NEUROPHYSIOLOGICAL EFFECTS OF ACUPUNCTURE ANALGESIA IN INDIVIDUALS WITH CHRONIC PAIN

EFEITOS NEUROFISIOLÓGICOS DA ANALGESIA POR ACUPUNTURA EM INDIVÍDUOS COM DOR CRÔNICA

EFECTOS NEUROFISIOLÓGICOS DE LA ANALGESIA POR ACUPUNTURA EN INDIVIDUOS CON DOLOR CRÓNICO

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ABSTRACT

Introduction: Chronic pain, a highly prevalent condition sustained by neurophysiological, emotional, and social alterations, remains a clinical challenge due to the limitations of exclusively pharmacological treatments. In this context, the use of non-pharmacological strategies has increased, including acupuncture, which is recognized for modulating endogenous analgesic mechanisms.

Objective: To analyze the neurophysiological mechanisms of acupuncture-induced analgesia and the evidence supporting its effectiveness in individuals with chronic pain.

Methodology: A narrative review conducted according to SANRA criteria, carried out in the PubMed, PEDro, Web of Science, and LILACS databases, using the descriptors “Acupuncture,” “Chronic Pain,” and “Acupuncture Analgesia.” After screening, 46 studies were included.

Theoretical framework: Acupuncture modulates peripheral, spinal, and supraspinal pathways through the release of endogenous opioids, serotonin, and noradrenaline, strengthening the descending inhibitory system and reducing central sensitization. Neuroimaging evidence demonstrates functional reorganization of structures such as the anterior cingulate cortex, insula, thalamus, and prefrontal cortex, as well as adjustments in default mode and salience networks. Comparative trials indicate greater specificity of true acupuncture compared to sham acupuncture, particularly in connectivity between the periaqueductal gray matter, serotonergic nuclei, and pain-modulating regions, with effects observed mainly between 4 and 12 sessions.

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Final considerations: The evidence indicates that acupuncture exerts consistent analgesic effects supported by integrated neurophysiological mechanisms, contributing safely and effectively to the interdisciplinary management of chronic pain.

Keywords: Acupuncture. Chronic Pain. Analgesia. Neurophysiology.

RESUMO

Introdução: A dor crônica, condição altamente prevalente e sustentada por alterações neurofisiológicas, emocionais e sociais, permanece um desafio clínico diante das limitações dos tratamentos exclusivamente farmacológicos. Nesse cenário, cresce o uso de estratégias não farmacológicas, entre elas a acupuntura, reconhecida por modular mecanismos de analgesia endógena.

Objetivo: Analisar os mecanismos neurofisiológicos da analgesia por acupuntura e as evidências que sustentam sua eficácia em indivíduos com dor crônica.

Metodologia: Revisão narrativa conduzida segundo os critérios SANRA, realizada nas bases PubMed, PEDro, Web of Science e LILACS, utilizando os descritores “Acupuncture”, “Chronic Pain” e “Acupuncture Analgesia”. Após triagem, 46 estudos foram incluídos.

Referencial teórico: A acupuntura modula vias periféricas, espinais e supramedulares por meio da liberação de opioides endógenos, serotonina e noradrenalina, fortalecendo o sistema inibitório descendente e reduzindo a sensibilização central. Evidências de neuroimagem demonstram reorganização funcional de estruturas como córtex cingulado anterior, ínsula, tálamo e córtex pré-frontal, além de ajustes em redes de modo padrão e de saliência. Ensaios comparativos indicam maior especificidade da acupuntura verdadeira em relação à simulada, sobretudo na conectividade entre substância cinzenta periaquedutal, núcleos serotoninérgicos e regiões moduladoras da dor, com efeitos observados majoritariamente entre 4 e 12 sessões.

Considerações finais: As evidências apontam que a acupuntura exerce efeitos analgésicos consistentes, sustentados por mecanismos neurofisiológicos integrados, contribuindo de forma segura e eficaz para o manejo interdisciplinar da dor crônica.

Palavras-chave: Acupuntura. Dor Crônica. Analgesia. Neurofisiologia.

RESUMEN

Introducción: El dolor crónico, una condición altamente prevalente sostenida por alteraciones neurofisiológicas, emocionales y sociales, sigue siendo un desafío clínico debido a las limitaciones de los tratamientos exclusivamente farmacológicos. En este contexto, ha aumentado el uso de estrategias no farmacológicas, entre ellas la acupuntura, reconocida por modular los mecanismos endógenos de analgesia.

Objetivo: Analizar los mecanismos neurofisiológicos de la analgesia por acupuntura y las evidencias que respaldan su eficacia en individuos con dolor crónico.

Metodología: Revisión narrativa realizada según los criterios SANRA, llevada a cabo en las bases de datos PubMed, PEDro, Web of Science y LILACS, utilizando los descriptores “Acupuncture”, “Chronic Pain” y “Acupuncture Analgesia”. Tras el proceso de selección, se incluyeron 46 estudios.

Marco teórico: La acupuntura modula las vías periféricas, espinales y supramedulares mediante la liberación de opioides endógenos, serotonina y noradrenalina, fortaleciendo el sistema inhibidor descendente y reduciendo la sensibilización central. La evidencia de neuroimagen demuestra una reorganización funcional de estructuras como la corteza cingulada anterior, la ínsula, el tálamo y la corteza prefrontal, además de ajustes en las redes de modo por defecto y de saliencia. Ensayos comparativos indican una mayor especificidad de la acupuntura verdadera en comparación con la simulada, especialmente en la conectividad entre la sustancia gris periacueductal, los núcleos serotoninérgicos y las regiones moduladoras del dolor, con efectos observados principalmente entre 4 y 12 sesiones.

Consideraciones finales: Las evidencias indican que la acupuntura ejerce efectos analgésicos consistentes, sustentados por mecanismos neurofisiológicos integrados, contribuyendo de manera segura y eficaz al manejo interdisciplinario del dolor crónico.

Palabras clave: Acupuntura. Dolor Crónico. Analgesia. Neurofisiología.

1 INTRODUCTION

Chronic pain, defined by the International Association for the Study of Pain (IASP) as persistent pain for more than three months and associated with neurophysiological, emotional, and social changes, is one of the main causes of global disability (Nicholas et al., 2019; Treede et al., 2019). It is estimated that about 20% of the adult population lives with this condition, recognized by the World Health Organization as a problem of high relevance in public health (WHO, 2021).

The complexity of this condition stems from its multifactorial etiology and the interaction between peripheral alterations, central sensitization, emotional and cognitive factors, which makes its clinical management challenging (Robinson et al., 2022). Despite pharmacological advances, many patients remain symptomatic or develop adverse effects related to long-term use of analgesics and opioids, limiting the effectiveness of conventional treatments (CDC, 2022). Thus, the search for safe and effective alternatives for the management of chronic pain has stimulated the use of complementary therapies, such as acupuncture, whose efficacy is supported by a growing body of literature covering clinical trials, meta-analyses, and neurophysiological studies (Chou et al., 2022; Lyu et al., 2021).

Recent evidence supports that acupuncture exerts measurable analgesic effects at both the peripheral and central levels, observed in clinical trials and meta-analyses (Kato et al., 2022). Stimulation of specific acupoints promotes the release of neurotransmitters endogenous and activates descending inhibitory circuits (Yuan et al., 2022). Neuroimaging studies demonstrate the reorganization of brain networks involved in pain perception, suggesting an integrated regulation of sensory and affective dimensions (Wen et al., 2021). These findings legitimize its use as an adjunctive and safe intervention in the treatment of chronic pain (Niruthisard et al., 2024).

Although there is solid evidence that acupuncture modulates chronic pain by recognized neurophysiological mechanisms, part of these processes still needs clarification (Lyu et al., 2021; Wen et al., 2021). Since chronic pain involves alterations in nociceptive processing, especially central sensitization and dysfunctions of modulatory pathways, understanding how acupuncture acts on these circuits becomes essential. Thus, the present study aims to analyze and synthesize the neurophysiological mechanisms described in the literature to explain the analgesic effects of acupuncture in individuals with chronic pain.

The methodological diversity of the available research justifies the adoption of a narrative review, allowing the integration of findings from different designs and the construction of an expanded and reasoned interpretation of the processes involved in analgesia.

2 METHODOLOGY

The present study is a descriptive narrative review of the literature, guided by the following guiding question: what are the main neurophysiological mechanisms described in the literature to explain the analgesic effects of acupuncture in individuals with chronic pain?

The bibliographic search was carried out in the first half of 2025 in the PubMed, PEDro, Web of Science and LILACS databases. The indexing descriptors were retrieved from the DeCS/MeSH descriptors portal, and applied in English, Portuguese and Spanish, without the use of Boolean operators, corresponding to: "Acupuncture"; "Chronic Pain"; "Acupuncture Analgesia". In addition, a feedback search was performed in the reference lists of the included articles, in order to identify additional relevant publications.

The selection of studies followed the quality criteria recommended by the SANRA (Scale for the Assessment of Narrative Review Articles) checklist. Articles published between 2019 and 2025, in English, Portuguese, and Spanish, that addressed the effects of acupuncture on the modulation of chronic pain in humans and that described, directly or indirectly, neurophysiological mechanisms, whether peripheral, spinal, supramedullary, autonomic or neurochemical. Studies with animal samples, duplicate texts between databases, and studies without access to the full text were excluded.

After reading the titles and abstracts, 45 articles met the eligibility criteria and were included for the critical synthesis. These articles were examined in an organized manner, grouping the findings by level of physiological performance, type of pain investigated, and techniques employed, seeking to identify the main neurophysiological mechanisms involved in acupuncture-induced analgesia and its clinical implications in people with chronic pain. The synthesis was constructed in a narrative way, privileging convergences, divergences, methodological limitations and gaps in the literature, as guided by the SANRA checklist.

3 THEORETICAL FRAMEWORK

3.1 NEUROPHYSIOLOGY OF PAIN

Pain is a complex sensory and emotional experience, associated or not with actual tissue damage, and results from the integration between multiple neuronal systems (Treede et al., 2019). Under physiological conditions, pain mechanisms begin in nociceptors, free nerve endings sensitive to potentially harmful mechanical, thermal or chemical stimuli. These receptors convert the energy of the stimulus into electrical signals that are conducted by the Aδ and C fibers to the dorsal horn of the spinal cord, constituting the ascending nociceptive pathways. It is at this point that the nervous system begins to decide what to do with the painful information that arrives from the body. (Di Maio et al., 2023).

Once in the spinal cord, these signals are modulated before making their way to the brain. The first level of this modulation occurs in the mechanism known as the spinal gate. In it, inhibitory interneurons use neurotransmitters such as GABA and glycine to reduce the excitability of projection neurons, filtering the nociceptive impulse and preventing low-intensity stimuli from being amplified in an exaggerated way (Stachowski et al., 2021). This process works as a "quality control" of pain: only relevant information goes to higher levels of the central nervous system (Rivera-Arconada et al., 2025).

After this initial filtering, the nociceptive signals that go up to the brain are interpreted by an integrated network of structures known as the "pain matrix". This matrix involves the somatosensory cortex, responsible for the location and discrimination of the stimulus, the insula, associated with subjective and interoceptive experience, the anterior cingulate cortex (CCA), linked to the affective component, in addition to the thalamus and the prefrontal cortex (PFC), which organize and modulate cognitive aspects of pain. It is in this interaction that the stimulus gains meaning and transforms, in fact, into the conscious experience of pain (Kato et al., 2022; Wang et al., 2024).

The coordinated activity of these regions is shaped by neuroplasticity processes. In adaptive modulation, the nervous system adjusts its response to maintain functionality and protection. In maladaptive neuroplasticity, connections are reorganized in a dysfunctional way, reinforcing circuits that sustain pain even after the initial injury has resolved. This phenomenon explains why chronic pain can persist even in the absence of ongoing peripheral damage (Pan et al., 2022; Robinson et al., 2022).

Parallel to spinal modulation and cortical processing, the body has a second regulatory axis: downward modulation. Structures such as the periaqueductal gray matter (PAG) and the rostroventromedial medulla (CABG) send projections that release serotonin, noradrenaline, and endogenous opioids, capable of inhibiting nociceptive transmission in the spinal cord (Rocha-Jacob; Silva; Ferreira, 2024). This system acts as a physiological brake on pain and is strongly influenced by emotional, cognitive, and contextual factors, demonstrating that thoughts, expectations, and affective states modulate the painful experience (Treede et al., 2019).

The continuous communication between neurons of the nociceptive pathway is mediated by several excitatory neurotransmitters, such as glutamate, substance P, and CGRP, which promote the activation of projection neurons and participate in the encoding of the intensity and duration of the painful stimulus (Rivera-Arconada et al., 2025). The balance between excitatory and inhibitory neurotransmitters is essential for pain to maintain its

protective and adaptive character, functioning as an alert for potential threats to the body (Stachowski et al., 2021).

Thus, the painful experience results from the coordinated interaction between ascending nociceptive pathways, spinal inhibitory circuits, descending modulatory systems, and the integrated activity of the pain matrix. This organization ensures that pain functions as a physiological signal of protection, reflecting the complex neurofunctional structure of the nervous system (Wang et al., 2024).

3.1.1 Pathophysiology and classification of chronic pain

Pain chronicity is a multifactorial process underpinned by interrelated neurophysiological mechanisms. Among them, the following stand out: central sensitization (Rivera-Arconada et al., 2025); maladaptive neuroplasticity (Pan et al., 2022; Robinson et al., 2022); the dysfunction of inhibitory interneurons (Stachowski; Dougherty, 2021); the weakening of descending inhibitory pathways (Rocha-Jacob et al., 2024; Treede et al., 2019) and persistent glial activation (Di Maio et al., 2023). The interaction of these mechanisms favors the amplification of nociceptive signals and the transition from acute to chronic pain.

Pain chronicity occurs when neurophysiological modulation mechanisms cease to function properly. In this condition, changes in the organization and functionality of the pain matrix are among the main mechanisms associated with the transition from acute to chronic pain. Persistent activation of this network alters the connectivity between the somatosensory cortex, insula, thalamus, and CCA, resulting in increased neuronal responsiveness and amplification of pain perception (Wang et al., 2024).

This process is supported by maladaptive neuroplasticity, in which synaptic reorganization and excessive excitability of the central nervous system favor the maintenance of pain even after the resolution of the initial lesion (Pan et al., 2022; Robinson et al., 2022). Among the mechanisms involved, central sensitization stands out, a condition in which neurons in the dorsal horn of the spinal cord become hyperexcitable in the face of repetitive stimulation of C and Aδ fibers, generating a progressive amplification of the synaptic response known as wind-up (Rivera-Arconada et al., 2025).

When persistent, this phenomenon contributes to the transition between acute pain and chronic pain, reflecting a short-term synaptic facilitation that raises the frequency of action potentials and prolongs the painful sensation. Under physiological conditions, inhibitory interneurons of the dorsal horn limit this activity by controlling nociceptive transmission through neurotransmitters such as GABA and glycine. The dysfunction of these

interneurons causes synaptic disinhibition and increased neuronal excitability, intensifying the conduction of nociceptive impulses (Di Maio et al., 2023; Stachowski; Dougherty, 2021).

At the supramedullary level, descending inhibitory pathways, originating in the GWP and the CABG medulla, act to modulate pain through the release of serotonin, noradrenaline, and endogenous opioids, which reduce the excitability of neurons in the dorsal horn and limit nociceptive transmission (Rocha-Jacob et al., 2024; Stachowski; Dougherty, 2021). The weakening of these pathways compromises endogenous modulation, reduces the pain threshold, and favors the amplification of nociceptive signals, a mechanism often observed in conditions of chronic pain and associated with the loss of balance between excitation and inhibition (Di Maio et al., 2023; Rivera-Arconada et al., 2025; Treede et al., 2019).

This imbalance favors the emergence of allodynia and hyperalgesia, conditions characterized by the exaggerated painful response to harmless or moderate stimuli (Rivera-Arconada et al., 2025). Another commonly associated mechanism concerns the sustained activation of glial cells, such as microglia and astrocytes. These cells release pro-inflammatory cytokines, enhancing neuronal excitability and reinforcing the maintenance of central sensitization (Di Maio et al., 2023).

This set of structural and chemical alterations characterizes pathological synaptic plasticity, in which neuronal connections cease to perform a modulating function and start to reinforce pain circuits. In this way, the painful experience becomes independent of peripheral stimuli, being supported by central mechanisms, composing the clinical picture of primary chronic pain described in the International Classification of Diseases (ICD-11) (Treede et al., 2019).

Understanding these neurophysiological dysfunctions is essential for the clinical management of chronic pain. Treatment should seek to restore the balance between excitatory and inhibitory pathways, reduce neuronal hyperexcitability, and strengthen endogenous pain modulation mechanisms (Pan et al., 2022; Wang et al., 2024).

3.1.2 Multidimensionality of chronic pain and biopsychosocial model

Understanding the pathophysiological mechanisms, however, is not sufficient to explain the complexity of chronic pain. It is a phenomenon that emerges from the interaction between neurophysiological, emotional, cognitive, and social factors, shaping both the perception and expression of suffering (Robinson et al., 2022).

Neuroimaging evidence indicates that the painful experience is modulated by neural networks that integrate sensory, affective, and cognitive dimensions, involving the CCA, insula, and prefrontal cortex (PFC) (Wang et al., 2024). This integration demonstrates that

pain is not limited to peripheral nociceptive transmission, but involves brain circuits capable of amplifying, suppressing, or reorganizing pain perception, according to emotional states, expectations, and previous experiences (Vlaeyen; Crombez, 2020).

Structural and functional changes in the brain, combined with emotional states such as anxiety, stress, and depression, contribute to amplifying pain perception and reinforcing pain maintenance over time (Simons et al., 2022). Individual sensory profiles and differences in cortical connectivity also influence vulnerability to chronicity (Wang et al., 2024).

In addition to biological and emotional factors, the social dimension exerts a significant influence on the painful experience. Family support, community insertion, working conditions, and living environment determine the individual's coping ability and functionality (Kovačević et al., 2024). When these elements are insufficient or adverse, greater pain intensity and lower therapeutic response are observed (Gava et al., 2025).

These findings reinforce that chronic pain should not be considered just an individual condition, but a biopsychosocial phenomenon (BPS), in which psychological and social factors interact with neurophysiological mechanisms, modulating the intensity, persistence, and functional impact of pain (Robinson et al., 2022). Given this complexity, the BPS model is the most appropriate approach to understand and treat chronic pain. This model proposes the integration of biological, psychological, and social factors in clinical assessment and intervention, enabling a more comprehensive and person-centered approach (Simons et al., 2022).

By considering the interactions between chronic pain, mental health, and social context, the BPS model broadens the understanding of human suffering, highlighting the importance of strategies that promote psychosocial support, prevention of negative emotional impacts, and improvement of quality of life (Gava et al., 2025; Kovačević et al., 2024).

3.2 CHRONIC PAIN MANAGEMENT STRATEGIES

The clinical management of chronic pain requires a comprehensive and integrated approach, capable of contemplating the biological, psychological, and social aspects involved in the painful experience. Because it involves multiple dimensions, this condition represents a challenge for health professionals, requiring individualized care, whose manifestations and therapeutic responses vary widely among individuals (CDC, 2022).

Clinical management should seek not only symptomatic relief, but also the restoration of functionality and overall improvement of health (Knopp-Sihota; Morris; Courtney, 2022). Treatment should prioritize functional recovery, combining resources as clinically needed, and carefully weighing the risks and benefits of each intervention. International

recommendations guide the organization of safe and effective care, with an emphasis on person-centered practices (CDC, 2022).

The contemporary paradigm of care is based on multimodal approaches, which integrate medications, physiotherapy, restorative therapies, behavioral strategies and psychosocial interventions (Wang; Doan, 2024). However, long-term use of analgesic drugs, especially opioids, is related to risks of dependence, tolerance, and overdose, in addition to offering limited long-term benefits (CDC, 2022).

Non-pharmacological interventions, such as physical and psychological therapies and pain education, have demonstrated efficacy comparable to the use of analgesics, contributing to the improvement of functionality and well-being. (Knopp-Sihota et al., 2022). Integrated with conventional treatment, complementary therapies can expand the possibilities of intervention and strengthen interdisciplinary care models (Lyu et al., 2021; Wang; Doan, 2024).

Among the complementary interventions, acupuncture stands out, whose use has been widely documented in clinical research and systematic reviews. Studies suggest that the technique can influence central and peripheral nociceptive processes and assist in autonomic regulation, integrating with modern care plans aimed at pain reduction and functional restoration (Lee et al., 2024; Vickers et al., 2022).

3.3 ACUPUNCTURE IN CHRONIC PAIN MANAGEMENT

Acupuncture is an ancient practice of Traditional Chinese Medicine that has been consolidated as a technique of physiological regulation through the stimulation of specific points of the body, called acupoints (Zhu; Wang; Liu, 2021). These acupoints have microstructures rich in nerve fibers, vessels, and connective tissue, which contributes to their high therapeutic responsiveness (Ifrim-Chen; Antochi; Barbilian, 2019). The standardization of these points, associated with the modernization of the materials, favored their integration and expanded their use in the management of chronic pain (Zhu et al., 2021).

True acupuncture consists of inserting needles into defined acupoints, capable of activating sensory fibers and triggering autonomic and neurochemical responses associated with analgesia and physiological regulation (Hamvas et al., 2023). While sham acupuncture uses superficial stimuli or points outside the traditional meridians, functioning as an experimental control and producing minimal or non-specific effects (Liu et al., 2019).

The clinical procedure involves the insertion of thin, sterile needles into selected points based on Traditional Chinese Medicine diagnostic criteria, corresponding to areas of high neural and vascular responsiveness (Zhang et al., 2022). After skin asepsis, the needles are

inserted at varying depths according to the region and therapeutic objective (Ang et al., 2025). Manual manipulations, such as rotation or light traction, can be applied to intensify sensory activation (Zhang et al., 2022).

This stimulation usually produces the sensation of deqi, described as heaviness, pressure, numbness, tingling, or warmth. Deqi is considered a clinical marker of effective acupoint activation and is associated with the activation of cutaneous and myofascial nerve endings (Du et al., 2019). In addition, it triggers immediate adjustments in the autonomic nervous system, contributing to increased parasympathetic activity and sympathetic-parasympathetic rebalancing (Hamvas et al., 2023).

These responses occur because mechanical stimulation of the needles activates afferent fibers that project to autonomic nuclei of the brainstem and hypothalamus, regions essential for homeostatic regulation (Li et al., 2021). Thus, acupuncture can be understood as a neurostimulatory intervention that combines controlled needle insertion, deqi attainment, and activation of autonomic and sensory circuits involved in physiological modulation (Li et al., 2021).

In addition to classical acupoints, clinical practice also incorporates Ashi points, defined as areas of increased sensitivity identified by palpation, especially in musculoskeletal conditions (Zhu et al., 2021). These points do not necessarily follow the paths of the meridians, but are selected according to local or segmental pain. Although widely used, they have a lower volume of specific mechanistic evidence when compared to standardized acupoints (Ifrim-Chen et al., 2019). Therefore, the literature recommends the combination of Ashi points with classical acupoints that have greater experimental support, especially in protocols aimed at neurophysiological investigation (Zhu et al., 2021).

3.3.1 Neurophysiological mechanisms of acupuncture analgesia

Acupuncture-induced analgesia is associated with the release of endogenous neurotransmitters, such as opioids, serotonin, and norepinephrine, which act on ascending and descending pain pathways, reducing neuronal excitability and pain sensitivity. Among the mechanisms described, opioid modulation is the physiological axis with the greatest methodological support, supported by studies with antagonists such as naloxone, which attenuate the analgesic effect observed (Dimitrova et al., 2021; Niruthisard et al., 2024).

Classical acupoints such as Hegu (IG4) and Zusanli (E36) appear frequently in experimental models precisely because they trigger robust neurochemical responses. Stimulation of these points activates descending inhibitory pathways and modulates core pain structures, an effect that is significantly attenuated by opioid receptor blockade (Zhu et

al.,2021; Niruthisard et al., 2024;). Even so, the magnitude of this response varies according to protocol, stimulus intensity, and clinical heterogeneity of the samples, which limits the direct extrapolation of the findings.

Moving to more integrated levels of the central nervous system, acupuncture modulates multiple brain regions responsible for pain perception and control, including the somatosensory and prefrontal cortices, the thalamus, insula, and brainstem structures (Niruthisard et al., 2024; Kato et al., 2022). Neuroimaging assays show that manual IG4 and E36 stimulation promotes selective activations and inhibitions in these areas, reflecting functional reorganization compatible with pain reduction (Lu et al., 2021).

This reorganization is accompanied by changes in neural oscillations, as demonstrated by magnetoencephalography (MEG) studies. These investigations identified low-frequency oscillations in the occipital lobe and high-frequency oscillations in the prefrontal and somatosensory cortices, indicating a dynamic pattern of electrophysiological response during the intervention. From these local responses, functional connectivity studies suggest distributed effects in large-scale networks (Kato et al., 2022).

Such adaptations contribute to the modulation of functional connectivity between widely distributed brain networks, including the Default Mode Network, Salience Network, and the Descending Pain Modulation System. The modulation of these networks has been consistently associated with reduced pain and improved affective processing, although methodological differences persist regarding connectivity analysis techniques and the statistical models employed (Lu et al., 2021; Niruthisard et al., 2024).

In individuals with chronic pain, acupuncture regulates the connectivity between areas related to emotional and autonomic modulation, such as serotonergic nuclei of the brainstem, reinforcing the role of descending mechanisms, especially the activation of raphe and PAG nuclei, in the analgesia observed after IG4 and E36 stimulation, strengthening endogenous inhibitory mechanisms responsible for suppressing nociceptive impulses at the medullary level (Dimitrova et al., 2021; Niruthisard et al., 2024).

Another important axis of analgesia involves the brain's reward system. Acupuncture stimulation activates the mesolimbic dopaminergic circuit, attenuating negative emotions from the painful experience and increasing tolerance to nociceptive stimuli (Lu et al., 2021; Pan et al., 2022). These emotional effects are articulated with sensory and cognitive processes, reinforcing the multidimensional character of analgesia (Niruthisard et al., 2024).

Finally, acupuncture also triggers autonomic and neuroimmune responses that contribute to the modulation of inflammation and the restoration of neural homeostasis,

evidencing the interaction between nervous and immune systems. These effects involve, among other processes, the regulation of the neuroimmune axis and sympathetic and parasympathetic autonomic activity, effects frequently observed in E36 stimulation (Lyu et al., 2021; Niruthisard et al., 2024). The combination of these mechanisms favors adjustments in the functional plasticity of brain networks related to chronic pain and underpins clinically observed therapeutic effects (Lu et al., 2021; Niruthisard et al., 2024)

3.4 EVIDENCE-BASED PROTOCOLS

The recent literature on acupuncture in chronic pain demonstrates a growing number of experimental studies characterized by significant methodological heterogeneity. The differences involve point selection, insertion depth, stimulation techniques and deqi acquisition, as well as the use of neuroimaging methods employed to understand analgesic mechanisms (Cao et al., 2019; Xiang et al., 2019).

This methodological diversity also reflects the heterogeneity of chronic pain itself, which encompasses conditions with distinct pathophysiology and specific therapeutic demands. Studies on shoulder pain, for example, have evaluated distal points such as ST38 (Liu; Zhao; Jin, 2019), while protocols targeting systemic conditions such as fibromyalgia (Baelz et al., 2023) and sickle cell disease (Li et al., 2021) tend to employ broader combinations of acupoints.

Research involving neck pain and primary dysmenorrhea reinforces this variability by using, respectively, sets of points based on traditional recommendations (Xu et al., 2022) or the isolated application of SP6 (Xu et al., 2022). Thus, Table 1 shows a synthesis of evidence that reflects the plurality of these conditions and the complexity inherent in the establishment of standardized protocols.

Table 1

Acupuncture protocols employed in chronic pain conditions

References	Sample and type of chronic pain	Intervention	Protocol used	Observed effects
Liu *et al.* (2019) Essay E xploratory randomized clinical	Pain Chronicle on the shoulder. N= 38 subjects (Real acupuncture group N= 19 vs. Group sham acupuncture	Manual acupuncture on ST38 or on local points of the shoulder; 1 single treatment session; retention time ~20 min; initial manipulation of 30s.	ST38 contralateral vs ipsilateral; depth 10–15 mm; manual rotation to *deqi*.	Both sides reduced pain; contralateral had greater pain reduction and better ROM; no adverse events.

	N=19)			
Cao *et al*., (2019) Experimental study (fMRI) with pain-induced in a controlled manner	Pain Induced experimental in healthy volunteers. N = 27 healthy adults (crossover design)	Four interventions in order Rand omized: real acupuncture, acupuncture *sham*, VGAIT (*Video-Guided Acupuncture i magery treatment*) and VGAIT control (video of swab touching the skin); classic point stimulus (e.g. LI4), with cold pain induction and recording fMRI. for 9 min.	Points used in fMRI interventions: points on the legs (real points and adjacent control points; 1–2 points applied per session Real Point: experimental point located 3 cun above and 1 cun posterior to KI8, on the Kidney meridian.	Real acupuncture and VGAIT increased the pain threshold vs their controls. Real acupuncture activated the insula more; VGAIT produced greater deactivation in rostral ACC. It shows the potential of acupuncture and placebo interventions, but in an experimental, non-clinical model.
Xiang *et al*. (2019) Experimental clinical trial with fMRI	Chronic nonspecific low back pain. N= 52 individuals (Group with low back pain N=27 vs. Healthy control group N=25)	Ankle *acupoint acupuncture* with manual stimulation for 8 min while resting fMRI was recorded; single session; comparison with tactile stimulus control at the same place.	Acupuncture on the ankle, perimallegia/ankle points.	Acupuncture reduced pain VAS and modulated ALFF in pain-related regions (thalamus, somatosensory cortex, default mode network). There were distinct changes between chronic pain vs controls, suggesting Specific modulation of networks
	(real VS tactile stimulus)			of pain, but with heterogeneity and Sample small.
Li *et al*. (2021) Mixed methods pilot study	Pain as sociated with sickle cell disease. N= 6 adults with sickle cell disease and chronic pain	Individualized protocol as an adjunct to standard care; up to 10 sessions lasting 30 min, 1-2x/week, in a palliative care hospital; points defined according to complaint and TCM standard.	Analgesia and toning points based on TCM and STRICTA Standardized 18-point protocol: CV17, CV6, PC6 (bilateral), LI4, SP6, SP10, ST36,	Reduction of pain intensity and interference; 82% acceptance; safe and feasible acupuncture; no serious adverse events reported.

			LR3, LR8, KI3, CV7 (transverse).	
Kato *et al*. (2022) Experimental study with neuroimaging	Pain ch ronic (various etiologies) N = 21 patients with chronic pain	Sessions Uniqu e acupu ncture with magnetoencephalography (MEG) recording; systemic points with *Dehere*; needle left for 30 s; immediate pre/post evaluation.	WHO standard stitches and individualized stitches (≤2 cm); needles held for 30s, without rotation. Miscellaneous Points: LI4, BL15, BL16, BL48, GB33, BL53, SI7, among many others (each participant had 1 point selected).	Acupuncture modulated low-frequency oscillations in occipital and high-frequency regions Posted on Regions prefrontal/somatosensory; pain reduction was associated, but with focus predominantly mechanistic, No Clinical long-term.
Xu, H. *et al*.(2022) Randomized clinical trial with fMRI	Pain chr onic cervical. N= 99 subjects (True acupuncture: n ≈49; Sham acupuncture: n≈50)	Manual acupuncture in a standardized protocol for neck pain, 3x/week for 4 weeks (12 sessions) lasting 30 min; cranial and cervical points described in the article; stimulation up to *deqi.*	Recommended points in the literature for neck pain due to the absence of specification in the article: GB20, GV14, SI15, SI14, GB21, LI4, SI3, TB5 AT vs AS.	True acupuncture group had pain reduction (VAS p<0.001), and robust brain serotonergic vs. *sham* effect connectivity, but with individual variation and short follow-up.
Xu, J. *et al*. (2022) Essay neuroi maging clinician (PET/MVPA)	Primary dysmenorrhea (chronic pelvic pain). N = 34 (Acupuncture *verum* SP6: n = 17; Sham acupuncture SP6: n=17)	Bilateral acupuncture in Sanyinjiao (SP6), 8 weeks intervention; serial sessions with *deqi*, associated with resting fMRI acquisition; 30 min needle retention; manipulation of 30s each	Unilateral SP6; depth 1–1.2 cun; manual stimulation up to *deqi.*	Both groups reported pain reduction, but only the verum group showed normalization of connectivity between GWP and areas involved in affective/attentional pain modulation, suggesting a specific effect on the system descending modulation.

10 min.

Baelz *et al.* (2023) Essay Double-blind randomized clinical	Fibromyalgia. N = 40 (Acupuncture: n=20; *Sham*: n=20)	Systemic acupuncture with points: PC6, HT7, LI4, ST36, SP6, LR2, with *deqi*; 30 min/session, 1x/week; total of 8 sessions; 4-week follow-up.	PC6, HT7, LI4, ST36, SP6, LR2 (*DEQI*); (needles 15 mm lateral to the points, outside the meridian).	Acupuncture reduced pain in ~16% and improved FIQ by **~21% vs baseline, with significant difference** vs sham group.
Astini; Riberto (2023) Essay Prospective clinical	Severe hip osteoarthritis N = 12 (case series, single group)	Standardized acupuncture protocol for hip OA, 10 weekly sessions; systemic points + locations, standard hold; focus on pain control in patients eligible for arthroplasty but in a queue or with restrictions.	Standardized 6-point protocol used in all patients: GB29, GB30, GB34, GB36, BL62, SJ5.	VAS reduced from ~75.8 mm to 20 mm at the end of the 10 sessions and ~48 mm in the follow-up; relevant improvement in pain and function.

Legends: ADM – Range of motion; ACC – Anterior Cingulate Cortex; Ashi – Tender points tender to palpation; ALFF – Low Frequency Fluctuations in Amplitude; AS – Simulated Acupuncture; TA / Verum – True Acupuncture; CV – Vessel Conception (Ren Mai); deqi – Characteristic sensation of acupuncture; fMRI – Functional Magnetic Resonance Imaging; FIQ – Fibromyalgia Impact Questionnaire; Follow-up – Follow-up assessment after treatment; GB – Gallbladder; GV – Du Mai (Governor Vase); HT – Heart; KI – Kidney; LI – Large Intestine; LR – Liver; MEG – Magnetoencephalography; TCM/TCM – Traditional Chinese Medicine; MVPA – Multivariate Pattern Analysis; OA – Osteoarthritis; GWP – Periaqueductal Gray Matter; CP – Pericardium; PET – Positron Emission Tomography; SJ/TB – Triple Heater (Sanjiao); SP – Spleen; Sham – Sham acupuncture (placebo); ST – Stomach; STRICTA – Guidelines for Reporting Interventions in Clinical Trials of Acupuncture; VAS – Visual Analogue Scale; VGAIT – Video-Guided Acupuncture Imagery Treatment.
Source: Survey data.

The analysis of the included studies shows that, despite the diversity in protocols involving differences in the selection of points, depth, type of stimulation, and number of sessions, they all converge in demonstrating analgesic effects mediated by central and peripheral neurophysiological mechanisms that are widely consistent in the literature (Astini: Riberto, 2023; Baelz et al., 2023; Cao et al., 2019; Kato et al., 2022; Li et al., 2021; Liu et al., 2019; Xiang et al., 2019; Xu H. et al., 2022; Xu J. et al., 2022). Frequently employed points, such as LI4, ST36, SP6, PC6, HT7, ST38, and the Ashi points, activate somatosensory,

autonomic, and visceral pathways associated with pain modulation (Baelz et al., 2023; Liu et al., 2019; Xiang et al., 2019; Xu et al., 2022).

In the cases of neck pain and low back pain, approaches that combine standardized acupoints, local points and Ashi have shown significant pain reduction. These effects were accompanied by reorganization of brain activity in regions associated with nociceptive processing, reinforcing acupuncture's ability to modulate central networks involved in pain (Xiang et al., 2019; Kato et al., 2022; Xu et al., 2022).

Trials comparing real and sham acupuncture showed greater specificity of the real technique, evidenced by clinical improvement and changes in connectivity between the FPC, INSULA, ACC, GP, and serotonergic nuclei of the brainstem (Cao et al., 2019; Xu H. et al., 2022). These findings reinforce that analgesia depends on the integrated activation of distributed circuits of descending pain modulation (Kato et al., 2022; Xu J. et al., 2022).

In shoulder pain, the ST38 point produced immediate effects on range of motion and reduction of ipsilateral and contralateral pain, suggesting the participation of intersegmental mechanisms and sensorimotor integration (Liu et al., 2019). This response indicates that distal points can promote adjustments in higher levels of the central nervous system, favoring cortical reorganization related to the upper limb (Cao et al., 2019; Liu et al., 2019). Similarly, in low back pain, changes in the ALFF (Amplitude of Low-Frequency Oscillations) and fALFF (Fraction of Low-Frequency Oscillations) indices revealed modulations in the default mode network and in thalamocortical areas, associating clinical improvement with brain functional reorganization (Kato et al., 2022; Xiang et al., 2019).

In systemic conditions, such as fibromyalgia, protocols using broad combinations of points such as PC6, HT7, LI4, ST36, SP6, and LR2, which resulted in reduced pain, improved quality of life, and decreased symptoms such as fatigue and discouragement (Baelz et al., 2023). Although neuroimaging was not used in this sample, the findings are compatible with limbic and prefrontal modulation patterns described in other chronic pain conditions (Kato et al., 2022; Xu H. et al., 2022).

The efficacy of SP6 in primary dysmenorrhea reinforces the role of visceral mechanisms in analgesia. Only the group submitted to true acupuncture showed normalization of connectivity between the periaqueductal gray matter, limbic regions, and cortical areas related to interoception and affective assessment (Xu J. et al., 2022).

Studies involving less frequent conditions, such as severe hip osteoarthritis and pain associated with sickle cell disease, have also demonstrated improved pain and functionality, even in small samples. Despite their preliminary nature, these results reinforce the versatility of acupuncture in different models of chronic pain (Astini; Riberto, 2023; Li et al., 2021).

Mechanistic studies evaluated by fMRI, MEG, and experimental paradigms show that acupuncture modulates core pain matrix structures, such as the CCA, insula, dorsolateral and ventromedial PFC, thalamus, and somatosensory cortex (Cao et al., 2019; Kato et al., 2022; Xu H. et al., 2022). The technique also adjusts networks such as the salience network, default mode network, and sensorimotor network, which are essential for the maintenance of chronic pain and its emotional and cognitive load (Kato et al., 2022; Xu H. et al., 2022).

In addition, these studies show that image-guided interventions, often used as a simulated acupuncture condition, can generate neural patterns partially similar to those of real acupuncture, especially in regions associated with attention, expectation, and cognitive pain processing. This helps explain some of the variability between protocols, since psychological factors also modulate cortical networks of the painful experience. However, such interventions do not reproduce the full neurochemical effects of true acupuncture, whose peripheral stimulation activates descending pathways and promotes the release of endogenous opioids, serotonin and noradrenaline, which are fundamental for consistent analgesia (Cao et al., 2019; Kato et al., 2022).

In an integrated way, the studies analyzed point to four pillars that explain the analgesic effects of acupuncture: (1) release of neurotransmitters and neuropeptides (2) functional reorganization of cortical and subcortical networks; (3) activation of inhibitory descending pathways; and (4) autonomic and neuroimmune modulation. Thus, even in the face of wide variation between protocols, there is convergence in the demonstration of consistent neurophysiological mechanisms that support acupuncture-promoted analgesia in different chronic pain conditions (Baelz et al., 2023; Kato et al., 2022; Liu et al., 2019; Xiang et al., 2019; Xu H. et al., 2022).

4 FINAL CONSIDERATIONS

The literature analyzed demonstrates that acupuncture exerts consistent analgesic effects in individuals with chronic pain, confirming the main neurophysiological mechanisms described in the literature. In an integrated manner, the studies show four fundamental pillars of the analgesic action of the technique: (1) release of neurotransmitters and neuropeptides (2) functional reorganization of cortical and subcortical networks involved in pain perception and modulation; (3) activation of inhibitory descending pathways that reduce neuronal excitability; and (4) autonomic and neuroimmune modulation, contributing to the rebalancing of systems related to the painful experience. These findings reinforce that acupuncture acts in an integrated manner on multiple levels of the nervous system, fully answering the guiding question of this review.

Despite the solidity of the neurophysiological results, challenges persist related to the heterogeneity of the protocols, the variability in the selection of acupoints, and the lack of standardization in stimulation parameters, which reinforces the need for larger and more rigorous clinical trials. Even so, the body of evidence demonstrates that acupuncture is a safe, effective intervention in line with the BPS model, contributing to pain reduction, functional improvement, and modulation of emotional dimensions frequently affected in chronic pain. Thus, it is a relevant therapeutic resource in interdisciplinary care, expanding quality of life and offering significant clinical support to people living with chronic pain.

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APPENDIX

Table 2

Acupuncture points cited in the studies analyzed

Point	Code	Location
Zusanli	ST36 (E36)	Located 3 cun below the knee joint line, lateral to the tibial tuberosity (TAT).
Tiaokou	ST38	Located 8 cun below ST35, 1 cun lateral to crest of the tibia.
Hegu	LI4 (IG4)	On the back of the hand, at the midpoint between the 1st and 2nd metacarpals.
Fengchi	GB20	Below the occiput, in the depression between the trapezius and sternocleidomastoid.
Jianjing	GB21	At the midpoint between the acromion and C7, over the trapeze.
Julião	GB29	In depression between the anterior superior iliac spine and the greater trochanter.
Huantiao	GB30	In the depression between the greater trochanter of the femur and the sacral hiatus (S2).
Xiyangguan	GB33	Above the lateral epicondyle of the femur, between vastus lateral and biceps femoris.
Yanglingquan	GB34	Inferior and anterior to the head of the fibula.
Waiqiu	GB36	7 cun above the lateral malleolus, anterior to the fibula.
Sanyinjiao	SP6	3 cun above the medial malleolus, near the medial border of the tibia.
Xuehai	SP10	2 cun above the superomedial border of the patella.
Xingjian	LR2	On the dorsum of the foot, 0.5 cun proximal to the membrane interdigital between 1st and 2nd fingers.
Taichong	LR3	Between 1st and 2nd metatarsals, 1.5 to 2 cun above the interdigital commissure.
Ququan	LR8	Depression prior to the semimembranosus tendon, medial aspect of the knee.
Neiguan	PC6	2 cun above the wrist crease, between the tendons palmaris longus and flexor carpi radialis.
Shenmen	HT7	In the wrist fold, ulnar side, in the radial depression to the Pisiforme.
Taixi	KI3	Depression between the medial malleolus and the tendon Achilles.
Houxi	SI3	On the ulnar edge of the hand, proximal to the 5th joint finger.