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Nociplastic pain: an introduction

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Chronic pain is common in chiropractic practice and often presents without clear evidence of tissue injury. Nociplastic pain is a recently defined concept that highlights altered nociceptive processing within the nervous system. This newer understanding of pain provides insight into chronic conditions such as chronic back or neck pain, chronic headaches, and fibromyalgia. These conditions are commonly encountered in chiropractic practice but may be challenging to address using traditional models. This commentary introduces nociplastic pain, outlining potential mechanisms and relevance to chiropractic care. We advocate a collaborative, multimodal management approach that includes patient education, exercise promotion,

Douleur nociplastique: une introduction

La douleur chronique est courante dans la pratique chiropratique et se manifeste souvent sans preuve évidente de blessure tissulaire. La douleur nociplastique est un concept récemment défini qui met en évidence un traitement nociceptif altéré au sein du système nerveux. Cette nouvelle compréhension de la douleur offre un aperçu des problèmes de santé chroniques telles que les douleurs chroniques au dos ou au cou, les maux de tête chroniques et la fibromyalgie. Ces problèmes de santé sont couramment rencontrés dans la pratique chiropratique, mais peuvent être difficiles à traiter lorsqu'on utilise des modèles traditionnels. Ce commentaire présente la douleur nociplastique, en décrivant ses mécanismes potentiels et sa pertinence en ce qui concerne les soins chiropratiques. Nous préconisons une approche de gestion collaborative

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and functional goal-setting within a biopsychosocial framework. Understanding nociplastic pain equips chiropractors to support patients with complex chronic pain through compassionate, evidence-based care that addresses the whole person.

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KEY WORDS: chiropractic; management, pain; pain, chronic; widespread chronic pain; central sensitization; interdisciplinary health teams; fibromyalgia; pain, back; pain, neck; headache; nociplastic; neuropathic; nociception; biopsychosocial

et multimodale qui inclut l'éducation des patients, la promotion de l'exercice et la définition d'objectifs fonctionnels dans un cadre biopsychosocial. Comprendre la douleur nociplastique permet aux chiropraticiens de soutenir les patients aux prises avec des douleurs chroniques complexes grâce à des soins prodigués avec compassion et fondés sur des données probantes, qui tiennent compte de la personne dans son ensemble.

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MOTS CLÉS : chiropratique; gestion, douleur; douleur, chronique; douleur chronique généralisée; sensibilisation centrale; équipes de santé interdisciplinaires; fibromyalgie; douleur, dos; douleur, cou; mal de tête; nociplastique; neuropathique; nociception; biopsychosocial

Introduction

Advances in pain science continue to transform our understanding of pain mechanisms. Traditionally, pain has been mechanistically classified as either nociceptive or neuropathic in nature, and cases that did not fall easily into one of these categories were often labeled as idiopathic or pejoratively suggestive of malingering.[1] This framework, however, was incomplete and left many patients without a clear explanation for their symptoms. By 2017, sufficient evidence had accumulated to describe a third pain mechanistic descriptor (i.e., type of pain), characterized by alterations in nociceptive processing.[2–4] This new understanding of pain is now recognized as nociplastic pain.[5–8] Nociplastic pain is defined as "pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain" (Table 1).[5,8] The purpose of this commentary is to introduce nociplastic pain, its purported pathophysiologic mechanisms, management strategies, and its implications for clinical decision-making within the chiropractic profession.

Nociplastic pain

Nociplastic pain represents a distinct mechanistic pain category characterized by aberrant pain processing within the nervous system.[10] Unlike nociceptive or neuropathic pain, nociplastic pain is not directly attributable to tissue damage, inflammation, or nerve injury. Instead, nociplastic pain develops from abnormal neuronal signaling, causing increased sensitivity to various sensory stimuli and perpetuating the cycle of persistent pain.[6,10]

Nociplastic pain can provide an explanation for how chronic and recurrent pain conditions, such as chronic non-traumatic low back or neck pain, become established and maintained for prolonged periods.[11] Understanding chronic pain from a nociplastic pain perspective offers a rationale for persistent pain that cannot be fully explained by traditional pathoanatomical models, while avoiding tendencies to attribute such conditions to solely psychopathologic causes.[12] The concept of nociplastic pain may provide validation and reduce stigma for patients who may have been led to believe that pain isn't *real* or that it is *all in their head*.[13] This new mechanistic descriptor of pain promotes a more nuanced approach to evaluating and treating chronic pain, which aligns with the latest advances in pain science.[13]

Mechanisms underlying nociplastic pain

Nociplastic pain develops from altered nociceptive processing in the central and peripheral nervous systems and can occur in the absence of nociceptor activation or

Table 1.
Three types of pain

Type of Pain	Definition[8]	How It Develops	Where the Pain is Felt	Examples
Nociceptive Pain	Pain arising from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors.	From tissue injury, inflammation, or physical stress causing activation of nociceptors.	Pain typically localizes to the area of injury or damage. Referred pain may occur but remains regionally connected to the nociceptive source.	Acute sprain or strain Fracture Burns Post-surgical pain Renal colic (kidney stone) Rheumatoid arthritis
Neuropathic Pain	Pain caused by a lesion or disease of the somatosensory nervous system.	From nerve damage, pressure on a nerve, or inflammation surrounding a nerve.	Pain, paresthesia, or weakness are limited to neuroanatomically plausible distributions (e.g., dermatomes or peripheral nerve distributions).	Lumbar or cervical radiculopathy Neurogenic claudication Diabetic neuropathy Postherpetic neuralgia Trigeminal neuralgia MS-related pain
Nociplastic Pain	Pain arising from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain.	Pain caused by alterations in sensory processing, which heightens pain sensitivity, even in the absence of obvious injury.	Pain is chronic, generally widespread or poorly localized, and often does not follow a neuroanatomically plausible distribution. Pain is often accompanied by fatigue, poor sleep, or a lack of mental clarity.	Fibromyalgia Chronic non-traumatic LBP Chronic non-traumatic neck pain Migraines Chronic tension-type headaches Irritable bowel syndrome Chronic TMD CRPS type I Chronic prostatitis Vulvodynia

CRPS, complex regional pain syndrome; LBP, low back pain; MS, multiple sclerosis; TMD, temporomandibular joint disorder. Table adopted from Nijs J, *et al*.[9]

somatosensory pathology.[10,14–17] Current understanding of the neurophysiologic mechanisms of nociplastic pain is complex and remains incomplete, but emerging evidence suggests that the key pathophysiologic changes associated with nociplastic pain include central sensitization, alterations in brain network connectivity, and peripheral nervous system changes.[10,15]

Central sensitization is the hallmark of nociplastic pain and refers to heightened excitability within the central nervous system and amplification of sensory input, resulting in increased pain perception.[5,11,18] Clinically, this may present as an exaggerated pain response to high-threshold stimuli (i.e., hyperalgesia) or a pain response to typically non-painful, low-threshold stimuli such as light touch (i.e., allodynia), reflecting a lowered pain threshold.[10,14,18] Central sensitization occurs due to a variety of mechanisms that remain incompletely understood, but changes within the spinal cord and brain network connectivity are known to play a role.[10,15,18] Spinal mechanisms include regional clustering and convergence of signals from different pain locations, spinal cord reorganization, hyperresponsiveness of spinal dorsal horn neurons, amplified spinal reflex transmission, decreased spinal inhibition, and temporal summation.[6,15,18–20] Evidence also suggests that neuroimmune activation occurs via spinal microglia along with increased concentration of substance P and glutamine levels within cerebrospinal fluid.[14–16,21] Altered connectivity within various brain regions is also implicated in the development of central sensitization, particularly increased connectivity between the default mode network, salience network, and sensorimotor network.[10,14,15] These large-scale brain networks

are involved in self-referential thought, attention and sensory integration, and sensory processing and motor initiation.[10,22] Although these networks appear to become enmeshed, the mechanisms by which this contributes to sensory, emotional, or cognitive aspects of the pain remain unclear. Changes in the size and shape of the gray and white matter, in areas of the brain related to pain perception, have also been observed on magnetic resonance imaging (MRI) as a consequence of chronic pain.[23–26] The function of normal descending inhibitory spinal pathways may also be altered in cases of nociplastic pain.[10,15,27,28] Disturbance of this normal inhibitory nociceptive signaling is referred to as *disinhibition,* but the mechanisms facilitating it are not yet well understood.

Neuronal changes may also occur in the peripheral nervous system of those with nociplastic pain, though they are generally considered to play a lesser role than central mechanisms.[10,14,15,18] *Peripheral sensitization* refers to increased sensitivity to sensory stimuli, resulting in a heightened pain response.[29,30] While this process serves a protective role following acute tissue injury or inflammation, by promoting healing,[30] it becomes pathological when it persists beyond the acute phase of tissue repair, contributing to maladaptive nociception in cases of nociplastic pain.[8,14,15,18] Less is known about peripheral sensitization than central sensitization, but peripheral sensitization is believed to involve an expansion of the receptive field, elevated concentrations of pro-inflammatory cytokines and chemokines, proliferation of sodium channels, and abnormal coupling of primary afferent neurons by sympathetic neurons, known as sympatho-afferent coupling.[14,15,18,31–33] Peripheral sensitization is believed to initiate or maintain central sensitization via perpetual bombardment of the central nervous system with nociceptive stimuli.[10,34] Persistent nociceptive stimulus is characteristic among individuals with chronic inflammatory autoimmune conditions, such as rheumatoid arthritis,[34] and may explain why fibromyalgia is more common among individuals with co-occurring autoimmune inflammatory conditions.[34–38]

Top-down versus bottom-up nociplastic pain subtypes

Emerging research suggests that nociplastic pain may involve potential subtypes, termed *bottom-up* and *top-down*, based on their predominant mechanistic pathway.[10,34,39] Top-down nociplasticity or nociplastic pain arises primarily from impaired descending pain modulation,[28] and is reportedly more common in individuals with substantial psychological comorbidities, often developing at a younger age[10,34]. In contrast, bottom-up nociplastic pain results from persistent peripheral nociceptive input, as seen in conditions like rheumatoid arthritis or advanced osteoarthritis, ultimately leading to central sensitization.[10,34]

Understanding these subtypes may help to inform treatment.[10,34] Treatment of bottom-up nociplastic pain may respond more favorably to treatments targeting peripheral sources of nociception, for example manual therapies, while top-down nociplastic pain is believed to respond more favorably to treatments targeting central pathways, such as cognitive-behavioral therapy (CBT), mindfulness-based strategies, or biofeedback (Figure 1).[10,34,40]

Nociplastic pain conditions

Nociplastic pain may be a component of any chronic pain condition,[10] but conditions characterized by nociplastic pain are those where nociplastic pain is believed to be the *predominant* pain mechanism[41]. These conditions are now beginning to be referred to as *chronic primary pain syndromes* by the International Classification of Diseases (ICD) coding system and have been described as painful conditions in their own right,[42] rather than conditions where pain emerges as a secondary feature of another disease process[43]. The most recognized nociplastic pain syndromes include a wide range of chronic pain conditions such as fibromyalgia, chronic nonspecific low back pain, migraines, chronic tension-type headaches, irritable bowel syndrome (IBS), and temporomandibular joint disorder (TMD).[10,14,41,44] Other conditions involving predominant nociplastic pain mechanisms include complex regional pain syndrome type I (CRPS-I), or chronic pelvic pain syndromes (e.g., chronic prostatitis, vulvodynia) (Table 1).[5,10]

Chronic pain conditions characterized by nociplastic pain often coexist with other chronic pain conditions (i.e., chronic overlapping pain conditions) and are more prevalent in individuals with a higher burden of comorbidities.[10,18,45] The relationship between nociplastic pain and the changes in neurophysiology helps to explain non-painful features that are known to accompany these chronic pain conditions. Comorbid conditions that are as-

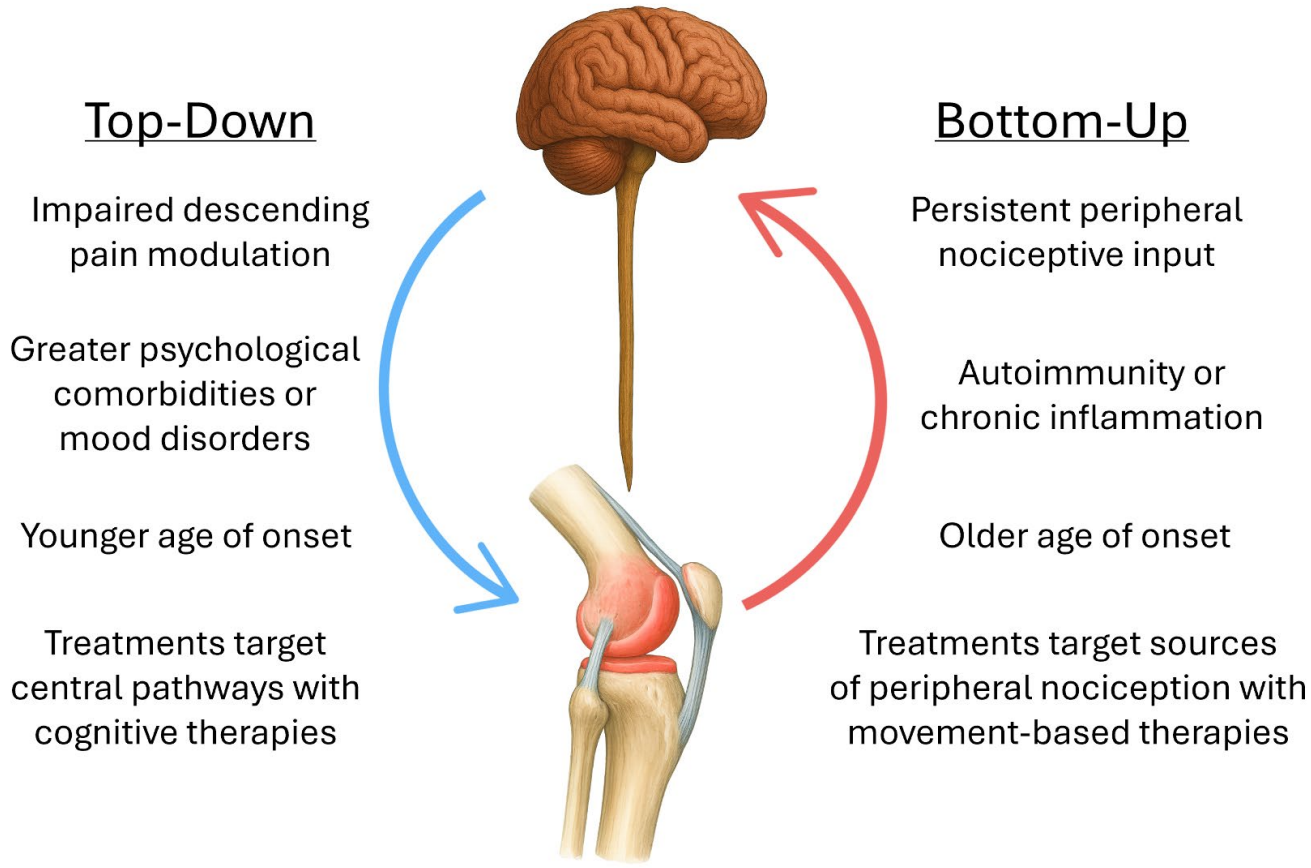

Figure 1.
*Top-Down and Bottom-Up Subtypes of Nociplastic Pain**

*This figure was adapted from the work of Kaplan CM, *et al*.[10] and Murphy AE, *et al*.[34]

sociated with nociplastic pain include depression, anxiety, post-traumatic stress disorder (PTSD), sleep disturbance, poor mental clarity (i.e., brain fog), chronic abdominal or pelvic pain, or other multisensory sensitivities to light, sound, or odors.[10,14,18] The clustering of these chronic conditions supports the concept of shared overlapping neurophysiologic mechanisms within the nervous system.

Diagnosing nociplastic pain

At present, no validated diagnostic tools or biomarkers exist to formally identify nociplastic pain and this mechanistic classification is based on a comprehensive history, physical exam, and clinical judgement.[10] In 2021 the IASP released a grading system to assist clinicians with identification, allowing for nociplastic pain to be qualified as *possible* or *probable* (Figure 2).[6,20] Efforts to further refine this grading criteria continue, with this criteria having recently been refined to include a *non-classifiable pain* designation.[46–48]

Clinical tools, such as the Central Sensitization Inventory (CSI),[49] screening for *yellow flags*,[50] or movement-evoked pain (MEP),[51] have shown potential for measuring aspects of nociplastic pain in clinical settings, but capture only limited aspects of nociplastic pain. Abnormalities associated with nociplastic pain may be quantified via the use of quantitative sensory testing (QST), sensory evoked potentials, or functional MRI,[6,10,11,15,39] but these methods are largely reserved for research settings and are not yet recommended for use in clinical practice[10].

The current lack of validated diagnostic criteria likely

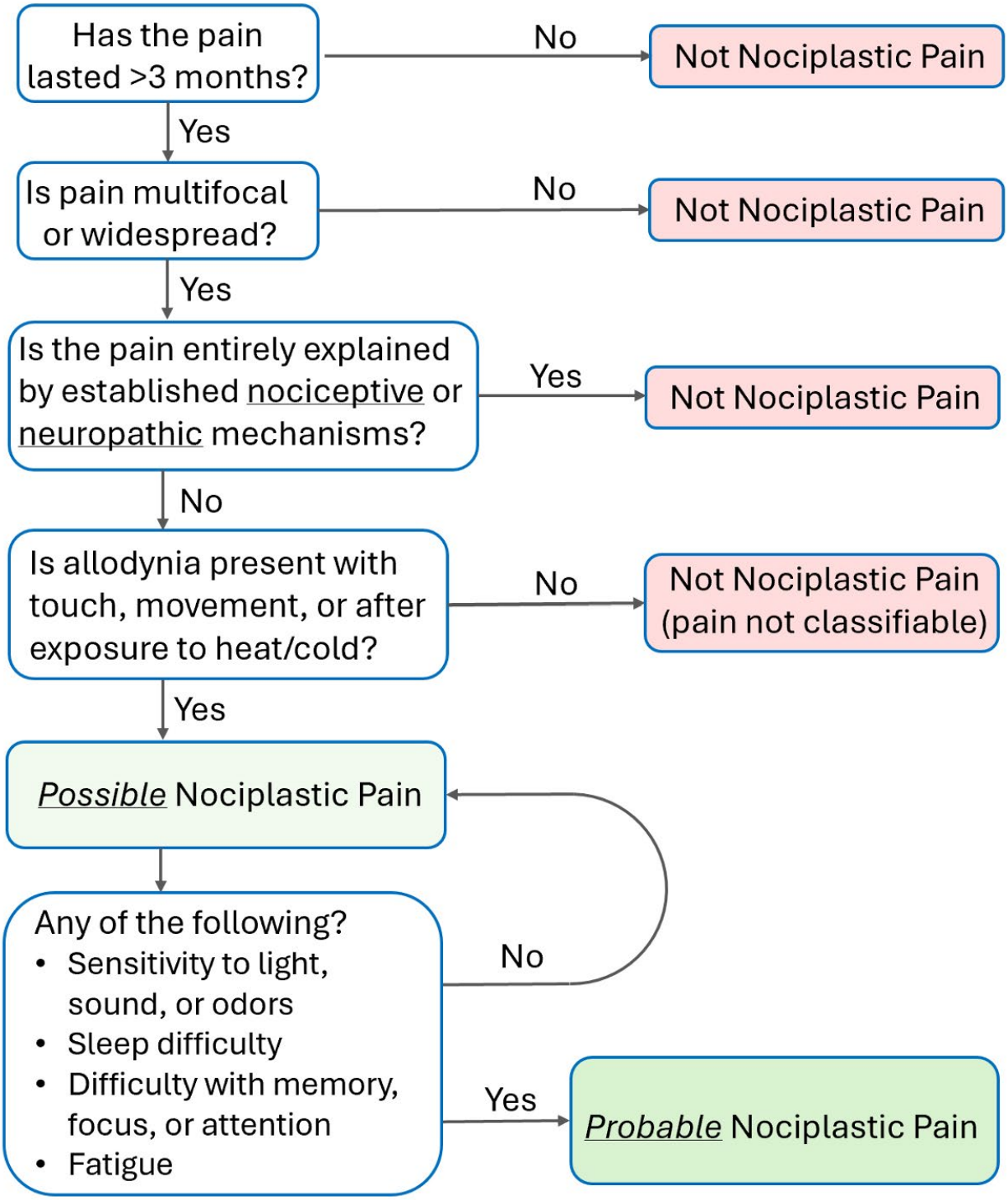

Figure 2.
*Flow chart for identifying and grading nociplastic pain**

*This figure was adapted from the work of Kosek E, *et al.*,[20,47] Nijs J, *et al.*,[58] and Yoo MY, *et al.*[18]

contributes to difficulties in quantifying the prevalence of nociplastic pain conditions.[10,40] Nociplastic pain conditions are likely to be common, but are often underrecognized or attributed to other causes.[40] For context, conditions primarily involving nociplastic pain are known to be widespread. Chronic low back pain affects approximately 13% of adults,[52] fibromyalgia impacts around 5% of the population,[53] and up to 4% of the population experiences chronic tension-type headaches[54]. Increased awareness and recognition of nociplastic pain stands to help guide more targeted treatments aimed at addressing the underlying mechanisms driving these painful conditions. Many conditions now understood to be predominantly nociplastic pain in nature are among the most common conditions managed by chiropractors,[55–57] highlighting the critical importance of recognizing nociplastic pain in clinical practice.

Mixed pain mechanisms

Nociplastic pain may be present in isolation or as part of a mixed pain state. *Mixed pain* refers to the simultaneous involvement of nociceptive, neuropathic, or nociplastic mechanisms, with one or more mechanisms potentially predominating.[59,60] Accordingly, nociplastic pain may occur in combination with nociceptive and/or neuropathic pain.[60,14]

To illustrate this concept, we present a hypothetical clinical scenario. A 58-year-old male experiences persistent back pain and stiffness localized to the thoracolumbar spine and paraspinal regions. His pain is moderate in intensity and began insidiously at age 40 and is aggravated by movements in all planes of thoracolumbar motion and by prolonged sitting. Sitting for longer than 30 minutes significantly increases his pain, while movements involving lumbar lateral bending and extension provoke pain flares lasting approximately five minutes. These activities occasionally lead to pain radiating into the lateral gluteal and posterolateral thigh regions, accompanied by intermittent subjective numbness and tingling into the proximal posterolateral aspects of his calves.

Previous radiographs reveal moderate lumbar spondylosis with zygapophyseal (i.e., facet) joint arthropathy and moderate bilateral L3-L5 lumbar neuroforaminal narrowing. His health history includes class II obesity, chronic bilateral knee pain, intermittent neck and right shoulder pain, migraines, irritable bowel syndrome, depression, anxiety, sensitivity to loud sounds, and non-restorative sleep with moderate daily fatigue. He also reports hesitancy towards exercise due to worries about damaging what he describes as his "crumbling discs" (a phrase reflecting the patient's fear-driven beliefs, rather than a formal diagnosis).

Figure 3 illustrates how varying degrees of overlapping pain mechanisms are present in this case.[61] Increased pain with extension and the presence of facet arthropathy are suggestive of a nociceptive component. Multilevel neuroforaminal narrowing, with pain radiating into the thighs and intermittent paresthesia in the legs, indicates a neuropathic component. Finally, his 18-year history of chronic widespread pain, multi-sensory sensitivities, sleep difficulty, and daily fatigue are suggestive of a *probable* nociplastic pain component.

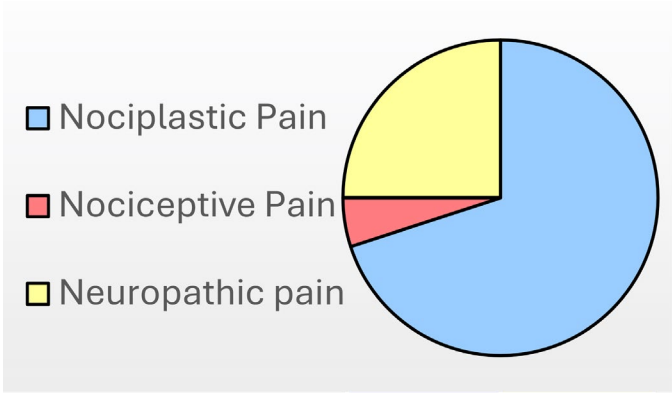

Figure 3.
Components of mixed pain in this chronic low back pain scenario

Implications for chiropractors and other health care providers

Chiropractors diagnose and manage a variety of painful neuromusculoskeletal conditions, which likely involve nociplastic pain.[55,62] These include both widespread pain conditions (e.g., fibromyalgia) and more localized pain conditions (e.g., chronic back pain, chronic migraine, chronic tension-type headaches, and temporomandibular joint disorders). Since some of these do not have an identifiable, peripheral lesion with which to target therapeutic interventions, patients and providers alike may experience distress and confusion regarding the best management approach. Moreover, an overemphasis towards no-

ciceptive and/or neuropathic pain mechanisms may lead to the improper use of imaging or prioritization of structural causes of pain, which have been shown to promote concerns or confusion and limit a patient's recovery.[63,64] It is therefore critical for clinicians and researchers to be aware of and acknowledge nociplastic pain as a legitimate pain mechanism in order to facilitate understanding and to provide reassurance and effective management.[58]

Diagnosis of nociplastic pain can prove clinically challenging with the clinical assessment forming the evaluative backbone.[15] Patients commonly report pain that is longstanding and poorly localized.[65] Nociplastic pain is often disproportionately and unpredictably impacted by aggravating and alleviating factors.[65] Additionally, it is associated with concomitant symptoms such as fatigue, sleep disturbances, cognitive issues, mood disorders, and heightened sensitivity to environmental stimuli.[6] Psychological factors such as stress, anxiety, depression, a history of trauma (e.g., PTSD, adverse childhood experiences), catastrophizing, or fear-avoidance behaviors or kinesiophobia are thought to play a role in the development and/or maintenance of nociplastic pain.[10] Patients may also report a protracted history of pharmacologic, non-pharmacologic, interventional, and surgical treatment, with minimal or transient benefit.[34] The clinical assessment should be used to help rule out other drivers of pain and to assess for features characteristic of nociplastic pain. Patients may display signs of hyperalgesia and allodynia, hesitancy and guarded movements during range of motion testing, and difficulty maintaining prolonged positioning.

It is understandable that many health care providers may be entirely unaware of the nociplastic pain classification as a recognized pain construct, although experienced practitioners may be intuitively familiar with its clinical presentation in their patients. The degree to which current chiropractic students and recent graduates are, or have been, exposed to nociplastic pain concepts in their respective training, is unknown. Research examining whether chiropractic programs are incorporating nociplastic pain education into their curricula would help clarify this gap, and efforts to integrate this content into chiropractic training are encouraged.

Clinicians are encouraged to provide reassurance and validation to patients, emphasizing that their pain will be acknowledged and respected.[66,67] Functional goal-setting and establishing realistic treatment expectations should be discussed from a management-focused perspective, rather than a curative one.[68,69] Clinicians are also encouraged to explain pain constructs, such as neuroplasticity and sensitization, in a patient-centered manner that avoids the use of technical jargon and is solutions-oriented.[40,70] The primary goal of this education is to validate the patient's experience, provide an explanation for their chronic pain, help them understand the drivers of their chronic pain, and discuss potential methods for modifying these factors. Stress management approaches including relaxation techniques, mindfulness, and meditation practices can be introduced to patients by chiropractors to help manage stress-related drivers of sensitivity. Moreover, considering sleep disturbances are a hallmark sign of nociplastic pain, chiropractors should discuss beneficial sleep strategies including adopting a regular sleep schedule, creating a relaxing bedtime routine, and optimizing the sleep environment.[40,71] Prompt referrals should be coordinated to sleep specialists if more intensive approaches are required.

Chiropractors can also help formulate and supervise graded exercise programs that are tailored to individual abilities and focused on functional goals. This may include a mixture of both aerobic exercise and resistance training. Recommendations for pacing activities should be provided and may be accompanied by strategies to track latent soreness (e.g. activity diaries) given that exercising in the presence of pain poses unique barriers to engagement compared to exercising pain-free.[72,73] More recently, a multidimensional rehabilitative approach known as cognitive functional therapy (CFT) has shown promise in reducing disabling chronic low back pain, likely driven by nociplastic pain mechanisms.[74–76] Through addressing negative cognitions (e.g., kinesiophobia), behaviors (e.g., guarded, non-varied movements), as well as healthy lifestyle changes, CFT aims to help patients make better sense of their pain and promote the extinction of safety behaviors through graded exposure to fearful movements. Chiropractors may consider integrating the principles of CFT, in combination with approaches to healthy lifestyle,[77] to help manage chronic nociplastic pain conditions.

The presence of nociplastic pain should not deter providers from evaluating and addressing peripheral dysfunctions as part of an overall treatment plan. Manual

therapies, including joint mobilization or manipulation, soft tissue techniques, and heat or cold applications may also play a role in treating patients with nociplastic pain by reducing peripheral nociception.[11] This "bottom-up" approach is directed to peripheral tissues, rather than central ones, and may help to attenuate peripheral drivers of central sensitization.[10,78–80] In 2010, Srbely proposed that spinal manipulative therapy may serve as a method for modulating neurophysiological sensitization.[81] Recent research has shown that a 4-week trial of 12 sessions of spinal manipulative therapy has the capacity to alter nociplastic pain factors and reduce segmental mechanical hyperalgesia among adults with chronic low back pain.[78] Clinicians are encouraged to avoid prolonged reliance on passive treatments alone, as these approaches are unlikely to provide more than temporary relief and may contribute to learned helplessness, potentially undermining the central role of self-care and lifestyle modification for the management of chronic nociplastic pain conditions.[82,83]

Pharmacologic treatments may play a role in nociplastic pain management. Pharmacologic agents are recommended in a stepwise approach when non-pharmacologic and self-management strategies fail to provide sufficient relief.[14,84] Importantly, national guidelines recommend that the use of pharmacologic treatment should only occur in tandem, rather than in lieu of, non-pharmacologic approaches.[39,84] Various central-acting medication classes have been found to be helpful for managing nociplastic pain including tricyclic antidepressants (e.g., amitriptyline, nortriptyline), selective norepinephrine reuptake inhibitors (e.g., duloxetine, venlafaxine), gabapentinoids (e.g., gabapentin, pregabalin), and low-dose naltrexone.[39,85] Traditional analgesics, such as nonsteroidal anti-inflammatories (NSAIDs) and acetaminophen, are often ineffective for nociplastic pain, while opioids are strongly discouraged.[14,39,86] Considering the significant sleep disturbances often experienced by patients with nociplastic pain, pharmacologic or supplemental sleep aids (e.g., melatonin) as well as advice on general sleep hygiene may play a role in certain cases.

Health care providers are encouraged to prioritize patients' needs and work collaboratively with other healthcare professionals to help provide comprehensive care for nociplastic pain. By integrating evidence-based non-pharmacologic treatments within a broader interdisciplinary pain management plan (Table 2), chiropractors can play a pivotal role in addressing the multifaceted nature of chronic pain conditions involving a significant nociplastic pain component. A patient-centered, multidisciplinary approach ensures that care is tailored to individual patients and stands to improve outcomes and empower patients to actively participate in their pain management.

Conclusion

The concept of nociplastic pain represents a fundamental shift in how chronic pain is understood and managed.[40] By recognizing the role of altered nociceptive processing within the nervous system, chiropractors can move beyond traditional structural and nociceptive models of diagnosis and treatment to providing more comprehensive, evidence-based, and patient-centered care. Many conditions, now considered to be primarily nociplastic in nature, are commonly encountered in chiropractic practice, emphasizing the need for a deeper understanding of this pain mechanism among clinicians.

Chiropractors are uniquely positioned to provide patient education, implement non-pharmacologic care, and collaborate within interdisciplinary teams to improve chronic pain management. While research on the direct effects of manual therapies for nociplastic pain conditions continues to evolve, evidence supports a multimodal approach that includes movement-based treatments, cognitive strategies, and lifestyle modifications within a biopsychosocial framework.[87] Integrating these strategies into chiropractic practice can enhance patient care by addressing both the physiological and psychosocial aspects of chronic pain.

Further research is needed to clarify nociplastic pain mechanisms, develop reliable diagnostic tools, and refine optimal treatment strategies. By staying informed and adaptable, chiropractors can play a pivotal role in advancing pain management strategies that validate patients' experiences and empower them toward meaningful functional improvements and improved quality of life.

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Table 2.
Overview of nociplastic pain management strategies

Non-pharmacological management (first line management)	Doctor-patient relationship	• Foster a trusting relationship involving open and honest communication • Validate the patient's experience • Provide reassurance and support • Set realistic goals, focused on improving function
	Patient education	• Explain nociplastic pain, while avoiding jargon • Promote self-care as the foundation to recovery • Explain treatment strategies in non-technical language
	Lifestyle modifications	• Physical activity • Healthy diet • Sleep hygiene • Stress management • Smoking cessation • Continued participation in work and social activities
	Psychological therapies	• Cognitive-behavioral therapy (CBT) • Acceptance and commitment therapy (ACT) • Mindfulness strategies • Pain reprocessing therapy (PRT)
	Rehabilitative and integrative therapies	• Physical therapy • Occupational therapy • Chiropractic • Acupuncture • Massage therapy • Yoga, Pilates, or Tai Chi
Pharmacological management	Centrally acting medications	• Tricyclic antidepressants (e.g., amitriptyline, cyclobenzaprine) • Serotonin-norepinephrine reuptake inhibitors (e.g., duloxetine, venlafaxine, milnacipran) • Gabapentinoids (e.g., gabapentin, pregabalin)
Management of complicating factors	Comorbidities	• Depression • Anxiety • Post-traumatic stress disorder (PTSD) • Insomnia • Obesity
	Psychosocial risk factors	• Catastrophizing (i.e., concerns about pain) • Fear-avoidance beliefs and behaviors • Kinesiophobia • Withdrawal from, or absence of, social support network

Adapted from Fitzcharles MA, *et al*.,[14] Kaplan CM, *et al*.,[10] and Ablin JN[40]

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