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eISSN 2450-3118 · Open Access · Peer-reviewed

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KOCIUBA, Katarzyna, BRZUSEK, Mikołaj, MAZUR, Anna, ZIĘBA, Natalia, TARCZYŃSKI, Dominik, CZECH, Mateusz, CISŁAK, Miłosz, CHOJEŃ, Michał, CZMOK, Jakub and CIESIELSKA, Dominika. The role of physical exercise in modulating migraine pathophysiology from mechanisms triggering attacks to adaptive regulation of nociceptive responses. *Quality in Sport*. 2026;69:74412. <https://doi.org/10.12775/QS.2026.69.74412>

ARTICLE TIMELINE

Received: 18.07.2026. Revised: 12.08.2026. Accepted: 12.08.2026. Published: 20.08.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych). © The Authors 2026.

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The role of physical exercise in modulating migraine pathophysiology from mechanisms triggering attacks to adaptive regulation of nociceptive responses

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Abstract

Background

Migraine is a chronic neurovascular disorder characterized by recurrent headache attacks accompanied by sensory, autonomic, and neuroinflammatory symptoms. Physical exercise has a dual role in migraine, acting both as a trigger and as a non-pharmacological therapeutic intervention.

Materials and methods

A narrative literature review was conducted using PubMed, Scopus, Cochrane Library and Google Scholar. Studies were selected according to their relevance to migraine and physical exercise.

State of knowledge

Acute high-intensity exercise may trigger migraine through increased cortical excitability, metabolic stress, and activation of the trigeminovascular system. In contrast, regular exercise may reduce neuroinflammation, improve mitochondrial function, enhance endogenous pain inhibition, and regulate autonomic balance. These adaptations are associated with lower migraine frequency, duration, and intensity and may involve modulation of calcitonin gene-related peptide, serotonergic pathways, endorphin release, and central sensitization.

Conclusions

Regular, individualized physical exercise represents an effective non-pharmacological strategy for migraine prevention. Its benefits depend on exercise type, intensity and individual patient characteristics. Understanding the mechanisms underlying both the triggering and protective effects of exercise may support personalized migraine management.

Keywords: migraine, exercise, exercise-induced migraine, exercise therapy, hypoalgesia

1. Introduction:

Migraine is one of the most prevalent neurological disorders and a leading cause of disability worldwide, particularly among young and middle-aged individuals. According to the World Health Organization (WHO), headache disorders affect approximately 40% of the global population, corresponding to nearly 3.1 billion people in 2021. These conditions substantially impair quality of life, occupational functioning, social activity and psychological well-being. Furthermore, migraine ranks among the leading neurological causes of disability-adjusted life years (DALYs), underscoring its considerable global health burden [1].

Despite progress in pharmacological treatments, migraine remains a complex neurobiological disorder marked by dysregulation of the trigeminovascular system, neurogenic inflammation, central sensitisation and altered nociceptive processing. Contemporary research indicates that migraine pathophysiology involves abnormal cortical excitability, activation of calcitonin gene-related peptide (CGRP) mediated pathways, autonomic dysfunction and impaired descending pain-modulatory mechanisms overall.

The aim of this review is to summarise the current evidence on the dual role of physical exercise in migraine pathophysiology, emphasising the mechanisms by which acute exercise may trigger migraine attacks and the neurobiological processes through which regular exercise may confer protective effects.

2. Contemporary Aspects of Migraine Pathophysiology

2.1. Migraine Pathophysiology in the Context of Physical Exercise

Migraine is a neurobiological disorder marked by heightened excitability of nociceptive pathways, altered sensory processing and neurovascular dysregulation. Central to its pathophysiology is the trigeminovascular system, comprising trigeminal nerve fibres that

innervate the meninges and intracranial blood vessels. Experimental studies have demonstrated the presence of calcitonin gene-related peptide (CGRP) and elements of its receptor complex within the trigeminal ganglion and the dura mater, findings that underscore the pivotal role of the trigeminovascular system in the initiation and propagation of migraine pain [2].

2.2. The Role of CGRP

Activation of trigeminal afferent fibers results in the release of pro-inflammatory mediators and neuropeptides, particularly calcitonin gene-related peptide (CGRP), which promotes vasodilation, activation of peripheral nociceptive nerve endings, and peripheral sensitization. In an experimental mouse model, Cohen et al. demonstrated a high expression of the transient receptor potential canonical 4 (TRPC4) channel in the trigeminal ganglion. Activation of TRPC4 enhanced CGRP release and potentiated nitroglycerin-induced migraine-like pain behaviors, whereas pharmacological inhibition of the channel significantly reduced both plasma CGRP levels and mechanical hypersensitivity. These findings support the concept that peripheral sensory neurons play a crucial role in the initiation and maintenance of migraine attacks [3]. Calcitonin gene-related peptide (CGRP) is currently recognized as one of the principal mediators of migraine pathophysiology. In a study by Maddahi et al. (2024), CGRP mRNA expression in the rat trigeminal ganglion was found to be substantially higher than that of other members of the calcitonin peptide family. Furthermore, key components of the CGRP receptor complex, including receptor activity-modifying protein 1 (RAMP1) and calcitonin receptor-like receptor (CLR), were localized in trigeminal ganglion neurons and A δ fibers, further supporting the pivotal role of CGRP signaling in trigeminovascular activation and migraine pain generation [2].

2.3. Cortical Hyperexcitability

Although activation of the trigeminovascular system represents a fundamental peripheral mechanism underlying migraine, increasing evidence indicates that migraine is also characterized by abnormal cortical excitability and altered central sensory processing.

Cortical hyperexcitability is considered one of the hallmarks of migraine pathophysiology. Studies employing transcranial magnetic stimulation (TMS) have consistently demonstrated altered cortical excitability in individuals with migraine, particularly within the visual and motor cortices. Yuksel et al. evaluated cortical excitability in women with migraine using TMS and demonstrated impaired intracortical inhibitory mechanisms together with enhanced cortical responsiveness, providing further evidence that migraine is associated with disrupted cortical excitability [4].

This abnormal cortical excitability may contribute not only to the development of migraine aura but also to the reduced threshold for visual, auditory, nociceptive, and metabolic stimuli commonly observed in patients with migraine.

2.4. Autonomic Dysregulation

Autonomic nervous system dysfunction represents another important component of migraine pathophysiology. Clinical manifestations such as nausea, vomiting, excessive sweating, impaired thermoregulation, fatigue, drowsiness, and exercise intolerance indicate the involvement of both the sympathetic and parasympathetic nervous systems during migraine attacks. Woldeamanuel et al. Assessed heart rate variability (HRV) as a marker of autonomic nervous system function in patients with migraine and demonstrated significant associations between HRV parameters and headache characteristics, supporting the role of autonomic dysregulation in migraine pathophysiology [6].

Autonomic dysfunction is closely linked to oxidative stress and chronic low-grade inflammation. Arab et al. Investigated the effects of the Dietary Approaches to Stop Hypertension (DASH) diet on oxidative stress biomarkers in patients with migraine, demonstrating that migraine pathophysiology extends beyond neuronal dysfunction to include metabolic disturbances and inflammatory processes [7]

Peripheral and central sensitization are considered fundamental neurophysiological mechanisms underlying migraine pathophysiology and are believed to play a crucial role in both the initiation and chronification of migraine attacks. Although migraine was historically regarded primarily as a vascular disorder, current evidence indicates that persistent alterations in nociceptive processing within the trigeminovascular system and the central nervous system are largely responsible for the frequency, intensity, and persistence of migraine symptoms. Sensitization is a dynamic process in which repeated activation of nociceptive pathways progressively lowers pain thresholds and enhances neuronal responsiveness, ultimately resulting in exaggerated responses to both painful and normally innocuous stimuli.

Peripheral sensitization develops during the early stages of a migraine attack following activation of the trigeminovascular system. The release of neuropeptides, including calcitonin gene-related peptide (CGRP), substance P, and pituitary adenylate cyclase-activating peptide (PACAP), promotes neurogenic inflammation, vasodilation, plasma protein extravasation, and activation of inflammatory cells within the meninges. These processes increase the excitability of peripheral trigeminal nociceptors by reducing their activation threshold and facilitating action potential generation. Consequently, physiological stimuli such as vascular pulsation, coughing, bending forward, or physical exertion may become sufficient to activate

sensitized nociceptors and aggravate migraine pain. This mechanism provides a plausible explanation for why acute high-intensity exercise may provoke migraine attacks in susceptible individuals.

Persistent peripheral nociceptive input subsequently induces functional and structural changes within second-order neurons of the trigeminocervical complex (TCC), followed by alterations in thalamic and cortical regions involved in pain processing. This phenomenon, known as central sensitization, is characterized by enhanced synaptic transmission, increased spontaneous neuronal activity, expansion of receptive fields, and impaired descending inhibitory control. As a result, stimuli that are normally perceived as non-painful become painful (allodynia), while painful stimuli evoke disproportionately intense responses (hyperalgesia). Importantly, these neuroplastic changes may persist during the interictal period, indicating that migraine is associated with long-lasting dysfunction of central nociceptive processing rather than solely transient activation during headache attacks.

One of the most clinically relevant manifestations of central sensitization is cutaneous allodynia, defined as pain elicited by normally innocuous tactile, thermal, or mechanical stimulation. Patients frequently report pain or discomfort during everyday activities such as combing their hair, wearing glasses, shaving, or lightly touching the scalp. Increasing evidence suggests that cutaneous allodynia reflects progressive sensitization of second- and third-order neurons within the trigeminovascular pathway and therefore represents a reliable clinical marker of central sensitization. In a prospective cohort study, Pijpers et al. Demonstrated that patients with chronic migraine presenting with cutaneous allodynia exhibited a more severe clinical phenotype and that the presence of allodynia was associated with treatment response. These findings support the concept that cutaneous allodynia may serve not only as a marker of persistent central nociceptive dysfunction but also as a clinically relevant predictor of disease severity and therapeutic outcomes [8].

Objective evidence for abnormal pain processing has also been obtained using quantitative sensory testing (QST). In a recent case-control study, Suzuki et al. Demonstrated that patients with episodic migraine exhibited significantly lower thermal pain thresholds during the interictal period compared with healthy controls. Migraine patients showed increased sensitivity to both cold and heat pain despite the absence of an ongoing attack, indicating that altered nociceptive processing represents a persistent feature of migraine rather than a phenomenon restricted to the ictal phase. Furthermore, the degree of thermal hypersensitivity correlated with headache-related disability, suggesting that repeated migraine attacks progressively reinforce mechanisms of central sensitization [9].

These findings are further supported by the recent systematic review and meta-analysis conducted by Cnockaert et al., which synthesized available human studies investigating interictal central sensitization in migraine. The authors demonstrated that migraine patients consistently exhibit lower pressure pain thresholds, increased mechanical and thermal hypersensitivity, and impaired endogenous pain modulation compared with healthy individuals. Importantly, these abnormalities were observed in both episodic and chronic migraine, suggesting that central sensitization develops early in the disease course and becomes increasingly pronounced with repeated attacks. The meta-analysis also highlighted that these abnormalities are not confined to the trigeminal region but may extend to extra-cephalic body sites, reflecting widespread dysfunction of central pain-processing networks [10].

Collectively, these findings indicate that peripheral and central sensitization are dynamic processes that contribute not only to migraine initiation and chronification but also to the variability of patients' responses to physical activity. Increased excitability of the trigeminovascular system and impaired pain modulation may explain why physical exertion can precipitate migraine attacks in some individuals, whereas repeated, appropriately prescribed exercise has the potential to induce adaptive neuroplastic changes and improve clinical outcomes. Understanding these mechanisms provides an important physiological framework for interpreting the complex relationship between migraine and physical exercise discussed in the following sections.

3. Physical Exercise as a Potential Trigger of Migraine Attacks

3.1. Type of Physical Activity and Fear of Exercise as Factors Influencing Migraine Attacks

The impact of physical exercise on migraine is influenced not only by exercise intensity but also by the type of activity performed and the individual's susceptibility to migraine attacks. Many patients develop kinesiophobia, defined as the fear of engaging in physical activity due to concerns that exercise may trigger a migraine attack. In a cross-sectional study, Reina-Varona et al. investigated perceived fear and the difficulty associated with different types of physical activity in individuals with migraine. Running and jumping based exercises were reported as the most fear-provoking activities, whereas exercises involving spinal extension were perceived as the most difficult to perform. Importantly, individuals with higher levels of habitual physical activity reported lower levels of exercise-related fear, whereas fear-avoidance beliefs were associated with greater concerns about engaging in exercise. These findings suggest that psychological factors may constitute a substantial barrier to regular

physical activity among patients with migraine and should therefore be taken into account when designing exercise-based therapeutic interventions [11].

However, these concerns are not entirely unfounded, as intense physical exercise may indeed trigger migraine attacks in a subset of patients. Koppen et al. Reported that approximately 38% of individuals with migraine experienced exercise triggered attacks, most commonly during running, team sports, and high-intensity resistance training. Moreover, more than half of the participants deliberately limited their physical activity because of fear of provoking a headache. Importantly, exercise-triggered migraine was more frequently observed in patients with a more severe disease course and a higher attack frequency, suggesting that physical exercise should not be regarded as a universal migraine trigger. Rather, it appears to precipitate attacks in susceptible individuals, likely reflecting an increased sensitivity of the nociceptive system [6].

3.2. The Role of Neck Muscle Exercises in Migraine Management

Due to the frequent coexistence of neck pain and migraine, increasing attention has recently been directed toward the role of the cervical musculature in the pathophysiology of this disorder. It is estimated that neck pain affects the majority of individuals with migraine and may occur before, during, or between migraine attacks. This phenomenon is explained by the convergence of afferent fibers from the trigeminal nerve and the upper cervical spinal segments within the trigeminocervical complex (TCC). Consequently, it has been hypothesized that improving neck muscle function through appropriately prescribed exercises may reduce the excitability of the nociceptive system, decrease nociceptive input originating from the cervical region, and ultimately contribute to a reduction in both the frequency and severity of migraine attacks.

Benatto and colleagues evaluated the effectiveness of an 8-week neck muscle strengthening program in patients with migraine in a randomized controlled trial. Compared with the placebo group, the intervention did not result in a significant reduction in either the frequency or the intensity of migraine attacks. Nevertheless, improvements in cervical muscle activation were observed, indicating that the exercise program had a beneficial effect on neuromuscular control. The authors concluded that isolated neck muscle exercises are not sufficient as a standalone preventive treatment for migraine. However, they may represent a valuable adjunct to a comprehensive rehabilitation program, particularly in patients presenting with concomitant cervical musculoskeletal dysfunction [12].

Further interesting findings on this topic were reported by Carvalho et al. The authors demonstrated that maximal neck muscle endurance exertion may trigger a migraine attack in

predisposed individuals. The risk of attack onset was particularly high in patients experiencing pain radiating from the cervical region to the head, highlighting the important role of the trigeminocervical complex in the mechanisms underlying migraine induction. These findings suggest that not all forms of physical exercise are beneficial and that exercises requiring maximal loading of the neck muscles may act as a trigger for migraine attacks in a subset of patients [13].

4. Protective Effects of Regular Physical Activity on the Course of Migraine

In a study by Butt et al., the effects of a six-week intervention consisting of aerobic exercise combined with progressive muscle relaxation as an adjunct to standard pharmacological therapy were evaluated in patients with migraine. Compared with pharmacological treatment alone, the combination of aerobic exercise and relaxation techniques resulted in greater reductions in pain intensity, migraine-related disability, and symptoms of central sensitization. These findings suggest that regular physical activity combined with relaxation techniques may enhance the effectiveness of non-pharmacological migraine prophylaxis and improve patients' overall functioning [14].

4.1 Exercise-Induced Hypoalgesia

One of the mechanisms underlying the analgesic effects of physical activity is exercise-induced hypoalgesia (EIH), which refers to a transient increase in pain thresholds following exercise. In an experimental study, Vaegter et al. Demonstrated that this effect is significantly attenuated in patients with migraine compared with healthy individuals. Following aerobic exercise, patients with migraine exhibited a smaller increase in pain thresholds, suggesting impaired activation of descending pain modulatory pathways and persistent nociceptive hypersensitivity. These findings indicate that an abnormal analgesic response to exercise may represent an important component of migraine pathophysiology and may partly explain why physical activity does not produce the expected pain-relieving effect in a subset of patients [7].

4.2 The Importance of the Exercise Environment in Migraine Prevention

An additional factor that may influence the preventive effects of physical activity in migraine is the environment in which exercise is performed. In an interventional study, Navalty et al. Compared the effects of an identical aerobic exercise program conducted either outdoors in a natural environment or indoors. Participants who exercised outdoors demonstrated greater adherence to the training program and experienced a larger reduction in monthly migraine burden, including fewer headache days. The authors suggested that the beneficial effects of exercising in natural surroundings may be attributed to reduced stress, improved

psychological well-being, and greater motivation to engage in regular physical activity, which may indirectly contribute to a lower frequency of migraine episodes [15].

4.3 Effectiveness of Different Types of Physical Exercise in Migraine Prevention

4.3.1 Aerobic Training

In a randomized controlled trial, Meydanal et al. Compared the effectiveness of a 12-week aerobic exercise program with a combined training program consisting of aerobic exercise and resistance training targeting the neck and shoulder girdle muscles in patients with migraine. Both interventions resulted in a significant reduction in the number of monthly migraine days; however, the combined training program demonstrated superior efficacy. In addition, improvements in physical fitness and quality of life were observed in both groups, whereas a significant reduction in anxiety levels was found only in the combined training group. These findings suggest that supplementing aerobic exercise with resistance training may enhance the effectiveness of non-pharmacological migraine prevention [16].

4.3.2 Yoga

Yoga has also been shown to provide beneficial effects in patients with migraine. In a randomised controlled trial, Sujana et al. evaluated the effectiveness of a 12-week yoga programme consisting of breathing exercises (pranayama) and relaxation techniques as an adjunct to standard treatment for chronic migraine. Compared with patients receiving pharmacological treatment alone, those participating in the yoga programme experienced a significant reduction in the number of monthly migraine days, attack intensity, and attack duration, along with improvements in quality of life. In addition, favourable changes in autonomic nervous system regulation were observed, indicating enhanced parasympathetic activity, which the authors proposed may represent one of the mechanisms underlying the reduction in migraine attack frequency [17].

4.3.3 Resistance Training

Resistance training has also shown promising effects on reducing the frequency of migraine episodes. In a randomized controlled trial, Sari Aslani et al. investigated the effects of an eight-week resistance training program in women with migraine. Regular exercise performed three times per week resulted in significant reductions in migraine frequency, attack duration and pain intensity. Additionally, improvements in quality of life and increases in upper and lower limb muscle strength were observed. These findings suggest that resistance training may represent an effective and safe adjunct to non-pharmacological migraine management by reducing disease burden while simultaneously improving patients' physical function [18].

4.3.4 Resistance Training and Aerobic Exercise

In a network meta-analysis, Woldeamanuel and Oliveira compared the effectiveness of aerobic and resistance training for migraine prevention based on evidence from randomized controlled trials. Both exercise modalities were found to significantly reduce the number of monthly migraine days; however, resistance training demonstrated the greatest overall effectiveness. The authors concluded that regular physical activity represents a valuable component of non-pharmacological migraine management. Nevertheless, they emphasized that the currently available evidence remains limited and that further large-scale randomized controlled trials are needed to confirm these findings [19].

4.3.5 Dose response Relationship of Exercise Training

In a dose response meta-analysis, Ogreseanu et al. investigated the relationship between the cumulative dose of aerobic exercise and its effectiveness in migraine prevention. The analysis demonstrated that regular aerobic exercise significantly reduced both migraine attack frequency and pain intensity, with the therapeutic effect depending on the total duration of the exercise program. The greatest clinical benefits were observed after a cumulative training volume of approximately 900–950 minutes, corresponding to 30-minute exercise sessions performed three times per week for 10–11 weeks. Increasing the total training volume beyond this level did not result in additional clinical improvements, suggesting the existence of an optimal exercise dose for migraine prevention [20].

4.3.6 Dual-Task Training

In a randomized controlled trial, Deodato et al. evaluated the effectiveness of a dual-task exercise program, which involved the simultaneous performance of motor and cognitive tasks in patients with migraine. The intervention resulted in improvements in postural control, selected cognitive functions, and neurophysiological parameters, along with a reduction in migraine symptom severity. These findings suggest that training integrating both motor and cognitive components may represent a valuable adjunct to comprehensive rehabilitation programs for patients with migraine [5].

4.4 The Impact of Regular Physical Activity

In a prospective cohort study, Hagan et al. investigated the effects of regular physical activity on the clinical course of episodic migraine. The authors found that individuals who engaged in regular exercise experienced a more favorable disease course, reporting a lower overall migraine burden and fewer headache episodes. These findings suggest that regular, appropriately prescribed physical activity may represent an important component of non-pharmacological migraine prevention and provide an interesting contrast to studies identifying acute or high-intensity exercise as a potential trigger of migraine attacks [21].

Additional evidence supporting the protective role of physical activity was provided by Gan et al., who applied a Mendelian randomization approach to investigate potential causal relationships between lifestyle factors and migraine risk. The authors demonstrated that higher levels of physical activity were associated with a lower risk of migraine, whereas prolonged leisure screen time was associated with an increased risk of developing the disorder. Furthermore, the analysis suggested that part of the detrimental effect of a sedentary lifestyle may be mediated by hypertension. These findings indicate that regular physical activity may not only serve as an effective non-pharmacological strategy for migraine management but may also reduce the risk of migraine development through its beneficial effects on cardiovascular function and overall metabolic health [22]

4.5 The Influence of Exercise Timing and Chronotype on Migraine

In a crossover study, Malek et al. examined the effects of exercise timing and the alignment of training sessions with participants' chronotypes on the clinical course of chronic migraine. The investigators found no significant differences in migraine symptom reduction when comparing exercise performed in the morning with exercise performed in the evening. However, participants who exercised in accordance with their individual chronotype experienced a significant reduction in migraine burden, whereas no comparable improvement was observed among those whose training schedule was misaligned with their preferred circadian rhythm. These results imply that individualising the timing of physical activity to match chronotype may enhance the effectiveness of non-pharmacological migraine prevention strategies [23].

4.6 Optimal Exercise Intensity for Migraine Prevention

In a randomised controlled trial, Eslami et al. compared the effects of moderate- and high-intensity aerobic exercise in women with migraine. Both training programmes produced significant reductions in migraine attack frequency, attack duration, and pain intensity. Nevertheless, moderate-intensity exercise demonstrated greater efficacy than the higher-intensity programme in reducing both pain severity and attack duration. Despite these clinically meaningful improvements, the trial did not detect significant changes in circulating levels of pituitary adenylate cyclase-activating polypeptide (PACAP) or substance P, suggesting that the beneficial effects of physical activity may be mediated by neurophysiological mechanisms other than direct modulation of these specific neuropeptides [24].

5. Conclusions

The relationship between physical exercise and migraine is complex and appears to be bidirectional in nature. Although vigorous physical exercise can trigger migraine attacks in susceptible individuals, habitual regular physical activity has consistently been associated with reductions in attack frequency, pain intensity, and migraine-related disability. These apparently contradictory effects are thought to depend on a range of factors, including exercise intensity and duration, exercise modality, underlying training status, and individual susceptibility. Current evidence suggests that regular exercise may improve autonomic regulation, enhance endogenous pain inhibitory mechanisms and influence nociceptive processing, whereas excessive or unaccustomed exercise may transiently exceed the threshold required for migraine initiation.

Disclosure

Authors do not report any disclosures

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Project administration:

All authors have read and agreed with the published version of the manuscript.

Funding Statement: The study did not receive special funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: The authors confirm that the data supporting the findings of this study are available within the article's bibliography.

Conflict of Interest Statement: The authors declare no conflicts of interest.

Declaration of Generative AI and AI-Assisted Technologies:

The authors used ChatGPT to improve language and readability, after which the content was reviewed and edited. The authors accept full responsibility for the substantive content of the publication

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