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Core muscle strength and hip muscle function in knee osteoarthritis: a literature review

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The lumbopelvic-hip complex plays a crucial role in tibiofemoral joint loading through kinetic-chain interactions in knee osteoarthritis (KOA). Dysfunction in proximal muscles, including core and hip musculature, may amplify mechanical stress at the knee joint, yet evidence synthesising these relationships remains limited. This review aimed to update the evidence on the biomechanical impact of lumbopelvic-hip and core muscle function on gait and proximal compensatory strategies in individuals with KOA. Methods. Electronic databases, including PubMed, PEDro, Cochrane, Elsevier/Science Direct, NoteKLM, and Google Scholar, were searched for publications from January 2020 to January 2026. While risk of bias was assessed independently using the ROBINS-I framework. Results. Fourteen cross-sectional observational studies were included. Evidence on neuromuscular control and gait studies is trustworthy with moderate risk of bias. Findings on kinetic chain asymmetry, lumbopelvic-hip and pelvic tilt, and muscle synergy adaptation derive predominantly reported in studies with serious risk of bias due to uncontrolled confounding. Conclusion. These findings suggest that KOA rehabilitation should integrate kinetic-chain knee exercises combined with lumbopelvic-hip muscle function and core stabilisation training to address the alteration of knee loading. Future longitudinal research with larger sample sizes and standardised biomechanical protocols is recommended.

Попереково-тазово-кульшовий комплекс відіграє вирішальну роль у навантаженні на колінний суглоб через взаємодії в кінематичному ланцюзі в разі остеоартрозу колінного суглоба (КОА). Дисфункція проксимальних м'язів, зокрема м'язів кора та кульшового суглоба, може посилювати механічне навантаження на колінний суглоб, проте узагальнені докази щодо цих взаємозв'язків залишаються обмеженими. Метою цього огляду було оновити дані щодо біомеханічного впливу функції попереково-тазово-кульшового комплексу та м'язів кора на ходу та проксимальні компенсаторні стратегії в осіб з КОА. Методи. Проведено пошук публікацій в електронних базах даних, зокрема PubMed, PEDro, Cochrane, Elsevier/ScienceDirect, NoteKLM та Google Scholar, за період із січня 2020 року до січня 2026 року. Ризик систематичної помилки незалежно оцінювали за допомогою інструмента ROBINS-I. Результати. До огляду було включено 14 перехресних обсерваційних досліджень. Дані щодо нейром'язового контролю та вивчення ходи були достатньо надійними та характеризувалися помірним ризиком систематичної помилки. Проаналізовано асиметрію кінематичного ланцюга, нахил попереково-тазово-кульшового комплексу та таза. Адаптації м'язових синергій переважно були отримані в роботах із серйозним ризиком систематичної помилки через неконтрольоване змішування факторів. Висновок. Ці результати свідчать про те, що реабілітація за КОА повинна поєднувати вправи для кінематичного ланцюга нижньої кінцівки з тренуванням функції м'язів попереково-тазово-кульшового комплексу та стабілізацією тулуба для врахування змін навантаження на колінний суглоб. Рекомендується проведення подальших поперевжних досліджень із більшими вибірками та стандартизованими біомеханічними протоколами. Ключові слова. Біомеханіка; стабільність тулуба; функція м'язів стегна; реабілітація в разі остеоартрозу колінного суглоба; попереково-тазовий контроль.

Keywords. Biomechanics; core stability; hip muscle function; knee osteoarthritis rehabilitation; lumbopelvic control

Introduction

Knee osteoarthritis (KOA) is a chronic, degenerative joint condition characterised by progressive structural damage to multiple tissues of the knee joint, involving articular cartilage, menisci and subchondral bone in older adults [1]. Approximately one quarter of individuals with KOA are over 55 years; 39 % are among those aged over 65 years [2, 3]. KOA presents clinically as pain and weakness, rigidity, and functional impairment, with morphological degradation observable by imaging techniques [4, 5]. A previous study reported KOA is the fourth and eighth leading causes of disability for women and men, respectively [6]. In East and Southeast Asia, the burden of KOA has escalated sharply. High-income Asia Pacific countries, namely Singapore, South Korea, and Japan, record the highest per-capita figures. Populous nations such as China and India drive the greatest overall volume [7, 8]. It's estimated that by 2035, the global KOA incidence, prevalence, and DALYs are expected to rise by 33–44 %, nearly doubling by 2050 [9, 10].

The site of knee pain in KOA is related to the primary structural deterioration within the joint and increased angulation of mechanical forces in the position. For example, pain felt in the medial compartment is caused by uneven pressure on the inner part of the knee joint [11]. In normal walking, the medial compartment allows approximately 60–70 % of total tibiofemoral contact force; whereas in varus malalignment, which coexists with an elevated knee adduction moment, this proportion rises further, sustaining the cycle of medial cartilage degradation and subchondral bone remodelling observed in progressive disease [12]. For example, lateral compartment pain is linked to excessive dynamic knee valgus during weight-bearing, causing lateral patellar maltracking, while iliotibial band tightness increases compressive forces on the lateral tibiofemoral and patellofemoral compartments, often differing from the medial phenotype clinically and radiographically [13, 14]. In some cases, biochemical and microstructural changes occur before the appearance of radiographical abnormalities and are associated with clinical symptoms and progressive disease [15]. The trend highlights the importance of early detection and personalized treatment in potentially reducing the risk of chronic arthritis development.

From a pathological perspective, KOA is caused by an active imbalance between proinflammatory and anti-inflammatory cytokines, resulting in abnormal metabolism in knee articular cartilage [16].

This reduces cartilage elasticity, cartilage synthesis and degradation, and eventual fissuring and erosion of the articular surface [5], leading to a gradual loss of proteoglycans and collagen [17]. As the disease advances, the development of osteophytes and subchondral bone remodeling causes further deterioration of joint mechanics and load distribution [18]. After these structural changes, neuromuscular adaptations occur, such as arthrogenic quadriceps inhibition and gait deviations, worsening of chronic pain and stiffness, and further reduction in functional capacity [5]. These adaptations are not only local; they can represent a general, compensatory mechanism needed to maintain mobility in the presence of the progressive deterioration of the joint [19]. Restricted knee flexion due to capsular stiffness and structural joint changes like osteophyte formation, which can cause pain, are all examples of pain-related inhibition [20].

The lower limb is part of a kinetic chain from the lumbar spine and pelvis to the knee joint from an anatomical and biomechanical standpoint. The psoas major muscles from the lumbar to pelvic attach to the femur bone, while the quadriceps, hamstrings, and sartorius are important for controlling the relationship of trunk, pelvis, and lower limbs. The "core" muscles, superficial and deep, of the lumbopelvic and lumbar areas have a central role in postural stability and force transmission between the upper and lower limbs [21]. Think of the lower limb as a chain of interconnected links. If one is weak or misaligned, the load is transferred to neighbouring links. In KOA, the compromised knee redistributes load upward to the pelvis and spine. Balance impairments reflect altered proprioception and compensatory neuromuscular strategies adopted to minimise joint loading [5]. Dysfunction in any of these proximal segments may increase mechanical stress at the distal knee joint. For instance, a decline in lumbar extensor muscle strength and endurance is associated with impaired balance and functional performance among older adults with KOA compared with healthy controls [22]. In addition, an increase in trunk flexed posture may further contribute to impaired core muscle function, hip and knee extensors, presenting in daily tasks such as sit-to-stand and walking [23, 24].

Posterior pelvic tilt, often linked to weak lower back muscles or tight hip flexors, reduces the natural curve in the lower back and moves the body's center of mass backward [25]. The resultant augmentation in knee flexion moment at mid-stance amplifies patellofemoral compressive stresses and necessitates heightened quadriceps exertion throughout the gait cycle [24]. Anterior pelvic tilt, associated with lum-

bar hyperlordosis and hip flexor dominance, modifies ground reaction force vectors because of the augmented hip flexion angle at initial contact; this subsequently intensifies the knee adduction moment and increases medial compartment loading [24]. In the case of lateral pelvic tilt, which arises from inadequate function of the gluteus medius muscle, the pelvis on the opposite side drops when standing on one leg [26]. This shift in position moves the center of mass of the trunk toward the body's center. This increases the length of the lever arm in the front-to-back direction, which then increases the forces acting on the medial part of the knee joint [13, 27], subsequently accelerating disease progression [28]. Each lumbopelvic deviation represents a unique mechanical pathway through which proximal dysfunction affects the knee joint.

In addition to trunk-related deficits, substantial evidence highlights the role of hip musculature, particularly the hip abductors and external rotators, in regulating knee mechanics in the frontal and sagittal planes, respectively [29]. Weakness in hip abductors and flexor and extensor muscle groups has been related to pelvic drop, compensatory trunk lean, dynamic knee valgus, and increased medial knee joint loading during gait [30]. Hip extensor weakness may further alter trunk posture and increase quadriceps demand, thereby exacerbating joint compression during weight-bearing activities [31]. Collectively, these findings suggest that dysfunction of the lumbopelvic and proximal hip musculature contributes to abnormal knee loading, pain, and functional decline in individuals with KOA. These findings suggest alteration of lumbopelvic sagittal alignment in individuals with KOA, exhibiting trunk flexion, posterior pelvic tilt, a center of body mass shift anteriorly, and being predisposed to higher medial tibiofemoral loading and knee flexion contracture [32, 33].

Conventional physiotherapy management for KOA has traditionally focused on localised knee impairments. These include knee pain, range of motion, and knee extensor and flexor muscle strength training. Narrative reviews have highlighted the importance of trunk and hip musculature in knee joint loading. There remains a paucity of evidence examining the role of core muscle function within rehabilitation interventions for KOA [34]. Accordingly, rehabilitation approaches that integrate targeted core strengthening alongside conventional knee- and hip-focused exercises may offer a more comprehensive strategy for addressing both local impairments and proximal kinetic-chain dysfunction contributing to KOA-related disability [21, 24, 34].

Contemporary clinical guidelines consistently recommend exercise therapy, patient education, and weight management as first-line interventions, with manual therapy and physical modalities used primarily as adjuncts [35, 36]. Weight reduction of approximately 10 % or more has been shown to significantly reduce symptoms [37]; however, a substantial proportion of individuals with KOA continue to experience persistent pain and functional limitations if receiving conventional therapy alone [38]. This discovery indicates that components beyond localised knee pathology may affect clinical outcomes, highlighting the necessity for clinicians to examine the lower limb within a more comprehensive kinetic-chain context. A more comprehensive understanding of the interplay between the biomechanics of the proximal kinetic chain, including core muscle and hip muscle function, and its association with clinical outcomes in individuals afflicted with KOA. Hence, this review aimed to update the current evidence-based physiotherapy rehabilitation protocols to minimise chronicity and disability caused by KOA.

Methodology

An integrative review methodology was used to review and synthesize existing findings regarding the biomechanical and anatomical links between knee osteoarthritis (KOA), lumbopelvic–hip function and the performance of core muscles. The methodological quality assessment and systematic search procedure were used to improve the rigor and transparency of the review process, but the integrative design was chosen to allow for inclusion and synthesis of evidence from observational biomechanical studies, which may not be appropriate for a traditional systematic review.

An extensive electronic database literature search was carried out across PubMed, PubMed Central (PMC), PEDro, the Cochrane Library, Google Scholar, NoteKLM, and Elsevier/ScienceDirect. Studies were searched from January 2020 to January 2026. Search terms were made of combinations of keywords focused on knee osteoarthritis, biomechanics, anatomical aspects, lumbopelvic–hip muscles, and core muscles. The search terms were combined using Boolean operators ("AND" and "OR") as per the search requirements of each database. Only full-text articles in the English language and those with human subjects were searched. The reference lists of eligible studies and relevant review articles were hand-searched for any other publications that fulfilled the inclusion criteria in addition to the electronic database search.

Studies were only included if they were original observational studies in adults with clinically and/or

radiographically diagnosed KOA that reported a biomechanical or clinically relevant outcome. Eligible outcomes were any measure of gait biomechanics, lower-limb kinematics or kinetics, muscle activation patterns, lumbopelvic and/or trunk function, core muscle endurance and/or strength (if measured), balance, mobility, physical function, pain intensity or health-related quality of life. Studies with non-KOA conditions (e.g., ligament injuries or fractures), without a confirmed diagnosis of KOA (e.g., post-surgical rehabilitation), pharmacological or surgical intervention only, narrative reviews, systematic reviews, case reports, conference abstracts without full text information, and those that could not reliably distinguish the contribution of the lumbopelvic or core musculature from general whole body exercise were excluded.

Two independent reviewers assessed methodological quality based on an adapted domain-based framework created for the Risk of Bias in Non-randomized Studies of Interventions (ROBINS-I) tool. The ROBINS-I domains were used for the included biomechanical studies as they were observational in nature, and because of this, possible sources of methodological bias within non-randomized observational studies were assessed, such as bias due to confounding, participant selection, classification of the exposure, deviations from the intended exposure, missing data, outcome measurement, and selection of reported results. All domains were assessed as low, moderate, serious, or critical risk of bias. Discussions were held among the reviewers when there were any disagreements to reach a consensus.

Information regarding the biomechanical and anatomical relationship between KOA, lumbopelvic/hip function, and core muscle performance was synthesized using an integrative review methodology. The reporting process was done using Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to maximize methodological transparency and reproducibility, even though there was a wide variety of observational evidence included in the integrative review.

Results

The observational studies included in this study contained cross-sectional biomechanical studies using motion capture, force platform, electromyography, musculoskeletal modelling, and radiographic imaging that assessed biomechanical features of KOA (STable 1). Across the studies, biomechanical outcomes measured involved trunk posture, spinopelvic alignment, gait mechanics, lower-limb kinematics and kinetics, joint loading, muscle activation patterns, and

intersegmental coordination in adults with clinically and/or radiographically diagnosed KOA. These studies together allowed insight into the adaptations of the kinetic chain in KOA and laid a foundation for understanding the impact of lumbopelvic–hip biomechanics on disease-related movement impairments (SFigure 1). The most common methodological issues identified in the included observational studies, across the adapted ROBINS-I framework, were bias caused by confounding (STable 2). This domain was given several studies serious ratings due to the fact that important covariates (BMI, level of physical activity, disease severity, and coexisting MS conditions) were not measured or statistically controlled consistently across studies. This is not necessarily indicative of poor study conduct, nor a deficiency of the studies included, as this is a common drawback of observational studies. However, bias associated with outcome measurement was generally considered low, due to the use of objective biomechanical assessment methods such as three-dimensional motion analysis, force platforms, electromyography, and standard imaging methods.

While, narrative synthesis involved arranging the included 14 studies into five domains of biomechanics: kinetic chain asymmetry ($n = 3$), lumbopelvic–hip and pelvic alignment ($n = 3$), muscle synergy adaptation ($n = 2$), neuromuscular control ($n = 2$), and lumbopelvic–hip influences on gait ($n = 6$); two studies contributed to more than one thematic category because they applied the same study sample to more than one biomechanical outcome. These thematic categories were also considered with the risk-of-bias findings to give background information on the quality of evidence in the respective areas (STable 3).

Singh et al., [39] presented the lowest overall risk of bias with moderate risk owing to partial adjustment for relevant confounding variables, within the kinetic chain asymmetry category. Bakker et al., [40] and [28] were rated as having a serious overall risk of bias, mainly due to the lack of adequate control of potential confounding factors. Therefore, interpretation of evidence of asymmetrical loading patterns and contralateral compensatory mechanisms should be treated with proper precaution. In the studies of lumbopelvic–hip and pelvic alignment, [41] had the highest quality of methodology and received a moderate rating for overall quality. Fu et al., [33] reported a higher overall risk of bias because of the potential risk of confounding and the potential risk of participant selection, whereas the overall risk of bias of [42] was mainly high due to the lack of attention to potential confounding variables. These studies all showed associations between KOA and changes in spinopelvic

alignment, but the limitations of the studies need to be taken into account when interpreting the findings.

All the studies in the muscle synergy adaptation category [16, 43] had a serious overall risk of bias due to the lack of attention given to key confounding variables, such as BMI and other coexisting musculoskeletal health conditions. Thus, their results offer preliminary insight into changes in muscle activation and co-activation patterns, but should be confirmed in future studies that have more methodological control. The quality of the methodological performance was relatively good in the category of neuromuscular control. Ana Luiza Costa e Silva Cabral, and Petrella et al., studies [44, 45] assigned moderate overall risk ratings, which indicated more detailed reporting of methodological aspects and a better consideration of potential confounding variables. Thus, these studies represent more credible evidence of the neuromuscular adaptations and how movement is controlled in people with KOA. In the lumbopelvic–hip influences on gait category, there were moderate overall ratings for [44–46]; this is the best available evidence for this category. The overall risk of bias for [2, 47, 48] was serious due, in part, to uncontrolled confounding. However, the changes in gait biomechanics and trunk posture that were found in all these studies were suggestive of KOA but not conclusive.

Discussion

Across the observational literature, a consistent pattern emerges in which biomechanical adaptations extend proximally and distally from the knee joint rather than functioning as a purely local degenerative process. KOA appears to involve compensatory adjustments across multiple segments of the lower kinetic chain.

Kinetic Chain Asymmetry

Observational evidence is firmly in favor of a coordinated change along the lower-limb kinetic chain, beyond a single joint, with biomechanical changes in KOA not being just an isolated degenerative process. Changes in gait mechanics, hip muscle activity, pelvic alignment, and trunk posture suggest that a compensatory strategy is created across several body segments in order to maintain locomotor function in the face of pain and joint dysfunction. A number of biomechanical analyses showed that mechanical loading is shifted away from the affected knee via proximal and contralateral compensatory mechanisms. Wang et al., [28] found that hip contact forces and gluteus medius activation in the contralateral leg increased with increasing severity of KOA, indicating that the hip and pelvis play an active role in load redistribution during gait. These

changes are not just knee-related but also support the idea that there is a lower-limb kinetic chain effect of KOA. The findings of the included studies suggest the same: When knee function is impaired, the proximal musculature plays a role in maintaining dynamic stability and forward movement.

A major conclusion from the existing evidence is that there are sex-related differences in biomechanical compensation. Singh et al., [39] reported that males were more likely to show varus knee alignment while females showed greater changes in spinopelvic parameters such as pelvic incidence–lumbar lordosis mismatch. The results indicate that anatomical features and neuromuscular control strategies may be factors affecting the development of compensatory mechanisms in people with KOA. The available evidence does not give women and men the same evidence-based rehabilitation strategies, but it does provide support for the importance of individual biomechanical assessment and not treating all patients as having the same compensation pattern. Clinical movement assessments, such as gait analysis, single-leg squat performance, and functional balance testing, might therefore help to detect movement impairments in individual patients prior to prescribing rehabilitation strategies.

The available evidence also suggests that compensatory loading is not uniform within the population of people with KOA. Contrary to studies that show loading patterns are asymmetrical, [40] found no significant interlimb differences in frontal plane knee and hip moments during gait. The results do not contradict the overall biomechanical model, but rather they reflect the diversity of compensatory strategies and may indicate that the extent of kinetic-chain compensation depends on the severity of pain, structural disease progression, walking strategy, and individual neuromuscular adaptations. Therefore, the lack of any quantitative asymmetry in some patients should not be regarded as normal biomechanical function but could be part of the variability of disease presentation or adaptive movement strategies. The majority of studies included were cross-sectional; however, it is unclear whether these compensatory mechanisms change over the course of KOA. Longitudinal studies are needed to explore the changes in biomechanical adaptations as the disease progresses, and whether early compensation strategies help to predict later limitations in function.

The evidence presented together points to a systems-based view of KOA, in which a combination of altered loading of the lower limb, proximal muscle activation, pelvic alignment, and intersegmental co-

ordination work together to maintain mobility while the joints degrade over time. These adaptations do not seem to be biomechanical dysfunctions but rather a pattern of coordinated compensatory responses along the lower-limb kinetic chain. This view is not limited to the traditional joint-centered approach and emphasizes evaluating the quality of movement in the lumbopelvic/hip complex as well as the knee.

The observational studies in this review do not demonstrate treatment efficacy, but they all conclude that modifiable biomechanical deficiencies may be suitable rehabilitation targets. There is stronger evidence for the clinical relevance of these findings. According to the results of the network meta-analysis by [49], which included 139 randomised controlled trials, active rehabilitation methods (such as structured exercise therapy and hydrotherapy) showed greater pain and physical function improvements than passive physical methods. Their findings revealed that exercise therapy ranked consistently among the most effective modalities for pain reduction (SUCRA: 22.8 %) and functional improvement (SUCRA: 32.6 %), while hydrotherapy demonstrated the highest probability of overall WOMAC improvement. Likewise, a large, recent network meta-analysis of 217 RCTs with more than 15,000 patients with KOA showed that exercise therapy was consistently beneficial for pain, physical function, and health-related quality of life, thus highlighting the importance of active rehabilitation in contemporary conservative management [50]. The current review data, combined with these findings, support the clinical importance of the biomechanical impairments identified and rehabilitation programs designed to include lumbopelvic control, hip muscle strengthening, and neuromuscular coordination, in addition to traditional knee-focused rehabilitation. However, the current evidence that biomechanical impairments are associated with rehabilitation outcomes is mostly observational, and further well-designed prospective and interventional studies are needed to identify which patients benefit the most from targeting interventions at the kinetic chain.

Pelvic tilt in relation to lumbar posture in KOA

There is increasing evidence to support the treatment of KOA as a disorder of the whole body sagittal alignment and not just the knee. The observation-based studies that were part of this review have all shown that the structural changes of the knee are linked to changes in the orientation of the pelvis and the posture of the spine, thus supporting the concept of a lumbopelvic–lower limb kinetic chain. Katsumi et al., [41] found that the femoral tilt angle (FTA) is a key factor associated with overall sagittal bal-

ance in KOA patients. In their study, they found that as the knee becomes flexed forward, the pelvis tilts backward and the lumbar lordosis decreases, showing that biomechanical changes are present in more proximal levels to maintain upright posture. These findings are in line with the idea that compensatory postural adjustments are whole-body responses to altered knee mechanics and not a local phenomenon of the lumbar spine or pelvis. Therefore, changes in loading through the axial skeleton due to increased retroversion of the pelvis and altered lumbar alignment could result in alterations in the demands of trunk muscles and postural control. While these studies are not explicit about low back pain, altered spinopelvic alignment has been suggested to be a plausible mechanism of altered biomechanics that may contribute to the common occurrence of lumbar complaints in individuals with KOA, which should be examined in future prospective studies.

The connection between the spine and the knee was also illustrated by [33], who performed full-body EOS imaging to assess global sagittal alignment. Pelvis tilt and higher pelvic incidence, lumbar lordosis (PI-LL) mismatch also showed an altered knee alignment with a tendency towards compensatory knee flexion. The results support the idea of sagittal imbalance as a global biomechanical adaptation in which the changes in each segment are coordinated instead of independent segmental problems. Therefore, spinopelvic alteration can either have an effect on or be a consequence of the compensatory movement strategies used for progressive knee degeneration.

This relationship is also supported by [42], who reported evidence from radiographs. There was a significant difference in posterior pelvic tilt between more and less advanced KOA and also between more and less advanced KOA and the PI-LL mismatch, indicating that posterior pelvic tilt increases with structural joint degeneration and that the PI-LL mismatch becomes greater as KOA progresses. These findings are only cross-sectional and therefore only show associations and do not prove the causal relationship. However, the uniformity of the observations noted in the various independent studies supports the hypothesis that sagittal alignment and KOA are biomechanically related. It is not yet known whether changes in spine alignment are a cause of KOA or a result of knee injury, or if these changes occur as a result of the interplay between the two.

These results all point to the need for a whole-body analysis of KOA. When evaluating and rehabilitating patients with knee pathology, clinicians should take into account the roles of spinopelvic alignment

and lumbopelvic function. This interpretation is supported by recent systematic evidence [50] that multi-component KCTB programs have a positive effect on pain, physical function, and quality of life in people with KOA. The best available biomechanical evidence suggests that postural assessment, lumbopelvic stability, and trunk control should be part of a broader rehabilitation program and may be useful components of various intervention studies in clinical practice once the direct therapeutic effect is established through prospective studies.

Muscle Synergy Adaptations

Existing evidence indicates that alterations in neuromuscular coordination are linked to the development of KOA, altered strategies of muscle activation during functional movements. These results are not the only evidence for lower-limb muscle weakness in KOA, but suggest a reorganisation of motor control along the whole of the lower-limb kinetic chain that could affect movement efficiency and dynamic stability. Roelker et al., [43] found that people with more severe KOA had fewer muscle modules during walking than healthy adults, suggesting reduced neuromuscular complexity in them. The decrease in muscle synergies indicates that more muscle groups are used together more closely, resulting in a more limited motor control strategy in walking. This simple organization of the neuromuscular system could be an adaptive reaction to pain, joint instability, or poor proprioceptive input, allowing for functional mobility when there are structural alterations occurring within the joint [51]. Such adaptations may, however, enhance joint stability during the initial stages of the movement, but may also reduce movement adaptability and put a greater mechanical load on the adjacent joints when they are used repetitively. These muscle synergies are altered, but the contribution of these changes to the progression of disease or their association with normal biomechanical adaptation is not known at this time, since these are cross-sectional data.

Liu et al., [52] also found that there are strong relationships among muscle stiffness, co-activation, and balance in older adults with KOA. The authors conclude that the functional implications of co-activating muscles may vary depending on the type of movement demands, and that the neuromuscular responses may vary across the force generation, controlled deceleration, and postural regulation phases of the movement. The evidence does not suggest that the co-activation of muscles is always good or bad, but rather that it is dependent on the task being undertaken and its biomechanical function. There are potential implications related to rehabilitation, such that

interventions aimed at optimizing movement quality and task specificity should account for increases or decreases in muscle activation.

These studies suggest that neuromuscular adaptations to KOA are more complex than muscle strength changes and that they are also manifested as altered motor control strategies, which impact dynamic stability and movement efficiency. The stability across all these studies justifies adding task-specific functional exercises and motor control retraining of the lumbopelvic-hip musculature to traditional strengthening exercises within the framework of rehabilitation. The present results show a biomechanical rationale to use neuromuscular training as part of a thorough KOA rehabilitation.

Neuromuscular Control

Based on the observational evidence, the neuromuscular dysfunction associated with KOA not only includes muscle weakness but also disturbances in motor control, movement coordination, and functional stability. The studies in this collection indicate that people with KOA use alternative (modified) neuromuscular strategies to preserve functional performance despite pain, joint deterioration, and reduced mechanical efficiency. Petrella et al., [44] showed that lower-limb kinematics, kinetics, and muscle activation were altered during the sit-to-stand task in individuals with unilateral and bilateral KOA, suggesting that neuromuscular adaptations occur during other functional weight-bearing activities, not just when walking. These results highlight that movement impairments in KOA are associated with changes in the coordination of multiple lower-limb muscle groups in challenging functional tasks, which is consistent with the idea that the neuromuscular dysfunction in KOA is related to both force production and movement organisation. This means programs that concentrate only on strengthening may not be sufficient to meet the motor control deficits of KOA. The addition of functional tasks challenging dynamic stability, intersegmental coordination, and/or movement quality may therefore be a source of added therapeutic benefit.

The changes in movement strategies are probably multifactorial. A structural degeneration of mechanoreceptors in the KOA may contribute to proprioceptive dysfunction, which [53] have noted. Decreased accuracy of JPS could affect sensorimotor integration and increase the use of compensatory neuromuscular strategies for stability during functional tasks. This theory is also supported by the observation of [43] finding simplified muscle synergy patterns, and [52] showing a relationship between muscle co-activation,

muscle stiffness, and balance performance in older adults with KOA.

The evidence presented above indicates that neuromuscular dysfunction in KOA should not be considered as a muscle weakness per se but rather as an integrated sensorimotor dysfunction. The changes in proprioceptive input, muscle activation strategies, and co-activation are adaptive responses to obtain joint stability during mechanically compromised conditions. While these compensatory strategies can provide short-term stability, they can also limit the ability to move and increase loading in the lower-limb kinetic chain. However, since the current evidence is largely based on cross-sectional observational studies, the direct role these neuromuscular adaptations play in disease progression remains unknown, as they may simply be compensatory mechanisms.

Lumbopelvic & Hip affect gait, Gait Biomechanics, and Intersegmental Coordination in KOA

One of the most consistently reported functional features of KOA is altered gait biomechanics. In all of the included observational studies, people with KOA tended to have a slower walking speed, a shorter step length, a different coordination pattern of the lower limbs, and a different pattern of trunk mechanics during walking than healthy adults. These gait changes are not simply due to isolated gait changes, but rather represent coordinated compensatory adaptations that shift the mechanical demands along the lower-limb kinetic chain in response to pain, joint degradation, and decreased mechanical efficiency.

Studies using motion analysis and musculoskeletal modelling have shown that these adaptations are not restricted to the affected joint but are a result of the coordination of the changes across the hip, knee, pelvis, and trunk. In stance and swing phases, [47] showed that there was more variability in thigh-shank coordination, with less consistency in movement coordination between segments. These findings are consistent with the simplified muscle synergy patterns reported by [43] and show that altered gait in KOA is related to the changes in the neuromuscular organisation as well as the changes in the joint mechanics. It seems as though the more movement forward is required while trying to stabilise a painful knee, the more complex the compensatory motor strategy of multiple body segments that is required.

Suzuki et al., [48] further investigated gait-related compensations and showed that kinetic changes occurred in advance of the appearance of postural compensation. In particular, a change in the contribution of knee flexion moments to knee adduction moments was seen in subjects with moderate KOA, while in-

creased forward trunk flexion was noted in advanced KOA. This observation indicates that the kinetic changes may occur first, before more apparent postural changes, but due to the cross-sectional nature of this data, a longitudinal study would be warranted to corroborate changes over time. In the same way, [28] found higher contralateral hip contact forces and higher gluteus medius muscle forces along with higher gait asymmetry in more severe disease. The results indicate that progressive redistribution of mechanical loading could be a significant compensatory mechanism in the progression of disease and could be a helpful biomechanical marker of functional decline, though its implication in prognosis needs to be proven.

Further evidence from perturbation-based studies shows that adaptations in gait occur beyond the steady-state walking condition. [2] found that people with KOA have shorter recovery steps and lower ground reaction forces after slip perturbations, suggesting a possible increased risk of falls in people with KOA due to impaired reactive balance control. However, [46] found a relatively small change in biomechanics for frontal plane gait perturbations, with broadly similar adaptation across participants with KOA and healthy older adults. Combined, these data indicate that although some reactive movement impairments may be linked to KOA, others could be a reflection of age-related adaptations and not necessarily disease-specific neuromuscular changes.

Compensatory movement strategies were also observed in activities other than level walking. Petrella et al., [44] found that bilateral KOA patients also had a decrease in knee extensor moments and an increase in trunk flexion when completing a sit-to-stand, suggesting that proximal body segments were used to compensate for decreased knee function. Likewise, [45] found higher degrees of activation in the tibialis anterior, gastrocnemius, and gluteus medius in the single-leg squat, indicating the neuromuscular demand to achieve lower limb stability while performing weight-bearing exercises. Given the consistency of these results across the various functional activities, altered lumbopelvic and hip mechanics are not only seen during gait but also reflect compensatory movements across the various functional activities of daily living.

Taken together, the evidence suggests that gait dysfunction in KOA is more than simply a decrease in walking speed or dysfunction at the knee. Intersegmental coordination is altered, mechanical loading is asymmetric, the muscles of the proximal segments are more involved, and the mechanics of the trunk

are modified; all of this points to a whole-body biomechanical response to progressive joint degeneration. The results of this study support the idea that the lumbopelvic–hip complex is crucial for the maintenance of locomotor stability in the presence of altered knee mechanisms. The clinical implications of this body of evidence are that rehabilitation programs aimed at improving gait, neuromuscular control, strengthening the hip, lumbopelvic stability, and functional movement retraining should not be limited to knee strengthening alone. While the studies reviewed here were observational studies that cannot prove the effectiveness of specific rehabilitation techniques, they consistently identified some proximal biomechanical changes that form a strong mechanistic basis for broad kinetic-chain rehabilitation in subjects with KOA.

Limitations of Current Evidence

The findings presented in this review should be interpreted in light of several methodological limitations that temper the strength of conclusions that can be drawn. A considerable proportion of the included observational studies employed cross-sectional designs, which by their nature preclude causal inference regarding the directional relationship between biomechanical alterations and disease progression. A fundamental question remains unanswered: whether lumbopelvic malalignment and simplified muscle synergy patterns develop prior to and contribute to knee degeneration, or whether they emerge secondarily as adaptive responses to pain and progressive structural joint changes. Resolving this directionality will require longitudinal prospective studies that track relevant biomechanical parameters from early-stage KOA through disease progression. This study design remains notably absent from the current evidence base.

Sample size is a recurring limitation across the observation studies included in this review. The majority of studies recruited participants from single clinical sites, and considerable variation in demographic characteristics, including age range, body mass index and habitual physical activity level, makes pooled interpretation across studies difficult to justify with confidence. These methodological gaps introduce the possibility of performance and detection bias influencing reported outcomes.

Conclusion

Biomechanical and neuromuscular dysfunction in the lumbopelvic–hip–knee kinetic chain is associated with KOA and is not a knee-only disease, as shown by this integrative review article. All of the observational studies included in this review consistently

showed alterations in spinopelvic alignment, muscle coordination, gait biomechanics, and intersegmental control, with the changes worsening as the disease progressed. The findings indicate that rehabilitation should involve more than emphasis on local knee strengthening activities, but should include lumbopelvic stability, neuromuscular control, gait retraining, and individual movement assessment. Longitudinal and interventional studies in the future are needed to confirm causal relationships and effectiveness of kinetic-chain-focused rehabilitation strategies.

Conflict of interest. The authors declare no conflict of interest.

References

1. Sharma, L. (2021). Osteoarthritis of the knee. *New England Journal of Medicine*, 384(1), 51-59. <https://doi.org/10.1056/NEJMcip1903768>
2. Ren, X., Lutter, C., Kebbach, M., Bruhn, S., Yang, Q., Bader, R., & Tischer, T. (2022). Compensatory responses during slip-induced perturbation in patients with knee osteoarthritis compared with healthy older adults: an increased risk of falls? *Frontiers in bioengineering and biotechnology*, 10, 893840. <https://doi.org/10.3389/fbioe.2022.893840>
3. Foo, C. N., Manohar, A., Rampal, L., Lye, M.-S., Mohd-Sidik, S., & Osman, Z. J. (2017). Knee Pain and Functional Disability of Knee Osteoarthritis Patients Seen at Malaysian Government Hospitals. *Malaysian Journal of Medicine & Health Sciences*, 13(2), 7.
4. Guede-Rojas, F., Benavides-Villanueva, A., Salgado-González, S., Mendoza, C., Arias-Álvarez, G., Soto-Martínez, A., & Carvajal-Parodi, C. (2024). Effect of strength training on knee proprioception in patients with knee osteoarthritis: A systematic review and meta-analysis. *Sports medicine and health science*, 6(2), 101-110. <https://doi.org/10.1016/j.smhs.2023.10.005>
5. Hsu, H., & Siwiec, R. M. (2023). Knee arthroplasty. StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK507884/>
6. Jordan, K., Arden, N., Doherty, M., Bannwarth, B., Bijlsma, J., Dieppe, P., Gunther, K., Hauselmann, H., Herrero-Beaumont, G., & Kaklamanis, P. (2003). EULAR Recommendations 2003: an evidence based approach to the management of knee osteoarthritis: Report of a Task Force of the Standing Committee for International Clinical Studies Including Therapeutic Trials (ESCISIT). *Annals of the rheumatic diseases*, 62(12), 1145-1155. <https://doi.org/10.1136/ard.2003.011742>
7. Xu, J., Lei, M., & Xu, D. (2025). Global, regional, and national burden of knee osteoarthritis attributable to high BMI: a systematic analysis from 1990 to 2021 and projections to 2050. *Frontiers in Public Health*, 13, 1668257. <https://doi.org/10.3389/fpubh.2025.1668257>
8. Zhou, J., Zhang, Y., Xie, S., Li, Y., Sun, Q., Tang, X., Zhao, T., & Chen, X. (2025). The escalating burden of osteoarthritis in East and Southeast Asia: an information-analytical study from the GBD 2021. *Clinical Rheumatology*, 1-14. <https://doi.org/10.1007/s10067-025-07740-1>
9. Chen, J., Chen, X., Wang, T., Li, M., Dai, H., Shang, S., Cheng, L., Tang, Z., Liu, S., & Huang, C. (2025). Global burden of knee osteoarthritis from 1990 to 2021: Trends, inequalities, and projections to 2035. *PLOS One*, 20(6), e0320115. <https://doi.org/10.1371/journal.pone.0320115>
10. Gill, T. K., Mittinty, M. M., March, L. M., Steinmetz, J. D., Culbreth, G. T., Cross, M., Kopec, J. A., Woolf, A. D., Haile, L. M., & Hagins, H. (2023). Global, regional, and national burden of other musculoskeletal disorders, 1990–2020, and projections to

- 2050: a systematic analysis of the Global Burden of Disease Study 2021. *The Lancet Rheumatology*, 5(11), e670-e682. [https://doi.org/10.1016/S2665-9913\(23\)00232-1](https://doi.org/10.1016/S2665-9913(23)00232-1)
11. McCormack, D. J., Puttock, D., & Godsiff, S. P. (2021). Medial compartment osteoarthritis of the knee: a review of surgical options. *EFORT open Reviews*, 6(2), 113. <https://doi.org/10.1302/2058-5241.6.200102>
 12. D'souza, N., Charlton, J., Grayson, J., Kobayashi, S., Hutchison, L., Hunt, M., & Simic, M. (2022). Are biomechanics during gait associated with the structural disease onset and progression of lower limb osteoarthritis? A systematic review and meta-analysis. *Osteoarthritis and Cartilage*, 30(3), 381-394. <https://doi.org/10.1016/j.joca.2021.10.010>
 13. Rinaldi, V. G., Prill, R., Jahnke, S., Zaffagnini, S., & Becker, R. (2022). The influence of gluteal muscle strength deficits on dynamic knee valgus: a scoping review. *Journal of Experimental Orthopaedics*, 9(1), 81. <https://doi.org/10.1186/s40634-022-00513-8>
 14. Sterneder, C. M., Faschingbauer, M., Haralambiev, L., Kasparek, M. F., & Boettner, F. (2024). Why kinematic alignment makes little sense in valgus osteoarthritis of the knee: a narrative review. *Journal of Clinical Medicine*, 13(5), 1302. <https://doi.org/10.3390/jcm13051302>
 15. Li, X., Roemer, F. W., Cicuttini, F., MacKay, J. W., Turmezei, T., & Link, T. M. (2023). Early knee OA definition—what do we know at this stage? An imaging perspective. *Therapeutic Advances in Musculoskeletal Disease*, 15, 1759720X231158204. <https://doi.org/10.1177/1759720X231158204>
 16. Liu, S., Deng, Z., Chen, K., Jian, S., Zhou, F., Yang, Y., Fu, Z., Xie, H., Xiong, J., & Zhu, W. (2022). Cartilage tissue engineering: From proinflammatory and anti-inflammatory cytokines to osteoarthritis treatments. *Molecular Medicine Reports*, 25(3), 1-15. <https://doi.org/10.3892/mmr.2022.12615>
 17. Favreau, H., Chennen, K., Feruglio, S., Perennes, E., Anton, N., Vandamme, T., Jessel, N., Poch, O., & Conzatti, G. (2025). Knee osteoarthritis diagnosis: Future and perspectives. *Biomedicine*, 13(7), 1644. <https://doi.org/10.3390/biomedicine13071644>
 18. Dudaric, L., Dumic-Cule, I., Divjak, E., Cengic, T., Brkljacic, B., & Ivanac, G. (2023). Bone remodeling in osteoarthritis—biological and radiological aspects. *Medicina*, 59(9), 1613. <https://doi.org/10.3390/medicina59091613>
 19. Zhang, Y., Han, Y., Sun, Y., Hao, L., Gao, Y., Ye, J., Wang, H., Zhang, T., Liu, Y., & Yang, Y. (2026). Osteoarthritis: molecular pathogenesis and potential therapeutic options. *Signal transduction and targeted therapy*, 11(1), 81. <https://doi.org/10.1038/s41392-025-02556-6>
 20. Geng, R., Li, J., Yu, C., Zhang, C., Chen, F., Chen, J., Ni, H., Wang, J., Kang, K., & Wei, Z. (2023). Knee osteoarthritis: Current status and research progress in treatment. *Experimental and therapeutic medicine*, 26(4), 481. <https://doi.org/10.3892/etm.2023.12180>
 21. Kibler, W. B., Press, J., & Sciascia, A. (2006). The role of core stability in athletic function. *Sports medicine*, 36(3), 189-198. <https://doi.org/10.2165/00007256-200636030-00001>
 22. Vanderlinden, A. O., Nevisipour, M., Sugar, T., & Lee, H. (2024). Reduced trunk movement control during motor dual-tasking in older adults. *Human Movement Science*, 95, 103223. <https://doi.org/10.1016/j.humov.2024.103223>
 23. Lupowitz, L. G. (2023). Comprehensive approach to core training in sports physical therapy: Optimizing performance and minimizing injuries. *International journal of sports physical therapy*, 18(4), 800. <https://doi.org/10.26603/001c.84525>
 24. Waiteman, M. C., Chia, L., Ducatti, M. H., Bazett-Jones, D. M., Pappas, E., de Azevedo, F. M., & Briani, R. V. (2022). Trunk biomechanics in individuals with knee disorders: a systematic review with evidence gap map and meta-analysis. *Sports Medicine-Open*, 8(1), 145. <https://doi.org/10.1186/s40798-022-00536-6>
 25. Molina IV, D., & Blumenthal, S. (2022). Lumbosacral Spine Plain Radiographs. In *Atlas of Spinal Imaging* (pp. 173-181). Elsevier
 26. Gogu, S., & Gandhir, V. N. (2022). Trendelenburg sign. In StatPearls [Internet]: StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK555987/>
 27. Yoon, J.-y., & Moon, S. W. (2024). Impacts of asymmetric hip rotation angle on gait biomechanics in patients with knee osteoarthritis. *Knee Surgery & Related Research*, 36(1), 23. <https://doi.org/10.1186/s43019-024-00226-5>
 28. Wang, J., Hu, Q., Wu, C., Li, S., Deng, Q., Tang, R., Li, K., Nie, Y., & Shen, B. (2023). Gait asymmetry variation in kinematics, kinetics, and muscle force along with the severity levels of knee osteoarthritis. *Orthopaedic Surgery*, 15(5), 1384-1391. <https://doi.org/10.1111/os.13721>
 29. Vadasz, K., Varga, M., Sebesi, B., Hortobágyi, T., Murlasits, Z., Atlasz, T., Fésüs, Á., & Vácz, M. (2022). Frontal plane neuromechanisms stabilizing the knee and the pelvis during unilateral countermovement jump in young trained males. *International journal of environmental research and public health*, 20(1), 220. <https://doi.org/10.3390/ijerph20010220>
 30. Powers, C. M. (2010). The influence of abnormal hip mechanics on knee injury: a biomechanical perspective. *Journal of orthopaedic & sports physical therapy*, 40(2), 42-51. <https://doi.org/10.2519/jospt.2010.3337>
 31. Teng, H.-L., & Powers, C. M. (2016). Hip-extensor strength, trunk posture, and use of the knee-extensor muscles during running. *Journal of athletic training*, 51(7), 519-524. <https://doi.org/10.4085/1062-6050-51.8.05>
 32. Amarasinghe, P., Wadugodapitiya, S., & Weerasekara, I. (2023). Biomechanical and clinical relationships between lower back pain and knee osteoarthritis: A systematic review. *Systematic reviews*, 12(1). <https://doi.org/10.1186/s13643-022-02164-3>
 33. Fu, P., Xu, W., Xu, P., Huang, J., & Guo, J. J. (2023). Relationship between spinal imbalance and knee osteoarthritis by using full-body EOS. *BMC Musculoskeletal Disorders*, 24(1), 402. <https://doi.org/10.1186/s12891-023-06508-5>
 34. Flowers, D. W., Brewer, W., Mitchell, K., & Ellison, J. (2021). Why are we neglecting the core muscles in patients with knee osteoarthritis? A narrative review of the literature. *Physical Therapy Reviews*, 26(4), 276-283. <https://doi.org/10.1080/1080196.2021.1907947>
 35. Kolasinski, S. L., Neogi, T., Hochberg, M. C., Oatis, C., Guyatt, G., Block, J., Callahan, L., Copenhaver, C., Dodge, C., & Felson, D. (2020). 2019 American College of Rheumatology/Arthritis Foundation guideline for the management of osteoarthritis of the hand, hip, and knee. *Arthritis & rheumatology*, 72(2), 220-233. <https://doi.org/10.1002/art.41142>
 36. NG226, N. G. (2022). Osteoarthritis: Care and management National Institute for Health and Care Excellence. <https://www.nice.org.uk/guidance/ng226>
 37. Lim, Y. Z., Wong, J., Hussain, S. M., Estee, M. M., Zolio, L., Page, M. J., Harrison, C. L., Wluka, A. E., Wang, Y., & Cicuttini, F. M. (2022). Recommendations for weight management in osteoarthritis: A systematic review of clinical practice guidelines. *Osteoarthritis and Cartilage Open*, 4(4), 100298. <https://doi.org/10.1016/j.ocarto.2022.100298>
 38. Jacobs, E., Stroobant, L., Victor, J., Elewaut, D., Tampere, T., Wallaert, S., Witvrouw, E., Schuermans, J., & Wezenbeek, E. (2025). Vascular occlusion for optimising the functional improvement in patients with knee osteoarthritis: a randomised controlled trial. *Annals of the rheumatic diseases*, 84(2), 341-350. <https://doi.org/10.1136/ard-2024-226579>
 39. Singh, R., Yadav, P., Agarwal, S., Kaur, S., & Jain, M. (2025). Spine-pelvis-lower extremity alignment in patients with knee osteoarthritis—a gender-based analysis. *Chirurgia Narządów Ruchu i Ortopedia Polska*, 90(2), 89-105. <https://doi.org/10.31139/chnriop.2025.90.2.4>

40. Bakker, N. F., Schrijvers, J. C., Van den Noort, J. C., Hall, M., Van der Krogt, M. M., Harlaar, J., & Van der Esch, M. (2021). A most painful knee does not induce interlimb differences in knee and hip moments during gait in patients with knee osteoarthritis. *Clinical Biomechanics*, 89, 105455 <https://doi.org/10.1016/j.clinbiomech.2021.105455>
41. KKatsumi, R., Mannen, E. M., Bajaj, G., Smith, J. R., Mears, S. C., Stambough, J. B., & Barnes, C. L. (2023). The influence of knee osteoarthritis on spinopelvic alignment and global sagittal balance. *The Journal of Knee Surgery*, 36(09), 917-924. <https://doi.org/10.1055/s-0042-1747947>
42. Yasuda, T., Togawa, D., Hasegawa, T., Yamato, Y., Kobayashi, S., Yoshida, G., Banno, T., Arima, H., Oe, S., & Hoshino, H. (2020). Relationship between knee osteoarthritis and spinopelvic sagittal alignment in volunteers over 50 years of age. *Asian Spine Journal*, 14(4), 495. <https://doi.org/10.31616/asj.2018.0266>
43. Roelker, S. A., Koehn, R. R., Caruthers, E. J., Schmitt, L. C., Chaudhari, A. M., & Siston, R. A. (2021). Effects of age and knee osteoarthritis on the modular control of walking: A pilot study. *Plos one*, 16(12), e0261862. <https://doi.org/10.1371/journal.pone.0261862>
44. Petrella, M., Selistre, L. F. A., Serrão, P. R., Lessi, G. C., Gonçalves, G. H., & Mattiello, S. M. (2021). Kinetics, kinematics, and knee muscle activation during sit to stand transition in unilateral and bilateral knee osteoarthritis. *Gait & posture*, 86, 38-44. <https://doi.org/10.1016/j.gaitpost.2021.02.023>
45. Cabral, A. L., Jorge, J. G., & Dionisio, V. C. (2021). Biomechanical analysis during single-leg squat in individuals with knee osteoarthritis. *The Knee*, 28, 362-370. <https://doi.org/10.1016/j.knee.2020.12.031>
46. Rutherford, D., Baker, M., Urquhart, N., & Stanish, W. (2022). The effect of a frontal plane gait perturbation bout on knee biomechanics and muscle activation in older adults and individuals with knee osteoarthritis. *Clinical Biomechanics*, 92, 105574. <https://doi.org/10.1016/j.clinbiomech.2022.105574>
47. Wang, Y., Zhang, K., Zeng, J., & Yan, S. (2021). Coordination of lower limbs in patients with knee osteoarthritis during walking. *Gait & posture*, 83, 160-166. <https://doi.org/10.1016/j.gaitpost.2020.10.024>
48. Suzuki, Y., Ohkoshi, Y., Kawakami, K., Shimizu, K., Chida, S., Ukishiro, K., Onodera, T., Iwasaki, K., Maeda, T., & Suzuki, S. j. (2023). Assessing knee joint biomechanics and trunk posture according to medial osteoarthritis severity. *Scientific reports*, 13(1), 19186. <https://doi.org/10.1038/s41598-023-46486-1>
49. Chen, X., Fan, Y., Tu, H., & Luo, Y. (2025). Clinical efficacy of different therapeutic options for knee osteoarthritis: A network meta-analysis based on randomized clinical trials. *PLOS One*, 20(6), e0324864. <https://doi.org/10.1371/journal.pone.0324864>
50. Yan, L., Li, D., Xing, D., Fan, Z., Du, G., Jiu, J., Li, X., Estill, J., Wang, Q., & Belal, A. A. (2025). Comparative efficacy and safety of exercise modalities in knee osteoarthritis: systematic review and network meta-analysis. *bmj*, 391. <https://doi.org/10.1136/bmj-2025-085242>
51. Hodges, P. W., & Tucker, K. (2011). Moving differently in pain: a new theory to explain the adaptation to pain. *Pain*, 152(3), S90-S98. <https://doi.org/10.1016/j.pain.2010.10.020>
52. Liu, P., Hamid, M. S. B. A., Shi, Y., Fan, X., Deng, H., Hafiz, E., & Zhang, X. (2025). Associations between muscle stiffness, muscle co-activation, and balance in older individuals with knee osteoarthritis: a cross-sectional study. *European Journal of Medical Research*. <https://doi.org/10.1186/s40001-025-03571-7>
53. Salamanna, F., Caravelli, S., Marchese, L., Carniato, M., Vocale, E., Gardini, G., Puccetti, G., Mosca, M., & Giavaresi, G. (2023). Proprioception and mechanoreceptors in osteoarthritis: a systematic literature review. *Journal of Clinical Medicine*, 12(20), 6623. <https://doi.org/10.3390/jcm12206623>

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CORE MUSCLE STRENGTH AND HIP MUSCLE FUNCTION IN KNEE OSTEOARTHRITIS: A LITERATURE REVIEW

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