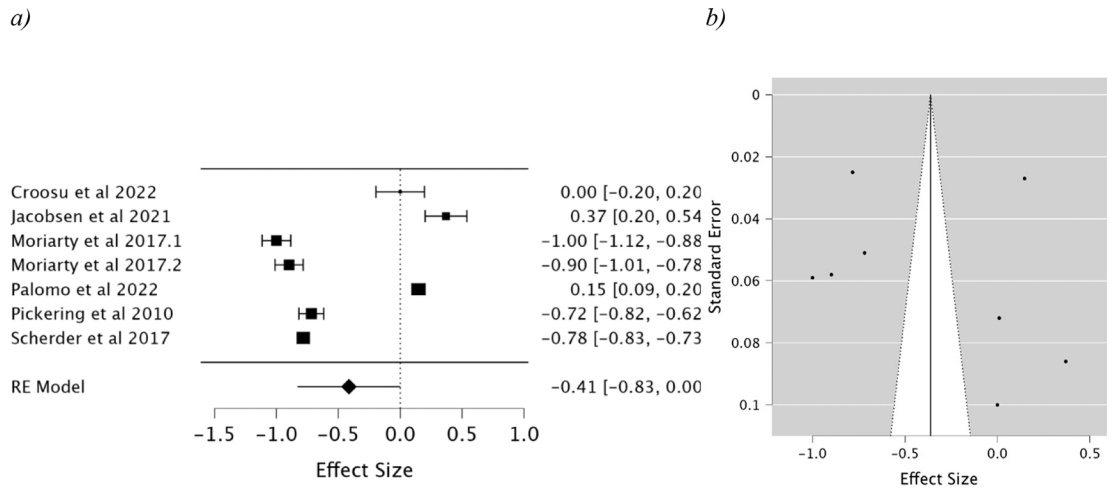
4.3. Limitations and future directions

This review has limitations that need to be considered. As pain was a primary focus, only papers that described their condition as having pain as a primary symptom or which included a measure of pain for the clinical group were eligible. This may have resulted in studies that contained similar populations to those within the study (e.g. a multiple sclerosis group) being excluded because in those studies researchers were more focused on other related symptoms such as tingling and thus, were not flagged in our search strategy. Given the broad range of conditions that can be associated with chronic pain it was not viable to include all these conditions within the search criteria. Moreover, symptom manifestation with some chronic conditions can vary from person to person (e.g., while most people experience pain, pain is not a requirement for diagnosis with that condition). In an attempt to counter this, studies were only included if they had pain as a primary descriptor and/or employed a measure of pain for participants. In the same vein it is important to acknowledge that many chronic conditions have comorbidities or symptoms which could impact cognition. Factors such as fatigue, anxiety or depression are known to also be present in many individuals with chronic pain (Duenas et al., 2016) and are established as having an impact on memory. While where possible we attempted to control for this (e.g a study has a pain group, a pain and depression group and healthy controls only data from the pain group and healthy



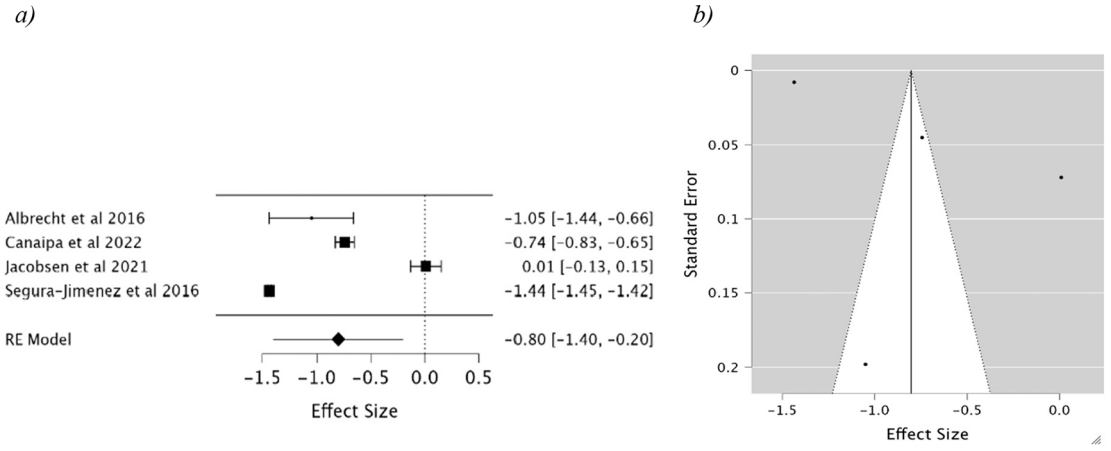
Note RE Model = Random Effects Model, negative effect sizes are indicative of poorer short term memory performance in the nociceptive pain group compared to healthy controls*

Fig. 2. A) forest plot comparing short term memory scores between people with nociceptive pain and healthy controls b) funnel plot of publication bias note* re model = Random Effects Model, negative effect sizes are indicative of poorer short term memory performance in the nociceptive pain group compared to healthy controls.



Note RE Model = Random Effects Model, negative effect sizes are indicative of poorer short term memory performance in the neuropathic pain group compared to healthy controls*

Fig. 3. A) forest plot comparing short term memory scores between people with neuropathic pain and healthy controls b) funnel plot of publication bias note* re model = Random Effects Model, negative effect sizes are indicative of poorer short term memory performance in the neuropathic pain group compared to healthy controls.



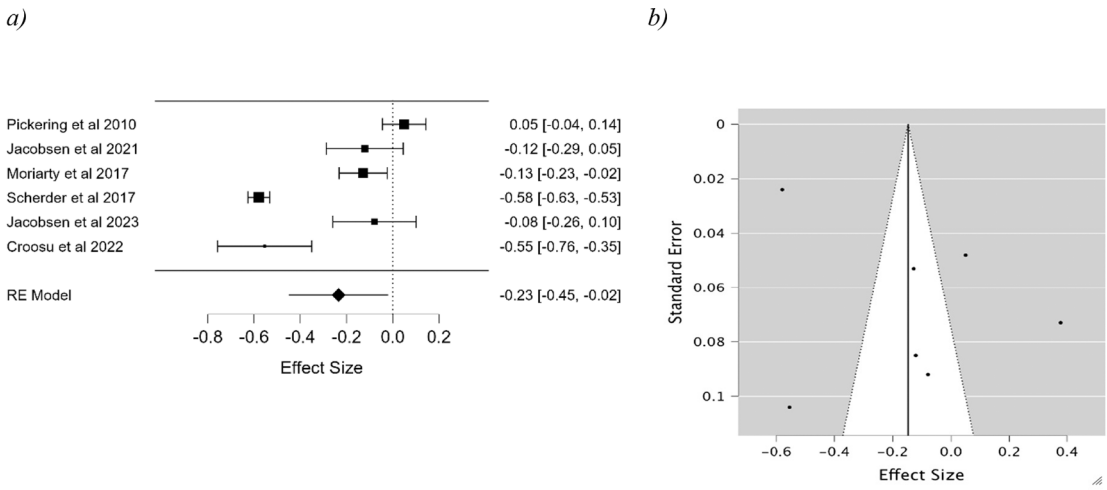
Note RE Model = Random Effects Model, negative effect sizes are indicative of poorer short term memory deficits in the nociplastic pain group compared to healthy controls*

Fig. 4. A) forest plot comparing short term memory scores between people with Nociplastic Pain and Healthy Controls b) Funnel Plot of Publication Bias Note* RE Model = Random Effects Model, negative effect sizes are indicative of poorer short term memory deficits in the nociplastic pain group compared to healthy controls.

controls were included) it was not possible to eliminate this entirely. Thus, results should be interpreted with caution as pain may not be the only causal factor contributing to memory impairments. Furthermore, factors such as medication use were not controlled for as many studies deemed it unethical to ask participants to stop their pain management regime to participate.

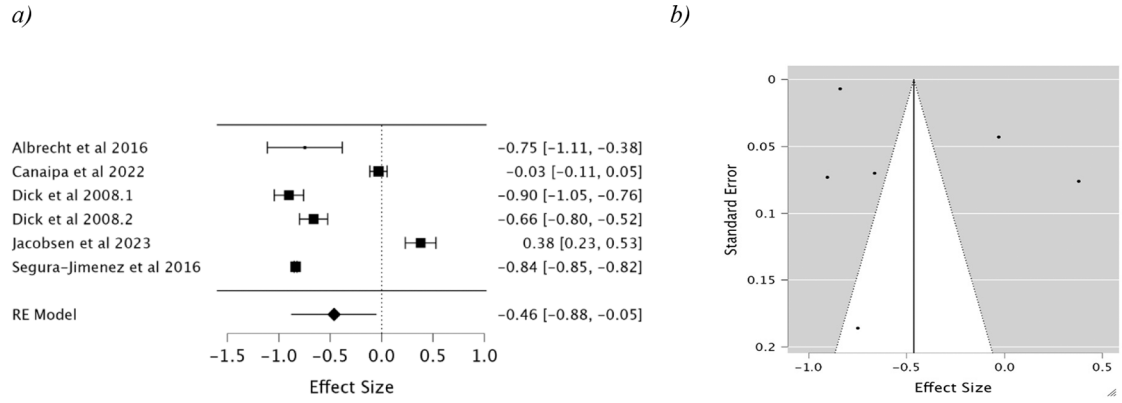
From a statistical standpoint, it is also important that due to the relatively small number of studies meeting our inclusion criteria, not all *meta*-analyses were able to be conducted, particularly for working memory and long-term memory. Furthermore, memory performance based on the modality of information (e.g., visual, verbal) was unable to be explored due to there not being enough studies ($n < 3$). While the findings highlight how different types of pain perform relative to controls, we were unable to glean an understanding of how they perform relative to each other.

Despite these limitations, the results of this research highlight a core finding, that memory performance does differ based on the type of pain one experiences. This finding paves the way for future research in a multitude of ways. Firstly, it highlights the importance of investigating cognitive performance based on the type of pain one experiences. This symptom-based approach (rather than one based on disease type) may provide researchers with a better understanding of the ongoing effects of chronic pain regardless of the specific condition with which patients are (or are not) diagnosed. Secondly, it highlights the importance of



Note RE Model = Random Effects Model, negative effect sizes are indicative of poorer working memory performance in the neuropathic pain group compared to healthy controls*

Fig. 5. A) forest plot comparing working memory scores between people with neuropathic pain and healthy controls b) funnel plot of publication bias note* re model = Random Effects Model, negative effect sizes are indicative of poorer working memory performance in the neuropathic pain group compared to healthy controls.



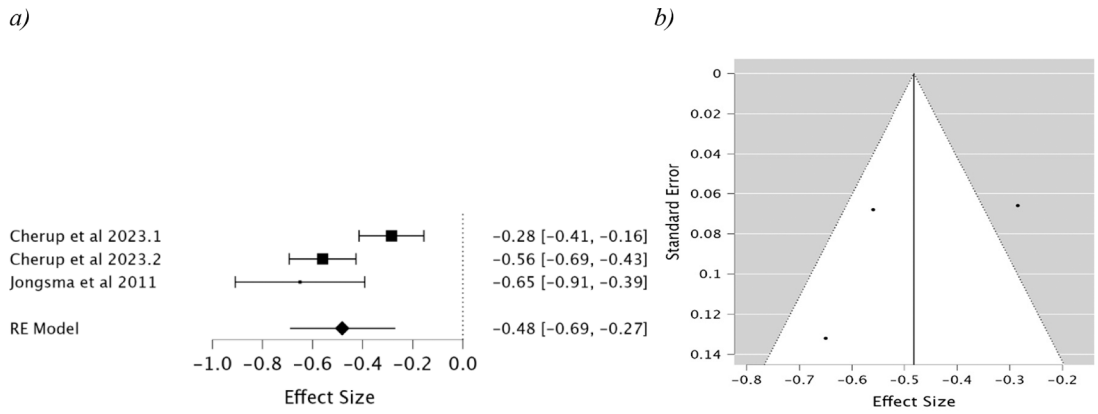
Note RE Model = Random Effects Model, negative effect sizes are indicative of poorer working memory performance in the nociplastic pain group compared to healthy controls*

Fig. 6. A) forest plot comparing working memory scores between people with Nociplastic Pain and Healthy Controls b) Funnel Plot of Publication Bias Note* RE Model = Random Effects Model, negative effect sizes are indicative of poorer working memory performance in the nociplastic pain group compared to healthy controls.

rethinking how we conceptualise pain for the purpose of research. Much research still considers acute pain and chronic pain as the two core subtypes, but given the increasing prevalence of chronic pain, developing a deeper understanding of this category, perhaps with respect to the three pain types referred to here, may result in more accurate findings and conclusions. Finally, further research exploring the secondary symptoms of chronic pain are crucial to effective ongoing pain management. While pain management is moving from a strictly pharmacological approach to a more holistic approach, without detailed research on the secondary impacts of pain, how these vary, how various symptoms interact (e.g. pain and fatigue) and what impacts are most associated with specific pain types, these management strategies may be at limited.

4.4. Conclusions and implications

In summary, the current systematic review and *meta*-analysis sought to better understand the impact that chronic pain type has on the various components of memory. Meta-analyses highlighted that individuals with nociceptive and nociplastic pain demonstrated impaired memory function when compared to controls, whereas those with neuropathic pain did not. This held true for short term and working memory. While the impact of neuropathic pain on long term memory was unable to be evaluated, nociceptive pain was and followed the same pattern. Evaluating more nuanced aspects of memory, such as information modality, were beyond the scope of the included research, but highlight an important consideration for future research.



Note RE Model = Random Effects Model, negative effect sizes are indicative of poorer verbal long term memory performance in the nociceptive pain group compared to healthy controls*

Fig. 7. A) forest plot comparing verbal ltm scores between people with nociceptive pain and healthy controls b) funnel plot of publication bias note* re model = Random Effects Model, negative effect sizes are indicative of poorer verbal long term memory performance in the nociceptive pain group compared to healthy controls.

From a clinical practice perspective, these results highlight the importance of understanding the type of pain an individual is experiencing with consideration for how it may impact their cognition. Providing information in short, simple language may be more supportive for individuals with nociceptive and nociplastic pain who, in this study, have shown to have impaired short term and working memory. Similarly, encouraging patients to bring a support person, write or record information, or provide information in way that they can access it again (e.g. pamphlet) may aid in better supporting individuals. Furthermore, acknowledging the challenges they may have around routinely taking medication, completing exercises or set tasks may be the result of cognitive deficits rather than noncompliance. Not all chronic pain is equal; those considering appropriate interventions and management strategies need to understand this, so that they can treat pain effectively both from a pharmacological perspective but also from a holistic perspective, including by facilitating support for patients from other allied health professionals such as psychologists and physiotherapists. While these findings evidence differences that are present in memory function, the interconnected nature of cognitive processes indicates that different pain types might also have varied impacts on cognition more generally. While we cannot draw these conclusions from the present review, we can highlight the importance of considering this while further research is hopefully being undertaken.

From a research perspective, this review also highlights the importance of developing a better understanding of the different types of pain, both in terms of anatomical pathways and activation and in terms of behavioural impacts such as how it influences cognition. The notion that 'pain happens in the brain' remains true, but with so many complexities and interconnections more research investigating the pathways and impacts is warranted. Reconceptualising chronic pain-based research to undertake a symptom-based approach alongside a disease-based approach will allow for a more in-depth understanding of both how specific symptoms impact the individual, as well as how the symptom cluster associated with the disease does. This research does not suggest prior/current research is inaccurate, but rather that symptom-based/pain-based research in conjunction with current research will aid in developing a deeper understanding of the impacts associated with chronic pain. Ultimately, this will allow for a more detailed understanding of how living with chronic pain impacts the body both in terms of physical health and psychosocial wellbeing.

Statements and declarations

The authors declare this is a unique submission that has not been submitted or considered for publication elsewhere.

Supplementary Information

The online version contains all relevant information

Ethics Approval Statement

As this paper is a systematic review/meta analysis ethics was not required

Patient Consent Statement

As this paper is a systematic review/meta analysis consent was not required.

Permission to Reproduce Material From Other Sources

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CRediT authorship contribution statement

Kate Kelly: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Resources, Project administration,

Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Emily Keohane:** Writing – review & editing, Validation, Project administration, Methodology, Data curation. **Gemma Davy:** Writing – review & editing, Validation, Project administration, Methodology, Investigation, Formal analysis, Data curation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

A link to the data file is in the declarations section of the title page

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