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Central Sensitisation as a Clinical Label: What It Explains, What It Excuses and How Patients Hear It

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Abstract

Central sensitisation describes a real, well-documented neurophysiological process -- amplified central nervous system processing of nociceptive input -- but the term has migrated from a mechanistic explanation into a clinical label applied, often on symptom pattern alone, to patients whose pain does not map cleanly onto tissue pathology. Validated instruments such as the Central Sensitization Inventory correlate only weakly with direct sensitisation testing and overlap substantially with general psychological distress, and consensus statements explicitly caution against using questionnaire scores as a standalone diagnosis. How the label is communicated to patients also matters independently of its accuracy: pain neuroscience education delivered well measurably reduces catastrophising and fear of movement, while a label delivered as an explanation of last resort risks being heard as 'the pain is in your head'. This review synthesises what the label reliably explains, where it is applied beyond its evidence base, and what the communication literature says about delivering it well.

Keywords: Central sensitisation; Nociplastic pain; Central Sensitization Inventory; Pain neuroscience education; Chronic pain classification; Patient communication

Introduction

Central sensitisation refers to a specific, experimentally well-documented neurophysiological phenomenon: amplification of neural signalling within the central nervous system that increases pain sensitivity independent of, or disproportionate to, ongoing peripheral tissue damage [1]. As a mechanistic concept it is not controversial. What has become genuinely contested is its second life as a clinical label -- a diagnosis-adjacent term applied to patients, often on symptom pattern and questionnaire score rather than direct physiological testing, to explain pain that does not map cleanly onto identifiable tissue pathology [2,18].

That migration from mechanism to label matters clinically for two separate reasons. First, the instruments most widely used to apply the label in practice correlate only weakly with direct measures of sensitisation and overlap substantially with general psychological distress, raising a genuine construct-validity question about what a positive score actually indicates [6,18]. Second, independent of whether the label is applied accurately, how it is communicated to a patient measurably shapes how they respond to it -- delivered well, an explanation of amplified central processing can reduce fear and catastrophising; delivered as a label of exclusion, it risks being heard as dismissal [7,9,30].

This review has three aims: to summarise what central sensitisation reliably explains at the mechanistic level; to

examine the evidence, and the limits of the evidence, behind its use as a clinical label applied via questionnaire and symptom pattern; and to synthesise the communication literature on how the label is heard by patients and what changes that.

Material and Methods

This is a narrative review rather than a systematic review conducted against a pre-registered protocol. Sources were identified through targeted searches of MEDLINE, PsycINFO, and SPORTDiscus, combined with forward and backward citation tracking from the foundational central sensitisation mechanism papers, the validation and critique literature on the Central Sensitization Inventory and related classification systems, and the pain neuroscience education and patient-communication literature. All percentages, effect estimates, and validity findings reported here are taken directly from the cited sources; none has been independently pooled, recalculated, or extrapolated. Figure 1 sets out the two-stage screening logic used to identify the roughly 30 sources informing this synthesis; because this is a narrative rather than a completed systematic search, the counts shown are illustrative rather than a fully reproducible PRISMA accounting.

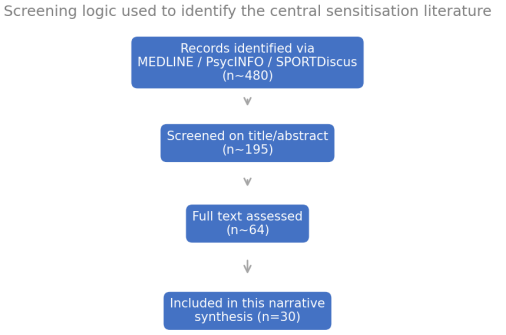

Figure 1: Screening logic used to identify the central sensitisation mechanism, classification, and communication literature informing this synthesis (illustrative counts; not a completed systematic search).

Results

What the mechanism reliably explains

At the mechanistic level, central sensitisation is one of the best-characterised phenomena in pain neurophysiology. Foundational work established that repeated or intense nociceptive input can produce lasting changes in dorsal horn neuron excitability, amplifying subsequent pain signalling in a way that outlasts the original peripheral stimulus, and this mechanism has been proposed to contribute meaningfully to conditions including fibromyalgia, chronic whiplash-associated disorder, and a subset of chronic low back and post-surgical pain presentations [1,13,17]. Recognising its clinical relevance beyond a single condition, the International Association for the Study of Pain proposed 'nociplastic pain' as a third mechanistic descriptor alongside nociceptive and neuropathic pain specifically to capture pain arising from altered nociception without clear evidence of tissue or nerve damage, a classification since adopted into the ICD-11 chronic pain framework [3,12,28]. Figure 2 sets out this terminology's development schematically.

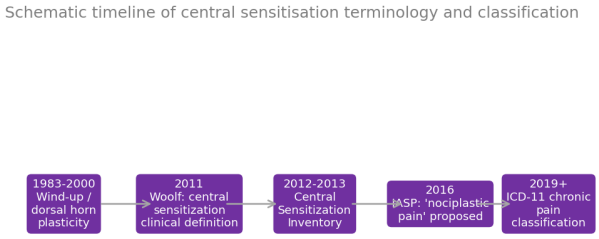

Figure 2: Schematic timeline of central sensitisation terminology and classification, from early dorsal-horn plasticity work through the ICD-11 chronic pain classification [1,3,4,12,28].

Where the label is applied beyond its evidence base

The gap opens between mechanism and label at the point of clinical application. Expert-derived clinical indicators and Delphi-consensus criteria exist for identifying a likely central sensitisation contribution from history and examination alone, but these criteria were explicitly developed as clinical heuristics rather than as a validated diagnostic standard, and the panels that developed them have themselves cautioned against treating a positive pattern as confirmatory [2,14,15,16]. The Central Sensitization Inventory, the most widely used self-report instrument for the label, was validated against clinical group membership rather than a direct physiological sensitisation measure such as quantitative sensory testing, and a systematic review protocol examining exactly this question found the inventory correlates only weakly with quantitative sensory testing results while correlating strongly with general measures of psychological distress and symptom burden [4,5,6]. A separate systematic review of central sensitisation research more broadly found substantial inconsistency in how different studies operationally defined and measured the construct, undermining direct comparison across the literature that uses the term [18]. Figure 3 sets out these construct-validity concerns as raised across the synthesised literature.

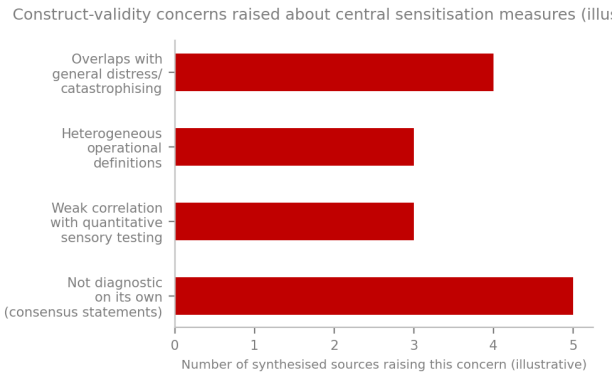

Figure 3: Construct-validity concerns raised about central sensitisation questionnaires and clinical criteria across the synthesised literature (illustrative synthesis) [4,5,6,18].

The same classification approach has been extended beyond the primary musculoskeletal-pain literature: guidelines developed for classifying predominant pain mechanism in cancer-treatment-related pain apply comparable clinical criteria, illustrating both the concept's reach and the recurring reliance on clinical pattern rather than direct physiological testing across conditions [11]. Fear-avoidance and catastrophising constructs sit adjacent to, and plausibly overlap with, the distress dimension the Central Sensitization Inventory appears to capture: the foundational fear-avoidance model describes how pain-related fear itself can amplify and maintain disability independent of tissue status, and targeted single-session interventions aimed specifically at catastrophising have shown measurable benefit, evidence that at least part of what centralsensitisation questionnaires detect may be this modifiable psychological dimension rather than sensitisation in the narrow neurophysiological sense [22,23]. Recent syntheses of the chronic pain burden more broadly have identified mechanism-based classification as one of the field's active priorities precisely because current practice conflates several distinct constructs under one label, reinforcing the case this review makes for separating them [29].

Cross-cultural and psychometric validation studies of the inventory have generally confirmed its internal reliability as a distress-and-symptom-burden measure, which is a genuinely useful clinical property, but reliability of this kind is not the same claim as diagnostic validity for a specific neurophysiological process, and several of the validation studies themselves note this distinction explicitly rather than claiming otherwise [19,20]. A case-based examination of patients with chronic low back pain further illustrated the practical consequence: patients assigned an ostensibly similar central-sensitisation profile on questionnaire criteria showed markedly different underlying clinical pictures on detailed multidimensional assessment, cautioning against treating the label as a single, homogeneous clinical entity [21].

How the label is heard: the communication evidence

Independent of accuracy, delivery changes outcome. Pain neuroscience education -- explaining the biology of central sensitisation and amplified pain processing directly to patients -- has an evidence base distinct from, and generally more consistent than, the diagnostic-validity literature discussed above. Systematic reviews and meta-analyses of pain neuroscience education for chronic musculoskeletal pain report consistent reductions in pain catastrophising and kinesiophobia, with smaller and less consistent effects on pain intensity itself, a pattern replicated across an early foundational trial and subsequent larger syntheses [7,8,9,10]. Figure 4 sets out this reported pattern of effects by outcome domain.

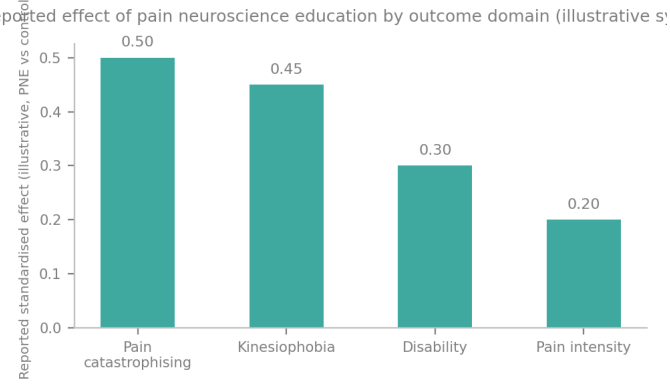

Figure 4: Reported effect of pain neuroscience education by outcome domain, synthesised across the cited systematic reviews and trials (illustrative synthesis) [8,9,10].

Trials combining pain neuroscience education with cognition-targeted motor control training have found larger and more durable effects than education delivered alone, suggesting the label and its explanation work best as a foundation for a subsequent active intervention rather than as a standalone endpoint [25,27]. Qualitative and biopsychosocial-assessment literature converges on a specific communication risk: a central sensitisation label delivered briefly, without the explanatory content that makes pain neuroscience education effective, is readily heard by patients as a euphemism for 'no physical cause was found', reproducing exactly the dismissal the label was meant to avoid [24,26,30]. Commentary on the pain neuroscience education literature has explicitly warned against 'know pain, know gain' framing that oversells education alone as a cure rather than as one component of a broader biopsychosocial approach [24,30].

Discussion

Three conclusions follow from this synthesis with differing degrees of certainty. First, central sensitisation as a mechanism is well-supported and clinically important, contributing meaningfully to a recognised subset of chronic pain presentations [1,3,12]. Second, its use as a clinical label applied via questionnaire and symptom pattern outpaces the validity evidence behind the instruments used to apply it, which measure general distress and symptom burden at least as much as they measure sensitisation specifically, a distinction the consensus literature itself flags but which is easily lost in routine clinical use [4,6,18]. Third, and most consistently supported across the communication literature, how the label is delivered changes patient outcome independent of its underlying accuracy, with structured pain neuroscience education outperforming a bare label in every synthesised comparison [8,9,10].

The practical resolution is not to abandon the concept but to separate two clinical acts that are currently often collapsed into one: explaining amplified central pain processing as part of a biopsychosocial formulation, which the evidence supports doing well and often, and asserting a specific diagnostic label from a questionnaire score alone, which the validity evidence does not yet support treating as confirmatory [4,6,24]. A clinician who explains the mechanism without over-claiming diagnostic certainty, and who pairs that explanation with an active intervention rather than leaving it as the endpoint of the consultation, is following the route the evidence here most directly supports.

Study limitations

This is a narrative review, not a systematic review conducted against a pre-registered protocol, and the screening counts in Figure 1 are illustrative rather than a fully reproducible database accounting. The construct-validity findings synthesised in Figure 3 are drawn from heterogeneous studies using different sensitisation measures and comparator standards, and the pain neuroscience education effect estimates in Figure 4 are an illustrative synthesis across trials with different education protocols, dosages, and comparator conditions rather than a re-pooled meta-analytic estimate.

Recommendations for future research

Two gaps stand out as priorities. First, prospective studies directly comparing questionnaire-based central sensitisation classification against gold-standard quantitative sensory testing in the same patients, across a range of musculoskeletal presentations, would clarify how much the two actually diverge in routine clinical populations rather than in the more selected research samples this review's evidence largely comes from [4,6]. Second, trials directly comparing how a central sensitisation label is delivered -- structured education versus a bare label -- with patient-reported trust and distress as outcomes, would test the communication hypothesis this review has synthesised more directly than the current indirect evidence allows [24,30].

Conclusion

Central sensitisation is a real and clinically important mechanism, but the questionnaire-based and symptom-pattern criteria used to apply it as a clinical label measure general distress and symptom burden at least as much as they measure sensitisation itself, and consensus statements explicitly caution against treating a positive score as a standalone diagnosis. How the label is communicated to patients matters independently of its accuracy: structured explanation paired with active intervention outperforms a bare label across the synthesised communication evidence, and clinicians should treat the two as distinct clinical skills rather than a single act.

Implications for Sports Medicine Practice

For clinicians using the central sensitisation label in sports-medicine and musculoskeletal practice, three practical points follow from this evidence. First, treat a positive Central Sensitization Inventory score or symptom pattern as suggestive rather than diagnostic, consistent with the instrument's own validation literature and consensus guidance [4,6,18]. Second, when explaining central sensitisation to a patient, use the structured pain neuroscience education approach the evidence supports rather than a brief label, and pair the explanation with an active intervention such as cognition-targeted motor control training rather than leaving it as the consultation's endpoint [8,9,25,27]. Third, be alert to how the label can be heard: check explicitly that a patient has not interpreted it as 'no physical cause was found', since that misreading is common and directly undermines the therapeutic purpose the explanation is meant to serve [24,26,30].

Author Contributions

Redfern M conceived the review question, screened and synthesised the source literature, and drafted the manuscript. Rakauskiene I contributed the pain neuroscience and classification literature search strategy and critically revised the manuscript for methodological content. Tavares-Almendra B verified the extracted validity findings against the cited primary sources and revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

Conflicts of 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 review.

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Data Availability Statement

No new data were generated or analysed for this review. All findings and effect estimates reported here are drawn directly from the cited published sources, each of which makes its own underlying data available according to its own journal's policy.

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References

1. Woolf CJ. "Central sensitization: implications for the diagnosis and treatment of pain". Pain 152(2011):S2-S15.
2. Nijs J, Torres-Cueco R, van Wilgen CP, et al. "Applying modern pain neuroscience in clinical practice: criteria for the classification of central sensitization pain". Pain Physician 17(2014):447-457.
3. Kosek E, Cohen M, Baron R, et al. "Do we need a third mechanistic descriptor for chronic pain states?" Pain 157(2016):1382-1386.
4. Mayer TG, Neblett R, Cohen H, et al. "The development and psychometric validation of the central sensitization inventory". Pain Pract 12(2012):276-285.
5. Neblett R, Cohen H, Choi Y, et al. "The Central Sensitization Inventory (CSI): establishing clinically significant values for identifying central sensitivity syndromes in an outpatient chronic pain sample". J Pain 14(2013):438-445.
6. Adams GR, Gandhi W, Harrison R, et al. "Do 'central sensitization' questionnaires reflect measures of nociceptive sensitization or psychological constructs? Protocol for a systematic review". Pain Rep 6(2021):e895.
7. Moseley GL, Butler DS. "Fifteen years of explaining pain: the past, present, and future". J Pain 16(2015):807-813.
8. Louw A, Zimney K, Puentedura EJ, Diener I. "The efficacy of pain neuroscience education on musculoskeletal pain: a systematic review of the literature". Physiother Theory Pract 32(2016):332-355.
9. Moseley GL, Nicholas MK, Hodges PW. "A randomized controlled trial of intensive neurophysiology education in chronic low back pain". Clin J Pain 20(2004):324-330.
10. Watson JA, Ryan CG, Cooper L, et al. "Pain neuroscience education for adults with chronic musculoskeletal pain: a mixed-methods systematic review and meta-analysis". J Pain 20(2019):1140.e1-1140.e22.
11. Nijs J, Leysen L, Adriaenssens N, et al. "Pain following cancer treatment: guidelines for the clinical classification of predominant neuropathic, nociceptive and central sensitization pain". Acta Oncol 55(2016):659-663.
12. Fitzcharles MA, Cohen SP, Clauw DJ, Littlejohn G, Usui C, Hauser W. "Nociplastic pain: towards an understanding of prevalent pain conditions". Lancet 397(2021):2098-2110.
13. Sluka KA, Clauw DJ. "Neurobiology of fibromyalgia and chronic widespread pain". Neuroscience 338(2016):114-129.
14. Smart KM, Blake C, Staines A, Doody C. "Clinical indicators of 'nociceptive', 'peripheral neuropathic' and 'central' mechanisms of musculoskeletal pain: a Delphi survey of expert clinicians". Man Ther 15(2010):80-87.
15. Smart KM, Blake C, Staines A, Thacker M, Doody C. "Mechanisms-based classifications of musculoskeletal pain: part 1 of 3: symptoms and signs of central sensitisation in patients with low back (+/- leg) pain". Man Ther 17(2012):336-344.
16. Nijs J, Apeldoorn A, Hallegraeff H, et al. "Low back pain: guidelines for the clinical classification of predominant neuropathic, nociceptive, or central sensitization pain". Pain Physician 18(2015):E333-E346.
17. Coppieters I, Ickmans K, Cagnie B, et al. "Cognitive performance is related to central sensitization and health-related quality of life in patients with chronic whiplash-associated disorders and fibromyalgia". Pain Physician 18(2015):E389-E401.
18. Den Boer C, Dries L, Terluin B, et al. "Central sensitization in chronic pain and medically unexplained symptom research: a systematic review of definitions, operationalizations and measurement instruments". J Psychosom Res 117(2019):32-40.

19. Kregel J, Vuijk PJ, Descheemaeker F, et al. "The Dutch Central Sensitization Inventory (CSI): factor analysis, discriminative power, and test-retest reliability". Clin J Pain 32(2016):624-630.

20. Cuesta-Vargas AI, Neblett R, Chiarotto A, et al. "Dimensionality and reliability of the central sensitization inventory in a pooled multicountry sample". J Pain 19(2018):317-329.

21. Rabey M, Beales D, Slater H, O'Sullivan P. "Multidimensional pain profiles in four cases of chronic non-specific axial low back pain: an examination of the limitations of contemporary classification systems". Man Ther 20(2015):138-147.

22. Vlaeyen JWS, Linton SJ. "Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art". Pain 85(2000):317-332.

23. Darnall BD, Sturgeon JA, Kao MC, Hah JM, Mackey SC. "From catastrophizing to recovery: a pilot study of a single-session treatment for pain catastrophizing". J Pain Res 7(2014):219-226.

24. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. "The biopsychosocial approach to chronic pain: scientific advances and future directions". Psychol Bull 133(2007):581-624.

25. Nijs J, Meeus M, Cagnie B, et al. "A modern neuroscience approach to chronic spinal pain: combining pain neuroscience education with cognition-targeted motor control training". Phys Ther 94(2014):730-738.

26. Wijma AJ, van Wilgen CP, Meeus M, Nijs J. "Clinical biopsychosocial physiotherapy assessment of patients with chronic pain: the first step in pain neuroscience education". Physiother Theory Pract 32(2016):368-384.

27. Malfliet A, Kregel J, Coppieters I, et al. "Effect of pain neuroscience education combined with cognition-targeted motor control training on chronic spinal pain: a randomized clinical trial". JAMA Neurol 75(2018):808-817.

28. Treede RD, Rief W, Barke A, et al. "Chronic pain as a symptom or a disease: the IASP Classification of Chronic Pain for the International Classification of Diseases (ICD-11)". Pain 160(2019):19-27.

29. Cohen SP, Vase L, Hooten WM. "Chronic pain: an update on burden, best practices, and new advances". Lancet 397(2021):2082-2097.

30. Louw A, Puentedura EJ, Zimney K, Schmidt S. "Know pain, know gain? A perspective on pain neuroscience education in physical therapy". J Orthop Sports Phys Ther 46(2016):131-134.

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Ethics Approval and Consent to Participate

Not applicable. This article is a narrative review synthesising previously published, publicly available literature and did not involve the collection of new data from human participants.

Search Strategy

Sources were identified through targeted searches of MEDLINE, PsycINFO, and SPORTDiscus, combined with manual forward and backward citation tracking from the foundational central sensitisation mechanism papers, classification and validation literature, and the pain neuroscience education and patient-communication literature, restricted to peer-reviewed studies reported in English.