SciMedCentral

Journal of Preventive Medicine & Healthcare

Editorial Article

Combine Brain and Muscle Signals to Make Strengthening Mechanistically Specific in Knee Osteoarthritis

Aifeng Liu[1,2]*

[1]First Teaching Hospital of Tianjin University of Traditional Chinese Medicine, China.

[2]National Clinical Research Center for Chinese Medicine, China

*Corresponding author

Aifeng Liu, First Teaching Hospital of Tianjin University of Traditional Chinese Medicine, National Clinical Research Center for Chinese Medicine, China

Submitted: 24 February 2026

Accepted: 23 April 2026

Published: 24 April 2026

ISSN: 2576-0084

Keywords

• Knee Osteoarthritis; Precision Rehabilitation; Joint Loading; Neuromuscular Control; Electromyography (EMG); Electroencephalography (EEG); Motor Learning

Abstract

Exercise therapy is widely recommended for knee osteoarthritis, yet clinical response varies markedly even when programmes appear similar. The decisive determinant is the neuromuscular dose actually delivered to the joint during functional tasks, rather than the surface similarity of prescriptions. Joint load is shaped by muscle recruitment amplitude, timing, sequencing, and co-contraction; inefficient or overly protective strategies can perpetuate unfavourable mechanical provocation. Such maladaptive loading may sustain a vicious cycle linking pain, guarding, and microenvironmental irritation, limiting durable symptom change even when strength increases. We propose a precision rehabilitation framework that explicitly couples three domains—central state, peripheral execution, and joint load—to guide progression. Surface electromyography can verify whether target muscles are truly recruited and whether antagonistic co-contraction is dominating, and can serve as temporary biofeedback to accelerate motor learning. Electroencephalography can index pain-related arousal, threat reactivity, and sensorimotor readiness, helping clinicians select tasks and dosing that the nervous system is prepared to learn. We emphasize that integrating cerebral and muscular signals is not yet an established superior therapy; its value should be tested in methodologically rigorous, phenotype-based trials that align mechanistic claims with clinically meaningful endpoints, including flare vulnerability, load tolerance, and durable changes in movement strategy.

Knee osteoarthritis (KOA) is among the most prevalent drivers of persistent pain and progressive loss of function [1]. Although exercise therapy is widely endorsed [2], clinical experience often reveals a puzzling disparity: two patients may adhere to ostensibly comparable training regimens, yet achieve markedly different outcomes. This divergence suggests that, over time, greater emphasis should be placed on the neuromuscular demands actually conveyed to the knee joint during everyday functional tasks—walking, ascending and descending stairs, and rising from a seated to a standing position—rather than on the exercise plan's surface similarity alone [3].

The load a joint ultimately bears is not merely imposed from without; it is fashioned from within by the recruitment patterns of the muscles that encircle it—by their amplitude of activation, their timing and sequencing, their coordination, endurance, and the subtle degree of synergistic co-contraction [4]. When this neuromuscular orchestration proves inefficient, unstable, or excessively protective, a patient may be "doing the exercises" in name, yet the knee joint may still be subjected, again and again, to unfavourable mechanical provocation. Much of the striking variability in how individuals with KOA respond to exercise can be traced to precisely this gap: the difference between the dose prescribed on paper and the dose that is, in reality, delivered to the joint [5].

In crafting a rehabilitation programme, we must articulate with precision the determinants of the joint's integrated microenvironment [6]. In KOA, pain and disease progression are not simply the consequences of altered structure or aberrant mechanics. Rather, local inflammatory activity, synovial irritation, metabolic mediators, and the sensitisation of nociceptors collectively shape how a patient moves. That altered movement strategy, in turn, redirects the very pathways through which forces are conveyed—across cartilage, subchondral tissues, and the synovium. Thus, a self-perpetuating cycle takes hold: microenvironmental irritation begets pain and protective behaviour; protective behaviour engenders maladaptive loading; maladaptive loading further amplifies microenvironmental stress. This loop makes plain that strength, by itself, is an insufficient remedy. One may augment muscular capacity, yet if the genesis and distribution of load remain unchanged, the

microenvironmental drivers of symptoms may endure [7]. Accordingly, the aim of rehabilitation is not merely "stronger muscles," but the cultivation of a steadier, more economical loading pattern—one that the patient can reliably reproduce in the cadence of everyday life [8].

Knee osteoarthritis is frequently accompanied by reflexive neural control processes that blunt the self-directed drive of pivotal musculature—most notably the quadriceps. A patient may strive intensely, yet the intended muscles may still not be recruited to their full capacity, so the training stimulus is only imperfectly conveyed to the very tissues we seek to fortify. Concurrently, the nervous system often resorts to compensatory co-contraction. By enlisting antagonistic muscles to "brace" the knee, the individual may feel safer, and the sensation of bodily steadiness may be briefly enhanced. Yet when such co-contraction is excessive—or mistimed—it elevates joint stress, hastens fatigue, and sustains heightened pain sensitivity [9]. If these constraints are overlooked and one merely escalates resistance or training volume, one risks reinforcing the very protective motor pattern that confines patients within the vicious cycle of pain, guarding, and maladaptive loading [10].

This, precisely, is why muscle-level monitoring is indispensable. Surface electromyography can reveal whether strength training is physiologically genuine—whether the quadriceps and hip-stabilising muscle groups are being recruited as intended, whether that recruitment remains faithful from repetition to repetition, and whether antagonistic co-contraction is quietly commandeering the motor strategy during functional tasks. More importantly, electromyography can be harnessed as biofeedback, rendering movement retraining truly actionable rather than a matter of merely following preset templates. One begins with low-risk tasks, enabling patients to acquire a more exact and economical motor pattern [11], and then deliberately translates that pattern into progressively more practical, real-world movements. The aim is not to become dependent on feedback itself, but to employ it as a temporary scaffold—an aid that hastens motor learning until movement is reliably stable—after which reliance on the feedback can be steadily tapered away.

Moreover, brain-level monitoring is equally warranted, for knee osteoarthritis is not merely a disorder of a peripheral joint. Pain can recalibrate attention, amplify threat appraisal, and diminish engagement in sensorimotor processing. In doing so, it modulates both the intensity of volitional drive and the fidelity of motor learning. Electroencephalography offers a window into this central milieu: the activity level of the sensorimotor system, whether the patient is poised in vigilant readiness or constrained by threat-related reactivity, and—crucially—whether the nervous system is likely to learn efficiently within a given training bout. Such signals can then inform the cadence of practice and the choice of tasks [12]. Two patients may execute the same programme under identical external loads, yet inhabit profoundly different neurophysiological states. One may be primed to acquire more nuanced motor strategies; the other may be curtailed by pain-driven arousal, compelled toward protective behaviour, and thereby deprived of autonomous control. If these distinctions can be recognised, progression can be orchestrated in concert with the nervous system rather than in opposition to it [13].

The three elements that are usually handled separately need to be connected: the central state, the peripheral execution, and the joint load [14]. It is necessary to assess whether the patient is in a state conducive to learning, confirm whether the expected muscles are performing the plan in an acceptable coordinated contraction manner, and only when the characteristics of the applied load are stable and repeatable, can the treatment plan be further advanced. This approach changes the purpose of strengthening. Strength becomes a tool for achieving mechanical changes: improving the stability and efficiency of the knee joint bearing in daily life. This framework also explains why some patients "failed" to complete the standard training plan. They did not fail in the exercise aspect; they failed in acquisition and control. Therefore, intervention measures must target the bottleneck issues - activation pathways, the accuracy of execution, or the preparatory state of the central nervous system.

The integration of cerebral signals with muscular signals should not be heralded as an established, superior therapy. At present, it is best understood as a precision-oriented rehabilitation framework—one that demands stringent, methodologically sound validation. It must be appraised with due responsibility, and any observed outcomes should cohere with the mechanistic claims it purports to support. Pain relief and functional improvement are, of course, indispensable endpoints; yet measures that capture load tolerance and vulnerability to symptom flare-ups should likewise be incorporated [15]. It is essential to determine deliberately whether movement strategies have truly been reshaped—not merely whether numerical strength values have risen. For if the characteristics of the applied load remain unchanged, there is little reason to anticipate reliable, durable clinical transformation.

Most importantly, if one wants to indicate that this research is related to the common microenvironment,

it cannot rely solely on words. It should be measured in feasible situations, the relevant end-point indicators related to the microenvironment, and the progress of improved execution methods and the development of state perception should be tested to see if they can reduce the frequency of attacks and improve the stability of symptoms over time [15].

When the rehabilitation of knee joint osteoarthritis is regarded as an integrated systemic issue, the rehabilitation effect will be more significant, and the rehabilitation process will be more stable: the central state affects the execution, the execution determines the load, and the load interacts with the joint microenvironment. When the strengthening training plan fails, the real obstacle is not the degree of effort, but the neuromuscular transformation ability - specifically including the accessibility of activation, the control ability of coordinated contraction, and the readiness to learn movement in the presence of pain.

By combining electroencephalography and electromyography, the goal is to make the strengthening process have a clear mechanistic nature: ensure that patients can implement the expected mobilization strategies and safely develop towards the functional load pattern that the knee joint can bear. This is a promising direction in the precise rehabilitation of knee joint osteoarthritis, and it is believed that its value will depend on carefully designed, phenotype-based trials that can link the central state, muscle execution, and clinically significant results.

REFERENCES

1. GBD 2021 Osteoarthritis Collaborators. Global, regional, and national burden of osteoarthritis, 1990-2020 and projections to 2050: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Rheumatol. 2023; 5: e508-e522.
2. Moseng T, Vliet Vlieland TPM, Battista S, Beckwée D, Boyadzhieva V, Conaghan PG, et al. EULAR recommendations for the non-pharmacological core management of hip and knee osteoarthritis: 2023 update. Ann Rheum Dis. 2024; 83: 730-740.
3. Wan Y, McGuigan P, Bilzon J, Wade L. Knee loading and joint pain during daily activities in people with knee osteoarthritis: A systematic review and meta-analysis. Clin Biomech (Bristol). 2025; 122: 106433.
4. Princelle D, Viceconti M, Davico G. EMG-Informed Neuromusculoskeletal Simulations Increase the Accuracy of the Estimation of Knee Joint Contact Forces during Sub-optimal Level Walking. Ann Biomed Eng. 2025; 53: 1399-1408.
5. Dennis PA, Anderson L, Coffman CJ, Webb S, Allen KD. Exploration of heterogeneity of treatment effects across exercise-based interventions for knee osteoarthritis. Osteoarthr Cartil Open. 2025; 7: 100571.
6. Tang S, Zhang C, Oo WM, Fu K, Risberg MA, Bierma-Zeinstra SM, et al. Osteoarthritis. Nat Rev Dis Primers. 2025; 11: 10.
7. Bi AS, Sachs JP, Mufti YN, Franzia CH, Jesse JE, Lemme NJ, et al. Why do osteoarthritic knees hurt? Leveraging the impact of load for symptom management. J Cartilage Joint Preservation. 2025.
8. Uhlrich SD, Mazzoli V, Silder A, Finlay AK, Kogan F, Gold GE, et al. Personalised gait retraining for medial compartment knee osteoarthritis: a randomised controlled trial. Lancet Rheumatol. 2025; 7: e708-e718.
9. Taniguchi T, Tanaka S, Nishigami T, Imai R, Mibu A, Yoshimoto T. Relationship between fear-avoidance beliefs and muscle Co-contraction in people with knee osteoarthritis. Sensors. 2024; 24: 5137.
10. Rashid SA, Hussain ME, Bhati P, Veqar Z, Parveen A, Amin I, et al. Muscle activation patterns around knee following neuromuscular training in patients with knee osteoarthritis: secondary analysis of a randomized clinical trial. Arch Physiother. 2022; 12: 19.
11. Gokeler A, Neuhaus D, Benjaminse A, Grooms DR, Baumeister J. Principles of Motor Learning to Support Neuroplasticity After ACL Injury: Implications for Optimizing Performance and Reducing Risk of Second ACL Injury. Sports Med. 2019; 49: 853-865.
12. Zhang LB, Chen YX, Li ZJ, Geng XY, Zhao XY, Zhang FR, et al. Advances and challenges in neuroimaging-based pain biomarkers. Cell Rep Med. 2024; 5: 101784.
13. Murray GM, Sessle BJ. Pain-sensorimotor interactions: New perspectives and a new model. Neurobiol Pain. 2024; 15: 100150.
14. Roos EM, Kroman S, Ageberg E. An Exercise Therapists' Guide to Neuromuscular Exercise for People with Knee or Hip Osteoarthritis. J Orthop Sports Phys Ther. 2025; 55: 1-9.
15. Queiroga F, Epstein J, Erpelding ML, Spitz E, Maillefert JF, Fautrel B, et al. The Flare-OA-16 questionnaire measuring flare in knee and hip osteoarthritis in the patient perspective: scale reduction and validation using a Rasch model. J Clin Epidemiol. 2024; 174: 111488.