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Neuromechanical Risk Factors for Knee Osteoarthritis: How Altered Muscle Activation and Ground Reaction Forces Drive Joint Degeneration

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ABSTRACT

Knee Osteoarthritis (KOA) is conventionally viewed through the lens of structural cartilage degeneration, yet accumulating evidence positions it as a manifestation of persistent neuromechanical dysfunction. This editorial argues that altered lower-limb muscle activation patterns and resultant abnormal Ground Reaction Forces (GRFs) are critical, underappreciated drivers of joint pathology. ElectroMyoGraphic (EMG) studies reveal characteristic changes, such as quadriceps inhibition and altered gluteal timing, which persist even post-recovery, suggesting maladaptive motor programs are continuously stressing the joint. These neuromuscular deficits translate directly into abnormal kinetic profiles, characterized by elevated vertical loading rates and asymmetric GRFs, which accelerate cartilage micro trauma. Critically, these mechanical signatures of impaired coordination often precede radiographic signs or symptoms. Once established, altered muscle control and abnormal GRFs reinforce each other, creating a vicious cycle that leads to cumulative joint damage. Effective KOA prevention requires a paradigm shift beyond static structural assessment toward dynamic neuromuscular evaluation. Incorporating EMG and GRF analysis into clinical decision-making can facilitate early, targeted interventions, such as gait retraining, offering a path toward restoring physiological joint loading and achieving sustainable long-term joint health.

Keywords: Knee Osteoarthritis; Neuromuscular; Ground Reaction Forces; Muscle Activation; Gait Analysis

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Introduction

Knee OsteoArthritis (KOA) remains one of the leading causes of pain, disability, and reduced quality of life worldwide [1]. Traditionally, its development has been attributed to structural degeneration of articular cartilage, age-related changes, and genetic predisposition. However, growing evidence suggests that KOA should not be viewed solely as a degenerative joint disease, but rather as the long-term consequence of persistent neuromechanical dysfunction. Altered lower-limb muscle activation patterns and abnormal Ground Reaction Forces (GRFs) emerge as critical, yet underappreciated, risk factors that progressively disrupt joint loading and accelerate osteoarthritic changes [2, 3].

Beyond Cartilage: A Neuromechanical Perspective

The mechanical environment of the knee joint is shaped not only by joint anatomy but also by the dynamic interaction between neuromuscular control and external loading. Muscles regulate joint stiffness, absorb impact forces, and modulate GRFs during daily activities such as walking, running, and deceleration [4]. When muscle activation patterns are altered due to injury, surgery, pain, or maladaptive motor strategies the resulting GRF profiles become abnormal, exposing knee joint structures to repetitive, non-physiological loading [5]. Studies consistently demonstrate that individuals at risk of KOA exhibit increased vertical loading rates, altered braking forces, and asymmetric GRF patterns during gait [1, 6]. These kinetic abnormalities are not incidental findings; rather, they represent mechanical signatures of impaired neuromuscular coordination that precede radiographic

osteoarthritis and clinical symptoms [6, 7]

Altered Muscle Activation as a Primary Driver

ElectroMyoGraphic (EMG) investigations have revealed persistent changes in lower-limb muscle activity in populations prone to knee osteoarthritis, including individuals following anterior cruciate ligament reconstruction (ACLR), meniscal injury, or chronic knee pain [8]. Quadriceps inhibition, excessive hamstring co-contraction, delayed gastrocnemius activation, and altered timing of gluteal muscles have all been reported [7, 9]. While such adaptations may initially serve as protective strategies to enhance joint stability, their long-term effect is increased joint compressive force and altered tibiofemoral load distribution (10). Importantly, these neuromuscular alterations often persist even after pain resolution and apparent functional recovery. This silent persistence suggests that abnormal EMG patterns are not merely a response to pain but may represent maladaptive motor programs that continuously expose the knee joint to excessive mechanical stress [7].

Ground Reaction Forces

Ground reaction forces represent the net outcome of neuromuscular control and movement strategy. Changes in GRF magnitude, direction, and temporal characteristics directly influence internal joint moments and cartilage stress [5, 11]. Elevated vertical GRF loading rates and increased braking impulses during gait have been associated with greater cartilage degeneration and faster progression of knee osteoarthritis [5, 6]. Critically, abnormal GRFs are often observed in individuals who demonstrate acceptable clinical outcomes, normal muscle strength, and absence of pain [8]. This dissociation highlights a fundamental limitation of current clinical assessments, which frequently overlook kinetic and neuromuscular variables that drive long-term joint health[1, 9].

The Vicious Cycle of Neuromechanical Dysfunction

Once established, altered muscle activation and abnormal GRFs reinforce each other in a self-perpetuating cycle. Inefficient muscle recruitment increases joint loading asymmetry, while abnormal GRFs further disrupt sensory feedback and motor control (10). Over time, this cycle leads to cumulative microtrauma of articular cartilage, subchondral bone remodeling, and the clinical manifestation of osteoarthritis (11). This neuromechanical model provides a compelling explanation for why knee osteoarthritis frequently develops years after seemingly successful surgical or rehabilitative interventions (1, 8). Restoring joint stability or muscle strength alone is insufficient if physiological loading patterns are not re-established (9).

Implications for Prevention and Clinical Practice

From a preventive standpoint, knee osteoarthritis should be approached as a modifiable outcome rather than an inevitable consequence of aging or injury. Incorporating EMG-based assessments and GRF analysis into rehabilitation and return-to-activity decision-making could enable early identification of individuals at high risk for joint degeneration[6]. Interventions targeting neuromuscular control such as gait retraining, eccentric muscle conditioning, deceleration-focused exercises, and load-modifying strategies hold significant promise in restoring physiological loading patterns [4]. Without addressing these neuromechanical risk factors, preventive efforts remain incomplete and potentially ineffective [1].

Future research and clinical practice must move beyond static structural assessments and embrace a dynamic neuromechanical framework for knee osteoarthritis prevention. Longitudinal studies integrating muscle activation profiles, GRF characteristics, and cartilage health are urgently needed to clarify causal pathways and optimize early intervention strategies [1, 6]. Knee osteoarthritis is not merely a disease of worn cartilage it is the biomechanical consequence of years of altered neuromuscular control and abnormal external loading [1]. Recognizing and addressing these hidden risk factors represents a critical step toward effective

prevention and sustainable joint health.

Conclusion

Knee Osteoarthritis is fundamentally driven not just by cartilage wear, but by persistent, underlying neuromechanical dysfunction. Alterations in lower-limb muscle activation patterns and subsequent abnormal GRFs create a vicious, self-perpetuating cycle of excessive and non-physiological joint loading. This framework suggests that success in preventing or managing KOA depends critically on moving beyond traditional structural assessments to dynamically address neuromuscular control. Future clinical practice must integrate kinetic and EMG analysis to guide targeted interventions, such as gait retraining and load-modifying exercises, ensuring the restoration of physiological loading patterns for sustainable long-term joint health.

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Ethical Permission

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