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Pain neuroscience education in persistent painful tendinopathies: A scoping review from the Tendon PNE Network

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ABSTRACT

Objective: to conduct and report a scoping review of the available evidence of the effects and content of pain neuroscience education for patients with persistent painful tendinopathies.
Methods: PubMed, Embase, Web of Science, CINAHL, SPORTDiscus, and grey literature databases were searched from database inception to May 2022. Randomised and non-randomised controlled trials, non-controlled clinical trials, cohort studies, case series, case studies including people with persistent painful tendinopathy aged ≥18 years, a pain education intervention, and in English were included. Studies were excluded if they were cross-sectional studies, reviews, editorials, abstracts, or full-text not available or if included heterogeneous study cohorts, patients with tendon rupture, or patients with systemic diseases.
Results: five studies (n = 164) were included. Pain neuroscience education entailed face-to-face discussion sessions or educational materials including videos, brochures, paper drawings, and review questions. All studies used pain neuroscience education in conjunction with other interventions, obtaining significant benefits in outcomes related to pain, physical performance, or self-reported function, among others.
Conclusions: The application of pain neuroscience education in conjunction with other interventions seemed to improve several outcomes. However, considering the current knowledge about tendon pain and the scarcity of well-designed trials studying pain neuroscience education in tendinopathy, additional research is needed.

1. Introduction

Tendinopathy was agreed in the International Scientific Tendinopathy Symposium Consensus (ICON 2019) as the preferred term to describe persistent tendon pain and loss of function related to mechanical loading (Scott et al., 2020). Traditional clinical signs for tendinopathy such as localised pressure pain associated with tendon loading were seen as suggestive for nociceptive pain (Rio et al., 2014; Smart et al., 2010). However, imaging showed that noticeable alterations in tendon tissue morphology is not consistently related to clinical symptoms (Corrigan et al., 2018); tendon changes can be present in absence of pain and pain can be present in absence of tendon changes (Corrigan et al., 2018, 2020; de Jonge et al., 2015; Gisslèn et al., 2005; Rio et al., 2014). Additionally, even though some potential nociceptive mediators have been identified in tendinopathy (Millar et al., 2021), the nociceptive driver and the pathways involved in pain perception are not fully understood (Cardoso et al., 2019). Thus, persistent painful tendinopathies do not seem to be fully explained by local tendon damage

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and/or inflammation (Plinsinga et al., 2015; Rio et al., 2014).

Different hypotheses have been suggested trying to explain the overlapping processes related to the persistence of pain in tendinopathies. Among them, peripheral and central sensitisation, considered the major underlying mechanism of a third mechanistic pain descriptor known as nociplastic pain (Kosek et al., 2021), are also suggested as a complementary persistent pain mechanism also seen in patients with tendinopathy (Jayaseelan et al., 2019; Plinsinga et al., 2015; Rio et al., 2014). Central sensitisation has been defined in different ways, including the definition by the International Association for the Study of Pain (IASP) as "an increased responsiveness of nociceptive neurons in the central nervous system to their normal or subthreshold afferent input" (Merskey et al., 1994). However, defining it as "an amplification of neural signalling within the central nervous system that elicits pain hypersensitivity" (Woolf, 2011) may be more useful from the perspective of studying this phenomenon in humans (Nijs et al., 2023). In any case, this term must be understood as an umbrella term that covers different mechanisms often related to biopsychosocial factors (Nijs et al., 2023). Central and peripheral nervous system alterations that may be associated with central sensitisation may include altered sensory processing in the brain, showing increased activity to some mechanical stimuli but also non-musculoskeletal stimuli such as light, sound, chemicals or stress, among others (van Ettinger-Veenstra et al., 2019). This altered sensory processing in the brain is also thought to be responsible for nociceptive facilitatory pathways (Bosma et al., 2016; Staud et al., 2008) and an attenuation of endogenous analgesia (Yarnitsky, 2010). In this regard, some studies found altered somatosensory profiles (i.e., symptoms of nervous system sensitisation) in people with persistent tendinopathy (Eckenrode et al., 2019; Heales et al., 2014; Plinsinga et al., 2021; Tompra et al., 2016; Vallance et al., 2021; van Wilgen et al., 2013). However, such signs of potential sensitisation are inconsistent among different studies and tendon locations (Nijs et al., 2021; Plinsinga et al., 2018). Some reviews have previously analysed sensory changes in patients with tendinopathy, finding that hyperalgesia can be present mainly locally but also at remote sites, the latter being suggestive of central sensitisation (Heales et al., 2014; Nijs et al., 2021; Plinsinga et al., 2015; Rio et al., 2021). It should be mentioned that most of these conclusions are primarily based on studies of upper-limb tendinopathy, and caution is urged when extrapolating findings to lower-limb locations (Heales et al., 2014; Nijs et al., 2021; Plinsinga et al., 2015; Rio et al., 2021). To understand persistent pain by peripheral and central sensitisation, central mechanisms involved in pain such as perpetuating psychological, behavioural, and social factors are investigated (Mc Auliffe et al., 2022). For instance, fear of movement and pain catastrophising have been observed in people with persistent tendinopathy (Chimenti et al., 2020; Wong et al., 2020). Thus, these signs should be considered potential contributing factors to persistent pain, as well as other patient characteristics like illness perceptions about pain and tendinopathy, anxiety, depression, stress, obesity, diabetes, motor alterations, or unhealthy behaviours and lifestyles (Belley et al., 2018; Edgar et al., 2022; Franceschi et al., 2014; Fredberg & Stengaard-Pedersen, 2008; Lui, 2017; van Wilgen et al., 2010).

Education together with exercise and load management are considered the cornerstones of managing tendinopathy (Beyer et al., 2015; Cardoso et al., 2019; Kongsgaard et al., 2009; Littlewood et al., 2015; Millar et al., 2021; Silbernagel et al., 2007a). Traditionally, different narratives have been proposed to explain the mechanisms of the tendon interventions through structural changes, in tendon and muscle, or a possible reversal of pathological processes (Malliaras, 2017), but the scientific literature shows that muscle dysfunction (Silbernagel et al., 2007b) and pathological changes in tendon (Drew et al., 2014) may remain even after resolution of the symptoms. Non-tissue mechanisms such as reduced pain-related fear and increased self-efficacy, along with a reconceptualisation of the perceptions of pain or a simple increase in tolerance to tendon load, might also be involved (Chester et al., 2018; Malliaras, 2017). Thus, education may collaborate in these mechanisms through the management of expectations of a quick fix, a decrease of anxiety related to imaging findings, reducing the confusion caused by the abundance of treatment options, or improving adherence in an exercise-based rehabilitation programme (Cardoso et al., 2019).

In this context, we should question ourselves what do we educate in patients with tendinopathy? Education interventions are mainly based on a biomedical model (Coombes et al., 2015; Mellor et al., 2018). The question is if traditional interventions including biomedical education are useful for all patients and if in some cases it can even serve as nocebo as it might increase fearful beliefs or treat. Although a high percentage of patients improve significantly with the current management options (Beyer et al., 2015; Mellor et al., 2018), still some studies reported that between 10 and 58% of patients (72% in the case of some locations such as insertional Achilles tendinopathy (Chimenti et al., 2017)) are not satisfied with treatment outcomes after the intervention (Beyer et al., 2015; Kongsgaard et al., 2009; Mellor et al., 2018; Peterson et al., 2014). It is theorised that patients with nociplastic pain, implying exclusion of dominant neuropathic and nociceptive pain (Kosek et al., 2021), may be approached from a biopsychosocial perspective by a different education namely pain neuroscience education, also known as explain pain (Butler & Moseley, 2013) or therapeutic neuroscience education (Louw et al., 2016), and that it is being studied in different populations (Louw et al., 2016). Additionally, even within the spectrum of dominant nociceptive tendinopathies, maladaptive beliefs and cognitions can be present and increase pain and provide a rationale for using pain neuroscience education. Within the framework of the pain neuroscience education, pain is related to neurophysiological changes and patients are educated about psychosocial and lifestyle factors, that have been suggested as a potential cause of suboptimal rehabilitation outcomes (Chester et al., 2018), may be addressed (Malliaras et al., 2015; Nijs et al., 2011, 2013).

In general, pain neuroscience education consists of an educational intervention that explains the complex biological and physiological processes involved in pain experience to patients in an easy-to-understand way (Louw et al., 2021). Pain neuroscience education uses metaphors, examples, or graphic resources such as videos or pictures, with the aim of modifying the patient's beliefs regarding pain and reducing its threat (Louw et al., 2021). In this way, pain neuroscience education aims to reduce the patient's fear and change inadequate perception of the relevance of issues related to anatomical structures when their predominance in the pain experience is low (Louw et al., 2016). This kind of intervention changes patient's perception about potential tissue damage and decreases attention on the painful side output of the brain. Therefore, it may lead to the willingness to perform physical activity with progression toward feared or avoided movements and loads, increasing activity and exercise tolerability (Malfliet et al., 2018). However, although pain neuroscience education is supported for musculoskeletal pain (Louw et al., 2016) and some guidelines have been developed for other entities (Nijs et al., 2019; Zimney et al., 2014), few studies have examined the use of pain neuroscience education in tendinopathy. Tendinopathy can have a nociceptive cause, but the intensity of the pain is also related to behavioural, psychological, and social factors. Within patients with tendinopathy, the influence of these factors differs, and it is important to individualise these explanations to the patient's context. The available evidence regarding pain neuroscience education in patients with tendinopathies has not been reviewed. Likewise, clinicians willing to provide pain neuroscience education to patients with tendinopathies are currently left without practical guidelines.

The aim of this paper is conducting and reporting a scoping review of the available evidence of the effects and content of pain neuroscience education for patients with persistent painful tendinopathies.

2. Methods

The current article encompasses a scoping review undertaken following the Preferred Reporting Items for Systematic Reviews and

Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) guidelines (Tricco et al., 2018) and prospectively registered in PROSPERO (CRD42021289628). The scoping review format allow the researchers to assess the extent, variety, and nature of the research activity available on a little-studied topic such as the application of pain neuroscience education in tendinopathy, as well as identify gaps in the literature (Tricco et al., 2018). Unlike other types of reviews, scoping review favours to address broader topics where heterogeneous study designs might be applicable.

2.1. Search methods and eligibility criteria

Pubmed, Embase, Web of Science, CINAHL, and SPORTDiscus databases were searched on May 6, 2022. The following search terms relating to pain neuroscience education and tendinopathy were combined for the search: "education AND (tendinopathy OR tendinitis OR tendinosis OR tendonitis OR bursitis OR "tendon pain")". Additionally, reference lists, existing networks, and key journals were hand-searched to identify studies that have been missed. Grey literature databases (New York Academy of Medicine Grey Literature Report and Google Scholar), clinical trial registries, conference papers, and thesis and dissertations repositories were explored to identify relevant unpublished data. The corresponding authors of the identified study protocols were contacted to obtain information on the current state of the research and the availability of results. Detailed information about the search strategy is available in the Supplementary Material 1.

2.2. Eligibility criteria

- **Inclusion criteria**
  - ○ Randomised controlled trials, non-randomised controlled trials, non-controlled clinical trials, cohort studies, case series, and case studies, including study protocols with available results
  - ○ Including people with persistent tendinopathy according to the tendinopathy definition of the International Scientific Tendinopathy Symposium Consensus (Scott et al., 2020).
  - ○ Participants ≥18 years
  - ○ Including a pain education intervention (in isolation or with other interventions, except with surgery). Those studies that introduced at least one alternative explanation to structure as a source of pain based on pain neuroscience (e.g. pain is not equal to harm) were included as pain neuroscience education interventions, regardless of whether they called it pain neuroscience education or not and whether or not it was integrated with another type of education.
  - ○ Published in English
- **Exclusion criteria**
  - ○ Cross-sectional study designs, systematic/narrative reviews, editorials, abstracts, or full-text not available
  - ○ Heterogeneous study cohorts (e.g. tendinopathies and osteoarthritis) that do not separate the results
  - ○ Studies including patients with tendon rupture
  - ○ Studies including an education intervention that does not include pain education
  - ○ Studies including patients with systemic diseases such as rheumatic arthritis or diabetes mellitus.

2.3. Study selection

Identified records were imported to the online Covidence software (https://www.covidence.org/) to remove duplicates automatically. The remaining results were imported to Rayyan (https://rayyan.ai/) for additional manual deduplication. Then, titles and abstracts were reviewed for identifying potentially eligible studies.

2.4. Data extraction and risk of bias assessment

The full-texts of the remaining studies were retrieved and analysed using an extraction form. The data extraction included details on the publication (author and year of publication), region, setting, sample size, participants characteristics (age, sex, body mass index), tendinopathy location, duration of symptoms, intervention and control groups if any, follow-up times, outcome measurements and results for the outcomes of interest, which are: pain, patient self-reported, function and performance, catastrophising, fear-avoidance, quality of life, and beliefs or perceptions.

The risk of bias of the included randomised controlled trials was assessed using the Cochrane Collaboration's Tool (Higgins et al., 2011). Non-randomised controlled trials and uncontrolled clinical trials were assessed using the Methodological Index for Non-Randomised Studies (MINORS) (Slim et al., 2003).

2.5. Data synthesis and analysis

Studied outcomes were reported using the post-intervention within-group and the post-intervention between-group effect sizes (Cohen's d) (Cohen, 1988) to report on the effect of pain neuroscience education in tendinopathy. For the analysis of the content of interventions, details on the pain neuroscience education interventions were extracted and described as per the TiDier guidelines (Hoffmann et al., 2014). All corresponding authors were contacted to provide additional details on the pain neuroscience education interventions.

The effectiveness of pain neuroscience education was assessed by using means and standard deviation (SD) of baseline and post-intervention measurements. The within and between-group effect size were analysed to quantify the effectiveness of the intervention in the one study that included control for pain neuroscience education intervention (Chimenti et al., 2023). In the rest of studies (Belley et al., 2018; Hasani et al., 2021; Jayaseelan et al., 2019; Sancho et al., 2019), the within-group Cohen's d was used as the effect size measure to quantify the effectiveness of the interventions (Cohen, 1988). The within-group changes were considered as the mean difference between the post-intervention assessment and the baseline measurement. Thus, the within-group Cohen's d was calculated as $\frac{\text{PostIntervention}-\text{Baseline}}{\text{SDpooled}}$, where SD_{pooled} was calculated as $\sqrt{\frac{SDBaseline^2+SDPostIntervention^2}{2}}$. The Cohen's d between groups were calculated using the difference between the intervention and control group means after the intervention because all studies reported the mean and the SD of each group at baseline and after the intervention. This analysis was carried out because it was not possible to assign the SD of the within-group differences to calculate Cohen's d between groups. Thus, the Cohen's d between groups was calculated as $\frac{\text{PostIntervention B}-\text{PostIntervention A}}{\text{SDpooled}}$. A negative value of Cohen's d in the tables reflects a reduction in the values of the outcome. Resulting effect sizes were classified into four levels: trivial ($d < 0.2$); small ($d \geq 0.2$); medium ($d \geq 0.5$); or large ($d \geq 0.8$) (for the magnitude of the effect size, all Cohen's d values are interpreted as positive (Cohen, 1988). The level of significance was established at 0.05. The presence or absence of significance was retrieved from the full-texts when available. One study did not report data about significance (Hasani et al., 2021). In the case of case reports, the percentage of change was calculated as a measure of effectiveness.

3. Results

In total, 5766 references were retrieved through all databases, of which 4849 remained after deleting duplicates. After reading the title and abstract, 139 full-text studies were identified. Reading the full-texts allowed 135 studies to be discarded because they did not meet the selection criteria (listed in Supplementary Material 2). A study protocol

(Post et al., 2020) (ClinicalTrials.gov ID: NCT04059146) was included as it was a finished study whose primary data were shared by the authors via email. The completed study was published during the preparation of this manuscript. Therefore, the protocol reference has been replaced by the definitive article reference (Chimenti et al., 2023). An additional study protocol was identified in clinical trial registries (NCT04985370) but was discarded after contacting the authors because it was a student work that had hardly progressed. Therefore, five studies were finally included in the scoping review (Fig. 1).

3.1. Characteristics of the included studies

Table 1 summarises the characteristics and interventions of the included studies. Among the results included in the scoping review, there are two randomised controlled trials (Belley et al., 2018; Chimenti et al., 2023), a randomised feasibility trial (Hasani et al., 2021), a single cohort feasibility study (Sancho et al., 2019), and a case series. Four studies looked at people with Achilles tendinopathy, one at patellar tendinopathy, and another one at rotator cuff. Sample size ranged from 3 to 66 participants.

3.2. Subjects

In total, 164 participants were included, of which the majority had midportion or insertional Achilles tendinopathy (n = 123, 75.0%) (Chimenti et al., 2023; Hasani et al., 2021; Jayaseelan et al., 2019; Sancho et al., 2019), 40 (24.4%) suffered from rotator cuff tendinopathy (Belley et al., 2018), and one participant (0.6%) was diagnosed with patellar tendinopathy (Jayaseelan et al., 2019). The age of the participants included in the different study samples was relatively homogeneous (mean age range 35.7–47) (Table 1). The duration of symptoms criterion ranged from three to 12 months (Table 1).

3.3. Content of pain neuroscience education interventions

All studies used pain neuroscience education in conjunction with a wide range of interventions, including real or sham anodal transcranial direct current stimulation (a-TDCS), sensory motor training, strength training, stretching and manual therapy (Belley et al., 2018), four exercise modalities (high and low intensity with high and low time-under-tension) (Hasani et al., 2021), exercise and general education education (Chimenti et al., 2023; Sancho et al., 2019), and joint mobilisation and aerobic exercise (Jayaseelan et al., 2019).

Four studies introduced face-to-face discussion sessions with additional material in the educational intervention (Belley et al., 2018; Hasani et al., 2021; Jayaseelan et al., 2019; Sancho et al., 2019). The additional educational material included videos (Belley et al., 2018), brochures (Hasani et al., 2021), and paper drawings (Jayaseelan et al., 2019; Sancho et al., 2019). Differently, in one study (Chimenti et al., 2023), patients were provided with material to work with independently

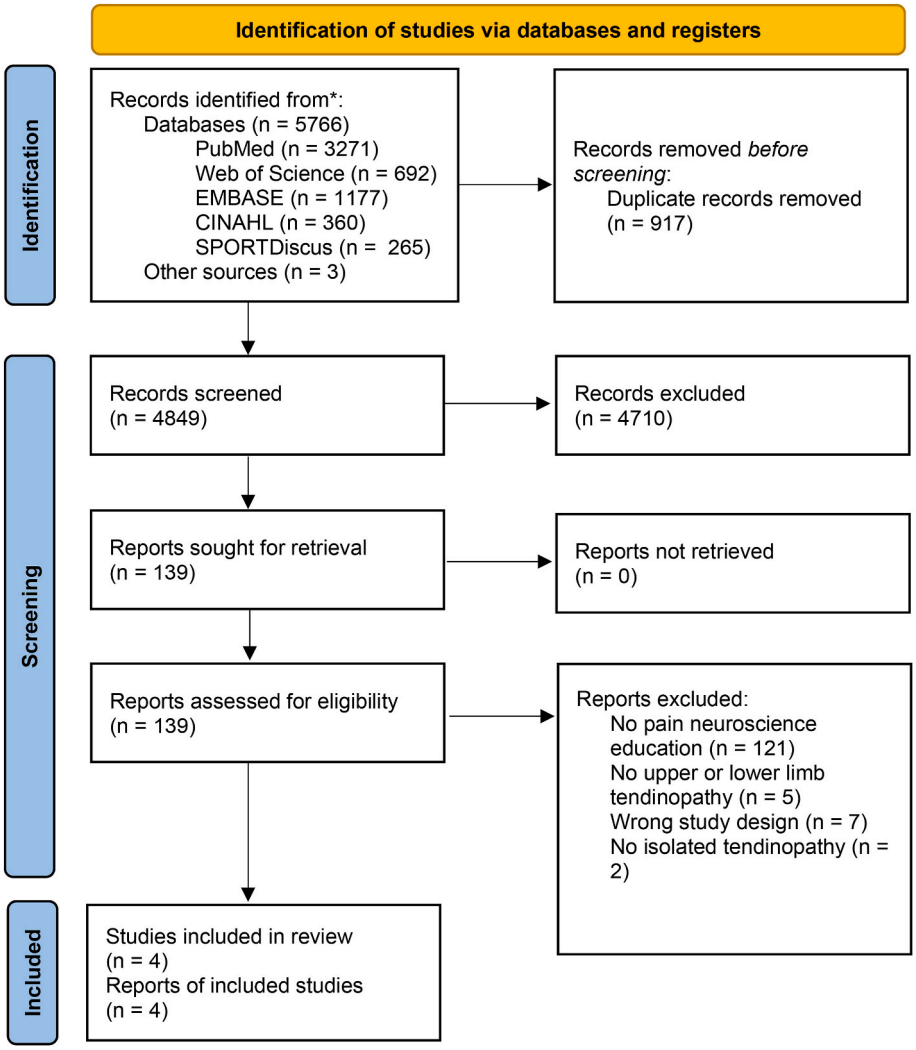

Fig. 1. Flow diagram of the selection process.

Table 1
Summary of the characteristics of the included studies examining pain neuroscience education in patients with tendinopathies.

Author (year)	Study type; N; mean age	Tendon; Duration of symptoms data	Details on interventions
Chimenti et al. (2023) (Chimenti et al., 2023)	RCT protocol (ClinicalTrials.gov ID: NCT04059146); N = 66; 43.4 ± 15.5 years	Midportion (n = 29) and insertional (n = 37) Achilles tendinopathy; At least three months from the onset of symptoms	**Setting:** Hospital (laboratory setting and/or by telehealth) and home **Duration:** 12 weeks **IG:** 8 weeks of pain neuroscience education **CG:** 8 weeks of pathoanatomic education **Both groups**: 8-week face-to-face and/or online exercise programme + 4-week home exercise programme **Adherence:** >90% (100% education) **Fidelity:** 91.5–92.1%
Belley et al. (2018) (Belley et al., 2018)	RCT; N = 40; 47 ± 9 years (IG), 44 ± 11 (CG)	Rotator cuff; Mean symptom duration: 45 weeks (IG: 41 ± 35 weeks; CG: 48 ± 31 weeks)	**Setting:** Research centre and home **Duration:** 6 weeks **IG:** Real a-tDCS; **CG:** Sham a-tDCS **Both groups:** Sensory motor training + strength training + patient education (including pain neuroscience education) + stretching and manual therapy **Adherence:** 86–98.7% **Fidelity:** N/A
Hasani et al. (2021) (Hasani et al., 2021)	Randomised feasibility trial (unpowered); N = 40; 43.2 ± 10.4 years	Midportion Achilles; At least three months from the onset of symptoms	**Setting:** Gym (face-to face and online) and university research centre **Duration:** 12 weeks **IG:** High-intensity exercise; **CG:** Low-intensity exercise **Both groups**: Education (including pain neuroscience education) + exercise **Adherence:** 70% **Fidelity:** 50%
Sancho et al. (2019) (Sancho et al., 2019)	Single cohort feasibility study (unpowered); N = 15; 37.86 ± 8.83 years	Midportion Achilles; At least six weeks from the onset of symptoms	**Setting:** Private physiotherapy clinic **Duration:** 12 weeks Education (including pain neuroscience education) + exercise **Adherence:** 49–68% **Fidelity:** 8–83%
Jayaseelan et al. (2019) (Jayaseelan et al., 2019)	Case series N = 3; 35.7 ± 7.77 years	Midportion Achilles (x2) and Patellar; At least 12 months from the onset of symptoms	**Setting:** N/A **Duration:** 8 weeks Pain neuroscience education + joint mobilisation + aerobic exercise **Adherence and fidelity:** N/A

a-TDCS: Anodal Transcranial Direct Current Stimulation; CG: Control group; IG: Intervention group; N/A: Not available.

as the main part of the educational intervention. In this study, the participants were given homework at each session where they had to watch a short video (5–10 min), review a brochure that summarised the content of the video, and complete some review questions (Chimenti et al., 2023). At the following visit, the physical therapist reviewed the responses to the questions and addressed any remaining questions (Chimenti et al., 2023). The videos and educational brochures used in this study can be accessed through Iowa Research Online, available at https://doi.org/10.25820/data.006166. Neuroanatomy, physiology, pain mechanisms, and acceptable degrees of pain were included in the educational content of all the studies. Patients were instructed in the idea that "pain is not equal to harm" and the concept of sensitisation. Pain neuroscience education interventions also included common maladaptive beliefs and maladaptive coping such as fear-avoidance. The pain neuroscience education intervention was accompanied by education in the concept of tendinopathy and load management (progressive loading).

Education including pain neuroscience education was included at the beginning of treatment with long sessions (45–90 min) in two studies (Jayaseelan et al., 2019; Sancho et al., 2019), but not in two others (Belley et al., 2018; Chimenti et al., 2023) which included education in regular sessions with other (non-educational) interventions such as exercise therapy. Pain neuroscience education content was usually included together with general education. The format and content of the educational interventions that included pain neuroscience education content are listed in Table 2.

3.4. Effect of pain neuroscience education interventions

The methodology of the study by Chimenti et al. (2023) (Chimenti et al., 2023), with a pain neuroscience education plus exercise versus pathoanatomic education plus exercise, is the only one that allows adequate analysis of the pain neuroscience education compared to the traditional practice of education and exercise in patients with tendinopathy. Regarding within-group analysis, the main outcomes (self-reported function, pain catastrophising, kinesiophobia, pain and motor function) showed a significant change over time in both groups, with the exception of conditioned pain modulation. This effect occurred at 8 weeks and was maintained at 12-weeks (when 12-week outcome available) (Table 3). Regarding differences between groups, there was only one outcome with significant differences in favour of the pain neuroscience education plus exercise group, which is kinesiophobia at 8 weeks (Cohen's d = −0.37). However, this effect was no longer significant at 12 weeks (Table 4).

In Belley et al. (2018) (Belley et al., 2018) and Sancho et al. (2019) (Sancho et al., 2019), the application of pain neuroscience education together with other interventions reported significant effects in rotator cuff and midportion Achilles tendinopathies, respectively, on variables of self-reported function (Cohen's d = 1.64–2.77), pain (Cohen's d = 2.59–2.65), kinesiophobia (Cohen's d = 0.59), Achilles tendon beliefs (Cohen's d = 0.99), stiffness (Cohen's d = 0.79–1.11), and physical performance (Cohen's d = 0.16–1.21).

Hasani et al. (Hasani et al., 2021) found positive changes in the four intervention groups, combining pain neuroscience education and exercise with different intensities and time-under-tension in midportion Achilles tendinopathy. These changes were observed in self-reported function (Cohen's d = 2.02–3.13), pain (Cohen's d = 0.65–1.09), other self-reported outcomes (quality of life [Cohen's d = 0.37–1.24], pain catastrophising [Cohen's d = 0.6–1.57], kinesiophobia [Cohen's d = 0.84–2.08]), and physical performance outcomes (Cohen's d = 0.29–1.31). However, this study did not report which of these differences were statistically significant as it was a feasibility study.

Jayaseelan et al. (2019) (Jayaseelan et al., 2019) found positive changes in self-reported function (40–102.2%), pain (100%), pain pressure thresholds (122.2-198-2% for the affected tendon, 91.9–147.2% for the contralateral tendon, and 34.2–61.4% for the

Table 2
Format and content of the educational interventions that included pain neuroscience education content.

Author (year)	Education including pain neuroscience education (format and content)
Chimenti et al. (2023) (Chimenti et al., 2023)	**Number and duration of sessions:** 6 homework sessions (of approx. 20 min) and 6 education sessions (of approx. 20 min) within the following intervention sessions **Format:** Video, brochure, review questions, and face-to-face discussion **Education content including pain neuroscience education:** **Common for both groups** Progressive loading exercises for tendinopathy: • Defining the term load for tendon pain rehabilitation • Types of loads placed onto the Achilles tendon during various activities • Use of symptoms 24 h after completion of exercises to inform exercise dosage **Specific for pain education group** Rethinking the role of exercise for AT: • Tendon load capacity and role of exercise to increase capacity • Progressive increase in exercise intensity and duration • Difference between AT and Achilles tendon rupture Common tendon adaptations to loading: • Common tissue adaptations seen on imaging including bone spurs, tendon calcification, and Haglund deformity • Lack of correlation between imaging and clinical presentation Factors influencing pain: • Pain neurobiological processing (nociceptor activity and signal interpretation by the brain) • Impact of psychological factors Understanding pain: • Hypersensitivity of the peripheral and central nervous system and persistent pain • Descending facilitation and inhibition on chronic pain • Multifactoriality of persistent pain Benefits of exercise for chronic musculoskeletal pain: • Neurotransmitters and inflammatory mediators present with persistent pain • Roles of exercise on improving immune system and neurotransmitter function to decrease pain •Physical activity guidelines
Belley et al. (2018) (Belley et al., 2018)	**Number and duration of sessions:** 8 sessions of approximately 10 min (25% of each physiotherapy session consisting of "teaching and revising the home exercises and providing education") **Format:** Video and face-to-face discussion **Education content including pain neuroscience education:** Structures affected Posture, sleeping, activities, work, and sports Shoulder and body mechanics Rehabilitation stages Graded exposure to exercise Pain management Pain neuroscience content
Hasani et al. (2021) (Hasani et al., 2021)	**Number and duration of sessions:** Not specified **Format:** Brochure and face-to-face discussion **Education content including pain neuroscience education:** Tendon pain mechanisms Acceptable levels of pain during exercise and activity
Sancho et al. (2019) (Sancho et al., 2019)	**Number and duration of sessions:** Approximately 90 min at baseline session **Format:** Face-to-face discussion and paper drawings **Education content including pain neuroscience education:** Achilles tendon pathology Risk factors and prognosis Strategies to address common maladaptive beliefs such as fear-avoidance and poor outcomes expectation Potential pain mechanisms
Jayaseelan et al. (2019) (Jayaseelan et al., 2019)	**Number and duration of sessions:** Two sessions (first and second session, at baseline) of approximately 45 min **Format:** Face-to-face discussion and paper drawings **Education content including pain neuroscience education:** Clinical diagnosis and the neurophysiology of pain Tissue injury and pain perception as separate concepts Contributing factors to persistence of pain through top-down sensitisation or bottom-up sensitisation Neuroanatomy including nociceptive pathways, neurons, synapses, action potentials, ascending and descending spinal symptom modulation mechanisms, peripheral and central sensitisation and neuroplasticity

AT: Achilles Tendinopathy.

contralateral hand) and central sensitisation (31.6–40%), combining pain neuroscience education, aerobic exercise, and joint mobilisation, in two cases with midportion Achilles tendinopathy and one case with patellar tendinopathy. The effect sizes (Cohen's d) or percentages of change of the within group analysis of each of the groups that included pain neuroscience education are available in Table 3.

Conflicts of interest of the included studies are reported in the Supplementary Material 3. The results of the risk of bias assessment are available in the Supplementary Material 4 and 5.

4. Discussion

This scoping review provides an overview of the current literature on the content and efficacy of pain neuroscience education in patients with tendinopathies. Five studies were identified, most of them (Belley et al., 2018; Hasani et al., 2021; Jayaseelan et al., 2019; Sancho et al., 2019) using pain neuroscience education in all the study groups and in combination with another type of intervention, a design that did not allow us to clearly assess the effectiveness of pain neuroscience education in patients with tendinopathies, so additional research is needed. The application of pain neuroscience education in conjunction with other interventions seemed to improve several outcomes including to pain, physical performance, self-reported function, pain catastrophising, kinesiophobia, or illness perceptions. Chimenti et al. (2023) (Chimenti et al., 2023) compared a pain neuroscience education intervention versus the traditional pathoanatomical education, in both cases applied with exercise and with similar results in both groups. The pain neuroscience education content was relatively consistent across studies, consisting of neuroanatomy, physiology, pain mechanisms, maladaptive beliefs, and coping strategies ((i.e., cognitive and behavioural attempts to manage or tolerate pain and its effects (Kraaimaat & Evers, 2003)), in sessions usually integrated at the beginning of treatment and with various formats that included face-to-face discussion (Belley et al., 2018; Hasani et al., 2021; Jayaseelan et al., 2019; Sancho et al., 2019) applied with additional material such as videos (Belley et al., 2018), brochures (Hasani et al., 2021), paper drawings (Jayaseelan et al., 2019; Sancho et al., 2019), and review questions (Chimenti et al., 2023).

Table 3
Post-intervention within-group effect size (change over time) of the included studies.

Author (year)	Outcomes and Effect Sizes (Cohen's d) or % of change		
	Within-group		
Chimenti et al., (2023) (Chimenti et al., 2023)	**8 weeks**[¥]		
	Pain (NPRS) during heel raises Pathoanatomic education Cohen's d = −2.10* Pain education Cohen's d = −1.46* **Conditioned Pain Modulation (CPM)** Pathoanatomic education Cohen's d = −0.60 Pain education Cohen's d = −0.27	**Motor function** **Single limb heel raises** Pathoanatomic education Cohen's d = 0.25* Pain education Cohen's d = 0.78*	
	12 weeks		
	Self-reported function (VISA-A) Pathoanatomic education Cohen's d = 1.06* Pain education Cohen's d = 1.28* **Pain catastrophising (PCS)** Pathoanatomic education Cohen's d = −0.28* Pain education Cohen's d = −0.61*	**Kinesiophobia (TAMPA scale)** Pathoanatomic education Cohen's d = −0.96* Pain education Cohen's d = −1.08* **PROMIS: Clinical Outcomes Assessment** Pathoanatomic education Cohen's d = 0.59* Pain education Cohen's d = 0.40*	
Belley et al., (2018) (Belley et al., 2018)	**12 weeks**		
	Self-reported function DASH aTDCS Cohen's d = −2.33* Sham aTDCS Cohen's d = −2.06*	WORC aTDCS Cohen's d = 2.77* Sham aTDCS Cohen's d = 2.22*	
Hasani et al., (2021) (Hasani et al., 2021)	**12 weeks**[‡]		
	Self-reported function (VISA-A) High Load with High TUT Cohen's d = 2.12 High Load with Low TUT Cohen's d = 2.02 Low Load with High TUT Cohen's d = 3.13 Low Load with Low TUT Cohen's d = 2.53 **Pain (NPRS)** High Load with High TUT Cohen's d = −0.65 High Load with Low TUT Cohen's d = −0.94 Low Load with High TUT Cohen's d = −0.97 Low Load with Low TUT Cohen's d = −1.09 **Self-reported Quality of life (EQ-5D-5L Index 0–1)** High Load with High TUT Cohen's d = 1.24 High Load with Low TUT Cohen's d = 1.09 Low Load with High TUT Cohen's d = 1.14 Low Load with Low TUT Cohen's d = 1.18 **Self-reported Quality of life (EQ-5D-5L Overall health status 0–100)** High Load with High TUT Cohen's d = 1.1 High Load with Low TUT Cohen's d = 0.74 Low Load with High TUT Cohen's d = 0.69 Low Load with Low TUT Cohen's d = 0.37 **Physical activity level (7-day Recall Physical Activity Questionnaire)** High Load with High TUT Cohen's d = −0.5 High Load with Low TUT Cohen's d = −0.03	Low Load with High TUT Cohen's d = 0.15 Low Load with Low TUT Cohen's d = −0.1 **Pain catastrophising (PCS)** High Load with High TUT Cohen's d = −1.57 High Load with Low TUT Cohen's d = −0.94 Low Load with High TUT Cohen's d = −0.6 Low Load with Low TUT Cohen's d = −1.07 **Kinesiophobia (TAMPA scale)** High Load with High TUT Cohen's d = −1.26 High Load with Low TUT Cohen's d = −2.08 Low Load with High TUT Cohen's d = −0.86 Low Load with Low TUT Cohen's d = −0.84 **Physical Performance** **Ankle plantarflexion torque (Nm)** High Load with High TUT Cohen's d = 0.97 High Load with Low TUT Cohen's d = 0.48 Low Load with High TUT Cohen's d = 0.29 Low Load with Low TUT Cohen's d = 0.33 **Ankle plantarflexion rated of torque development torque (Nm/s)** High Load with High TUT Cohen's d = 0.99 High Load with Low TUT Cohen's d = 0.38 Low Load with High TUT Cohen's d = 0.32 Low Load with Low TUT Cohen's d = 0.60	**Plantarflexion coefficient of variation of torque** High Load with High TUT Cohen's d = −0.51 High Load with Low TUT Cohen's d = −0.43 Low Load with High TUT Cohen's d = −0.4 Low Load with Low TUT Cohen's d = −0.5 **Mass lifted in seated (kg)** High Load with High TUT Cohen's d = 0.7 High Load with Low TUT Cohen's d = 1.2 Low Load with High TUT Cohen's d = 1.31 Low Load with Low TUT Cohen's d = 1.05 **Mass lifted in standing (kg)** High Load with High TUT Cohen's d = 1.22 High Load with Low TUT Cohen's d = 0.52 Low Load with High TUT Cohen's d = 0.7 Low Load with Low TUT Cohen's d = 0.78
Sancho et al., (2019) (Sancho et al., 2019)	**12 weeks**		
	Self-reported function (VISA-A) Cohen's d = 1.64* **Pain** **Hop VAS** Cohen's d = −2.65* **Hop to fatigue VAS** Cohen's d = −2.59* **Achilles tendon beliefs (ATBQ)** Cohen's d = −0.99* **Pain anxiety symptoms (PASS20)** Cohen's d = −0.36 **Kinesiophobia (TAMPA scale)** Cohen's d = −0.59*	**Physical Performance** **Standing heel raise to fatigue (reps/BW)** Cohen's d = 0.72* **Seated heel raise 6RM (kg/BW)** Cohen's d = 1.21* **Leg Extension 6RM (kg/BW)** Cohen's d = 0.76* **Leg curl 6RM (kg/BW)** Cohen's d = 0.25 **Hip abduction (MVIC)** Cohen's d = 0.16* **Hip extension (MVIC)** Cohen's d = 0.17 **Fatigue duration** Cohen's d = 0.73*	**ROM** Ankle dorsiflexion (straight knee) Cohen's d = −0.15 Ankle dorsiflexion (bent knee) Cohen's d = −0.16 **Stiffness** **Leg stiffness submaximal hop (kN/m)** Cohen's d = 1.11* **Leg stiffness begin fatigue (kN/m)** Cohen's d = 0.79* **Leg stiffness middle fatigue (kN/m)** Cohen's d = 0.65 **Leg stiffness endfatigue (kN/m)** Cohen's d = 0.98*
Jayaseelan et al., (2019) (Jayaseelan et al., 2019)	**8 weeks**		
	Self-reported function (VISA-A or VISA-P) S1 (AT): % of change = 102.2% S2 (AT): % of change = 40.0%		**PPT (IT, CT, CH)** S1 (AT): % of change = IT: 187%; CT: 137.9%; CH: 42.5%

(*continued on next page*)

Table 3 (*continued*)

	S3 (PT): % of change = 71.7%	S2 (AT): % of change = IT: 122.2%; CT: 91.9%; CH: 34.2%
	Pain (NPRS)	S3 (PT): % of change = IT: 198.2%; CT: 147.2%; CH: 61.4%
	S1 (AT): % of change = Rest: 0.0% (baseline = 0); Activity: 100.0% (baseline = 8)	**Central sensitisation (CSI)**
	S2 (AT): % of change = Rest: 0.0% (baseline = 0); Activity: 100.0% (baseline = 7)	S1 (AT): % of change = −36.7%
	S3 (PT): % of change = Rest: 100.0% (baseline = 1); Activity: 100.0% (baseline = 7)	S2 (AT): % of change = −31.6%
		S3 (PT): % of change = −40.0%

¥ Outcomes only measured after the end of the educational intervention plus exercise but not at 12 weeks (after 4 weeks of continuing the home exercise program at home); ‡Significance level not available; *p < 0.05.
APB = Avoiding Pain-Based; AT: Achilles Tendon; ATBQ: Achilles Tendon Beliefs Questionnaire; BW: Body weight; CG: Control group; CH: Contralateral Hand; CSI: Central Sensitisation Inventory; CT: Contralateral Tendon; DASH: Disabilities of the Arm, Shoulder and Hand questionnaire; EQ-5D-5L: EuroQol 5 dimensions 5 Levels questionnaire; HL: High load exercise; IG: Intervention group; IT: Involved Tendon; LL: Low load exercise; NPRS: Numerical Pain Rating Scale; PASS20: Pain Anxiety Symptoms Scale-20; PCS: Pain Catastrophizing Scale; PROMIS CAT: Patient-Reported Outcomes Measurement for pain management self-efficacy, anxiety, and depression; Computerised Adaptive Testing; PPT: Pain Pressure Threshold; PT: Patellar Tendon; RM: Repetition maximum; ROM: Range of Movement; S1: Subject 1; S2: Subject 2; S3: Subject 3; TUT: Time-under-tension; VAS: Visual analogue scale; VISA: Victorian Institute of Sport Assessment; WORC: Western Ontario Rotator Cuff Index.

Table 4
Post-intervention between-group effect size (between-group differences) of the included studies.

Author (year)	Outcomes and Effect Sizes (Cohen's d)	
	Between-group	
Chimenti et al. (2023) (Chimenti et al., 2023)	**8 weeks¥**	
	Pain (NPRS) during heel raises	**Motor function Single limb heel raises**
	Pain education VS Pathoanatomic education	Pain education VS Pathoanatomic education
	Cohen's d = 0.19	Cohen's d = 0.12
	Conditioned Pain Modulation (CPM)	
	Pain education VS Pathoanatomic education	
	Cohen's d = 0.18	
	12 weeks	
	Self-reported function (VISA-A)	**Kinesiophobia (TAMPA scale)**
	Pain education VS Pathoanatomic education	Pain education VS Pathoanatomic education
	Cohen's d = 0.15	Cohen's d = −0.37
	Pain catastrophising (PCS)	**PROMIS: Clinical Outcomes Assessment**
	Pain education VS Pathoanatomic education	Pain education VS Pathoanatomic education
	Cohen's d = 0.28	Cohen's d = 0.16

¥Outcomes only measured after the end of the educational intervention plus exercise but not at 12 weeks (after 4 weeks of continuing the home exercise program at home).

4.1. Contrast of our findings with other literature

In the current review, pain neuroscience education in combination with other interventions resulted in favourable outcomes but the only study that compared it with other type of education did not find any significant difference between groups. The use of pain neuroscience education as an adjunct to other interventions is in line with the current recommendations on pain neuroscience education. Pain neuroscience education has shown clinically significant effects on some outcomes in other populations, but when applied as a stand-alone treatment it has minimal effects on chronic pain relief (Louw et al., 2021). Thus, it has been suggested that pain neuroscience education should not be applied as a stand-alone intervention, but as a complementary intervention to promote exercise and movement (Louw et al., 2021). Indeed, pain neuroscience education primarily intends breaking down cognitive-emotional barriers for more active parts of the treatment plan, such as exercise therapy, but also increasing activity through daily routines and activities, work, or socialising (Louw et al., 2021).

The results of our review are also consistent with the findings of the review by Louw et al. (Louw et al., 2016) who found beneficial effects of pain neuroscience education in combination with other treatments in reducing pain, disability, pain catastrophism, fear-avoidance, and movement limitations, modifying unhealthy and maladaptive behaviours, and limiting healthcare utilisation in other musculoskeletal disorders (chronic low back pain, chronic fatigue syndrome, fibromyalgia, lumbar radiculopathy, or chronic neck pain). A recent review with four studies in patients with osteoarthritis also found a reduction in pain when pain neuroscience education was applied compared to other interventions and other educational approaches (Ordoñez-Mora et al., 2022). Likewise, in this review, differences in kinesiophobia were also found in favour of the groups with pain neuroscience education in one study (Ordoñez-Mora et al., 2022).

Regarding the findings of the comparison between pain neuroscience education and traditional pathoanatomical education, one of the studies included in our review (Chimenti et al., 2023) found no differences in the main outcomes between these two types of education, both applied together with exercise. Different studies included in the review by Louw et al. (Louw et al., 2016) compared the application of pain neuroscience education versus other educational intervention (pathoanatomical education (Moseley, 2004), pacing self-management education (Van Oosterwijck et al., 2013), education on relaxation exercise (van Ittersum et al., 2014), or traditional pre-operative education (Louw et al., 2014)) in chronic low back pain (Moseley, 2004), fibromyalgia (van Ittersum et al., 2014), and lumbar radiculopathy (Louw et al., 2014). In line with our results, few or no differences were observed between the different types of education in variables of pain, function or pain catastrophising, although some benefits were observed in the patient's illness perception, the endogenous pain inhibition, and the use of healthcare utilisation (Louw et al., 2014; Moseley, 2004; van Ittersum et al., 2014; Van Oosterwijck et al., 2013). Possible interpretations of these findings are discussed in the following section.

Is pain neuroscience education necessary in patients with tendinopathy?

Despite high level of evidence for exercise treatment for tendinopathies, 10–72% of patients are not satisfied with current treatments (Beyer et al., 2015; Chimenti et al., 2017; Kongsgaard et al., 2009; Mellor et al., 2018; Peterson et al., 2014), and 5–15 years after treatment 20–60% of patients report not being fully recovered (Kettunen et al., 2002; Silbernagel et al., 2011; van der Plas et al., 2012). It has been proposed that the additional use of pain neuroscience education could improve the outcome in patients with persistent tendon pain and failed conservative treatments (Jayaseelan et al., 2019).

Previous studies (Hanlon et al., 2021, 2023) suggested the existence of four subgroups in Achilles tendinopathy, described as activity dominant, function dominant, structure dominant, and psychosocial dominant. These findings underscore that patients with tendinopathy are different and respond differently to their injury. Patients in the

psychosocial dominant subgroup had greater degree of kinesiophobia and pain catastrophising, worse quality of life, and lower functional scores compared to the other subgroups, along with minimal structural changes (Hanlon et al., 2021, 2023). Thus, these findings could be explained by the relationship between kinesiophobia and pain catastrophism with load avoidance behaviours, which may affect the execution of exercise programmes and limit the effect of the interventions (Hanlon et al., 2021). Pain neuroscience education has been suggested to improve self-efficacy and management of symptoms and decreasing pain catastrophising and fear of movement in chronic pain patients (Watson et al., 2019). Therefore, including pain neuroscience education may help to reeducate possible maladaptive beliefs and cognitions and reducing pain avoidance behaviours and other maladaptive coping mechanisms that may persist despite the resolution of the initial real or potential damage that caused it (Baliki & Apkarian, 2015; Brodal, 2017; Fenton et al., 2015).

In our review, the findings of Chimenti et al. (2023) (Chimenti et al., 2023) adds to the discussion about the need (or not) to apply pain neuroscience education to all patients with tendinopathy. The results of both groups (pathoanatomic and pain education) seem to indicate the appropriateness of including an educational intervention in patients with tendinopathy. These findings are consistent with evidence in other musculoskeletal conditions (with characteristics different from tendinopathy that could limit the comparison of the results) such as chronic low back pain, chronic fatigue syndrome, fibromyalgia, lumbar radiculopathy, or chronic neck pain, supporting the use of pain neuroscience education in reducing pain, improving function, decreasing disability, and reducing psychosocial factors (Louw et al., 2016). However, the lack of differences between groups (except in kinesiophobia at 8 weeks) could indicate that in most patients with tendinopathy an educational intervention from a biomedical point of view is sufficient to achieve the desired changes, since both different approaches could share most of their action mechanisms, including providing reassurance. However, as the authors suggest, another possible cause for the lack of differences is the intervention used in the biomedical education group (Chimenti et al., 2023). Thus, although negative approaches exist in real-world clinical settings (which are, for example, a clear source of nocebo or fear of movement), for ethical reasons an evidence-filtered biomedical approach avoiding those clear sources of nocebo was used, so the potential differences between both models could have been underestimated (Chimenti et al., 2023).

These findings may also suggest the need for a previous screening that allows researchers and clinicians to be specific in the selection of responders. In this regard, as has been suggested, it might be difficult to find differences in outcome of using pain neuroscience education or not if all patients with a specific diagnosis are included and the individual need of pain neuroscience education is not considered (Hanlon et al., 2021). Thus, the development of screening tools that can identify those patients who require pain neuroscience education may be of importance for professionals who deal with patients with tendinopathy.

It may also be argued that if pain neuroscience education does not have a negative effect on this population, all interventions should include pain neuroscience education to ensure reaching those responders who may escape screening. A previous review found no outcome to be worse in those interventions including pain neuroscience education compared to the control interventions when pain neuroscience education was applied in other musculoskeletal disorders (chronic low back pain, chronic fatigue syndrome, fibromyalgia, lumbar radiculopathy, or chronic neck pain), which may imply a lack of risk versus potential benefits (Louw et al., 2016). A similar interpretation could be drawn in tendinopathy from the lack of differences between the biomedical education and the pain neuroscience education groups of the Chimenti's study (Chimenti et al., 2023), suggesting that clinicians should not be concerned that treatment outcomes will be lowered by the application of pain neuroscience education in non-responders. However, this should be explored from other perspectives, since, as reported in a recent study, some people with persistent pain tend to express negative attitudes towards pain science education (Weisman et al., 2022). This could result in a counterproductive misalignment between therapists' beliefs and perceptions with those of these reluctant patients. For this reason, the patient's understanding of the intervention rationale must be guaranteed in advance to achieve the therapeutic alliance (Weisman et al., 2022).

4.2. Format and content of pain neuroscience education interventions

The pain neuroscience education content was relatively consistent across studies, consisting of neuroanatomy, physiology, pain mechanisms, maladaptive beliefs, and coping strategies. Likewise, all interventions instructed patients in the idea that "pain is not equal to harm" and the concept of central and peripheral sensitisation, especially important in case of nociplastic pain. Some authors included content related to maladaptive beliefs and maladaptive coping such as fear-avoidance, which may be especially relevant both in patients with nociplastic pain and in patients with dominantly nociceptive pain but who present maladaptive beliefs and cognitions. These pain neuroscience education interventions were accompanied by tendinopathy-specific and load management content.

Additionally, pain neuroscience education interventions may also include concepts such as descending nociceptive inhibition and facilitation, neuroplasticity, contributing factors in the pain experience such as the behavioural, psychological and social factors, or other concepts related to complex issues associated with persistent pain (Louw et al., 2016; Moseley, 2003; Nijs et al., 2013).

In the current literature, pain neuroscience education is applied predominantly through face-to-face discussion (Belley et al., 2018; Hasani et al., 2021; Jayaseelan et al., 2019; Sancho et al., 2019). This format promotes a dialogue based on questions and answers to extract underlying presuppositions and to develop critical thinking. The use of additional material (videos, brochures, paper drawings, and review questions) may favour the acquisition of content. The inclusion of educational sessions at the beginning of treatment (e.g., through long sessions (Jayaseelan et al., 2019; Sancho et al., 2019)) may be used to early achieve an adequate adherence and a knowledge base that encourages movement and exercise. However, the content of these initial educational sessions can also be integrated into shorter sessions combined with other non-educational interventions such as exercise throughout the programme (Belley et al., 2018; Chimenti et al., 2023). In a different way, the proposal of a study (Chimenti et al., 2023) to provide patients with material to work at home as the main part of the educational intervention is novel. In this way, an active educational learning strategy is used in which the patient acquires knowledge using material such as videos and brochures, after which they complete some review questions. In this way, the therapist can adapt the intervention in an individualised way, delving into those aspects that the patient refers to as doubtful in the subsequent face-to-face sessions.

In any case, and importantly, a comprehensive assessment may be necessary to identify the patient's needs and to tailor the pain neuroscience education content and format accordingly (Wijma et al., 2016). Subsequently, this individualisation may also favour the patient's willingness to receive this type of intervention and promote the therapeutic alliance.

5. Limitations

The literature available includes only four locations of tendinopathy: Achilles (midportion and insertional), rotator cuff and patellar tendons. Although these locations are some of the most frequent in the population, it also means that we do not have literature on the use of pain neuroscience education in a large number of tendons with diverse characteristics. Another limitation of the study was the difficulty in analysing the effectiveness of the pain neuroscience education due to the

methodology used in the available studies. Pain neuroscience education was always used in conjunction with other interventions. Additionally, in four studies (Belley et al., 2018; Hasani et al., 2021; Jayaseelan et al., 2019; Sancho et al., 2019), the pain neuroscience education was applied in all study groups, cohorts and cases, lacking a control group, so this intervention was not controlled. This fact, together with the methodological weaknesses associated with some of the study designs, limits the extraction of results regarding the effectiveness of pain neuroscience education in patients with tendinopathy.

6. Conclusions

The methodology of the available studies precludes adequate analysis of the effectiveness of the pain neuroscience education in patients with tendinopathy. The application of pain neuroscience education together with other interventions in patients with tendinopathy seems to be related to an improvement in outcomes such as self-reported function, illness perceptions about cause, pain catastrophising, kinesiophobia, pain, and physical performance. There is no overwhelming evidence that pain neuroscience education is better than any education. Nevertheless, current knowledge about the existence of subgroups in tendinopathy may suggest the need for prior clinical and research screening to identify those subjects with tendinopathy who require pain neuroscience education, as the available studies might not have found a difference in the tendinopathy population as a whole, even though pain neuroscience education was necessary for some. Considering the characteristics of tendon pain, and the scarcity of studies that allow a clear analysis of the application of pain neuroscience education in this population, additional research is needed. Studies need to consider the individual need and subgroups rather than the tendinopathy population as a whole.

Implications and future research

The findings of our scoping review suggest that there is a gap in the literature in terms of studies that examine the effectiveness of pain neuroscience education interventions in patients with tendinopathy. Although a trend has been observed in recent years towards the use and study of this type of intervention, a larger volume of studies with adequate designs that allow the analysis of the effectiveness of this intervention is still necessary.

The findings of this review provide the framework for the development of a practical guide for how to evaluate the need for and how to apply pain neuroscience education in persistent painful tendinopathies.

Ethical statement

Ethical approval is not required due to the nature of the study.

Data availability statement

The authors confirm that the data supporting the findings of this study are available within the article and its supplementary materials.

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Declaration of competing interest

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ptsp.2023.07.002.

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