

*Review Article*

# Biomechanical Links Between Knee Posture, Strength, and Low Back Pain: A Narrative Review

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## ABSTRACT

**Background:** Low back pain is a major cause of disability worldwide and is increasingly understood as a multifactorial condition influenced by interactions among the lumbar spine, pelvis, hips, knees, and distal lower limbs. Although traditionally managed as a spinal disorder, biomechanical alterations in knee posture, knee range of motion, muscle strength, and neuromuscular control may affect lumbopelvic alignment, spinal loading, gait mechanics, and functional movement. **Objective:** This narrative review aimed to synthesize current biomechanical and clinical evidence on the relationship between knee posture, knee muscle strength, lumbopelvic alignment, and low back pain, with emphasis on kinetic-chain mechanisms and implications for assessment and rehabilitation. **Methods:** Relevant literature was identified through searches of PubMed, Scopus, Web of Science, Google Scholar, and ScienceDirect using terms related to low back pain, knee alignment, knee osteoarthritis, knee extension limitation, quadriceps strength, hamstring strength, lumbopelvic alignment, sagittal balance, kinetic chain, gait, lifting, neuromuscular control, and rehabilitation. Evidence from biomechanical studies, observational studies, systematic reviews, clinical trials, gait analyses, and rehabilitation literature was synthesized narratively using a kinetic-chain framework. **Results:** The strongest evidence supports an association between restricted knee extension, knee flexion contracture, knee osteoarthritis, sagittal imbalance, and increased lumbar loading. Biomechanical studies indicate that knee extension limitation can increase lumbar extension moments, paraspinal activation, and lumbar or thoracolumbar compressive forces during gait. Moderate evidence suggests that quadriceps and hamstring weakness may impair shock absorption and alter force transmission to the pelvis and lumbar spine. Emerging evidence supports neuromuscular and myofascial pathways linking knee dysfunction with altered trunk control, proprioception, and movement compensation. **Conclusion:** Knee posture, mobility, strength, and neuromuscular control are clinically relevant contributors to lumbopelvic biomechanics and low back pain. Integrated assessment and rehabilitation should address knee alignment, knee range of motion, lower-limb strength, gait mechanics, and trunk–pelvis–knee coordination. **Keywords:** Low Back Pain; Knee Joint; Biomechanics; Posture; Muscle Strength; Lumbopelvic Alignment; Kinetic Chain; Rehabilitation.

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## INTRODUCTION

Low back pain is one of the most prevalent and disabling musculoskeletal conditions worldwide, affecting individuals across the lifespan and imposing substantial clinical, functional, and socioeconomic burden. It is commonly defined as pain or discomfort localized between the lower costal margin and the inferior gluteal folds, with or without referred lower-limb symptoms. Although

traditionally approached as a disorder of the lumbar spine, contemporary biomechanical and rehabilitation perspectives increasingly recognize low back pain as a multifactorial condition influenced by interactions among the spine, pelvis, hips, knees, ankles, and feet. This broader view is clinically important because altered loading, impaired movement control, and compensatory postural strategies in one region of the kinetic chain may influence mechanical demands in another region (1,2).

The lumbar spine functions as both a load-bearing and motion-permitting structure. Its stability depends on the coordinated contribution of passive structures, including vertebrae, intervertebral discs, facet joints, ligaments, and active structures, including the paraspinal, abdominal, pelvic, and hip musculature. During standing, walking, lifting, squatting, and athletic tasks, the lumbar region does not operate in isolation; rather, it responds continuously to ground reaction forces transmitted through the lower extremities and pelvis. Consequently, changes in lower-limb alignment or function may alter pelvic position, trunk orientation, lumbar muscle activation, and spinal loading. This mechanical interdependence provides the foundation for considering the knee joint as a potential contributor to lumbopelvic biomechanics and low back symptoms (3,4).

The knee is a central link in the lower-limb kinetic chain because it connects the foot and ankle to the hip, pelvis, and trunk. Its alignment and range of motion influence force transmission during closed-chain activities such as standing, walking, stair negotiation, squatting, and lifting. In the sagittal plane, knee flexion contracture, restricted knee extension, or knee hyperextension may change the position of the body's center of mass and require compensatory adjustments at the pelvis and lumbar spine. In the frontal and transverse planes, genu varum, genu valgum, tibial rotation, and altered foot mechanics may influence hip rotation, pelvic control, and trunk posture. These compensations may be adaptive in the short term, but when persistent, they may increase lumbar muscular demand, modify spinal curvature, and contribute to pain or functional limitation (5,6).

The relationship between the knee and lumbar spine is particularly evident in the concept of knee–spine syndrome, in which knee pathology and spinal dysfunction coexist and may mutually influence symptoms, posture, and disability. Knee osteoarthritis, knee extension limitation, and flexion contracture have been associated with altered sagittal alignment, reduced lumbar lordosis, increased trunk flexion, and changes in global postural balance. Conversely, lumbar dysfunction may affect lower-limb movement strategies, knee loading, and neuromuscular control. These bidirectional interactions are especially relevant in older adults, individuals with degenerative joint disease, and patients requiring complex rehabilitation or surgical decision-making involving both the knee and spine (7,8).

Muscle strength and neuromuscular control provide another important pathway linking knee function to lumbar mechanics. The quadriceps and hamstrings contribute not only to knee stability but also to shock absorption and controlled transfer of forces during gait and functional tasks. Weakness or imbalance in these muscle groups may reduce the capacity of the lower limb to attenuate ground reaction forces, thereby increasing compensatory demand on the hip, pelvis, and lumbar musculature. Evidence also suggests that individuals with low back pain may demonstrate reduced knee extensor strength, altered lower-limb kinematics, impaired postural control, and changes in trunk–lower-limb coordination. These findings support the need to assess knee strength and movement control when evaluating patients with low back pain, rather than focusing exclusively on the lumbar region (9,10).

Despite increasing recognition of the knee–lumbar relationship, the available literature remains heterogeneous. Existing studies vary widely in population, methodology, biomechanical measures, clinical outcomes, and interpretation. Some evidence derives from gait analysis and musculoskeletal modeling, some from observational studies in patients with knee osteoarthritis or low back pain, and some from athletic or rehabilitation contexts. As a result, the strength of evidence differs across proposed mechanisms, and causal pathways remain incompletely established. A narrative synthesis is therefore appropriate to integrate anatomical, biomechanical, clinical, and rehabilitation perspectives while identifying areas where evidence is strong, emerging, or still speculative.

This narrative review aims to synthesize current evidence on the biomechanical links between knee posture, knee muscle strength, lumbopelvic alignment, and low back pain. Specifically, it examines how altered knee alignment, restricted knee motion, knee muscle weakness, and impaired neuromuscular control may influence lumbar loading, pelvic posture, gait mechanics, functional movement, and clinical symptoms. By integrating these mechanisms within a kinetic-chain framework, this review seeks to support more comprehensive assessment and rehabilitation strategies for patients with low back pain and coexisting or contributing knee dysfunction.

## **MATERIALS AND METHODS**

This narrative review was designed to synthesize biomechanical and clinical evidence concerning the relationship between knee posture, knee muscle strength, lumbopelvic alignment, and low back pain. A narrative approach was selected because the available literature spans diverse study designs, populations, biomechanical models, clinical conditions, and rehabilitation contexts, making an integrative conceptual synthesis more appropriate than statistical pooling. The review focused on evidence describing the knee as part of the lower-limb–pelvis–lumbar kinetic chain, with particular attention to knee alignment, knee extension limitation, knee flexion posture, genu varum or valgum, quadriceps and hamstring strength, neuromuscular control, gait mechanics, sagittal balance, spinal loading, and clinical presentations involving concurrent knee and lumbar dysfunction.

Relevant literature was identified through searches of major biomedical and rehabilitation databases, including PubMed, Scopus, Web of Science, Google Scholar, and ScienceDirect. The search strategy combined terms related to low back pain and lumbar biomechanics with terms related to knee posture, knee alignment, knee osteoarthritis, knee extension limitation, knee flexion contracture, quadriceps strength, hamstring strength, lower-limb kinematics, lumbopelvic alignment, sagittal balance, kinetic chain, gait, lifting, neuromuscular control, and rehabilitation. Reference lists of relevant reviews, biomechanical studies, and clinical articles were also examined to identify additional studies addressing anatomical, biomechanical, or clinical links between the knee and lumbar spine.

Studies were considered relevant when they addressed at least one direct or indirect relationship between knee mechanics and lumbar or lumbopelvic function. Priority was given to studies evaluating knee posture or alignment, knee range of motion, lower-limb strength, knee osteoarthritis, patellofemoral pain, anterior cruciate ligament injury, gait biomechanics, lifting mechanics, landing mechanics, spinal loading, sagittal spinopelvic alignment, trunk compensation, or rehabilitation strategies involving both the knee and lumbar region. Evidence from systematic reviews, observational studies, biomechanical modeling studies, gait analyses, clinical trials, and expert biomechanical literature was included when it contributed to understanding the knee–lumbar relationship. Studies focusing exclusively on isolated lumbar pathology without lower-limb biomechanical relevance, or isolated knee pathology without implications for pelvic, trunk, or spinal mechanics, were not prioritized.

The synthesis was organized using a conceptual kinetic-chain framework. Evidence was grouped thematically into anatomical and myofascial connections, biomechanical interactions among the ankle, knee, hip, pelvis, and lumbar spine, effects of knee posture on lumbopelvic alignment, relationships between knee muscle strength and lumbar function, neuromuscular control and movement-pattern adaptations, knee pathology and knee–spine syndrome, and implications for clinical assessment and rehabilitation. Within each theme, findings were interpreted according to the type and strength of evidence, distinguishing established clinical or biomechanical associations from emerging mechanistic explanations and expert interpretation.

Because this was a narrative review, no formal meta-analysis was performed. Quantitative findings from individual studies were summarized descriptively when they provided clinically meaningful estimates of altered loading, muscle activity, range of motion restriction, sagittal alignment, or functional impairment. The synthesis emphasized biomechanical plausibility, consistency of findings across related

evidence, relevance to clinical assessment, and implications for rehabilitation. Potential selection bias was addressed by including evidence from multiple study types and by interpreting conclusions cautiously when findings were based primarily on cross-sectional data, small samples, modeling studies, or indirect mechanistic inference.

## RESULTS

The evidence synthesized in this review supports a clinically meaningful relationship between knee mechanics and lumbar function within an integrated lower-limb–pelvis–spine kinetic chain. Low back pain remains a major global disability burden, with estimated cases increasing from approximately 377–387 million in 1990 to 619 million in 2020, and projections suggesting up to 843 million affected individuals by 2050 (11,12). This growing burden strengthens the rationale for examining modifiable biomechanical contributors beyond the lumbar spine alone, including lower-limb alignment, knee range of motion, knee muscle strength, and neuromuscular control.

The most consistent biomechanical evidence concerns sagittal-plane interaction between knee extension limitation and lumbar loading. Studies evaluating knee flexion contracture, restricted knee extension, and sagittal imbalance indicate that reduced knee extension can shift the body's center of mass and require compensatory changes in pelvic and trunk posture (13,14). In gait analysis, unilateral knee extension restriction increased lumbar extension moment by approximately 2.2 times and raised anterior vertical joint force at L4 and L5 by up to 36.5% and 20%, respectively (15). This finding provides direct biomechanical evidence that even unilateral knee motion restriction may increase lumbar mechanical demand during walking.

Evidence from older adults with knee osteoarthritis further supports the knee–lumbar loading relationship. Restricted knee extension increased compression forces across thoracolumbar and upper lumbar levels, including T12–L1, L1–L2, L2–L3, and L3–L4, during walking (10). These findings suggest that the lumbar effects of knee restriction are not limited to the lower lumbar spine but may involve multilevel spinal loading. Clinically, this is important because older adults with knee osteoarthritis often present with combined knee pain, spinal degeneration, reduced mobility, and sagittal postural compensation.

*Table 1. Global Burden of Low Back Pain and Relevance to Knee–Lumbar Biomechanics*

Year / Evidence Period	Estimated Global Cases	Projected Cases by 2050	Years Lived With Disability	Main Risk Factors Reported	Relevance to Knee–Lumbar Review
1990	377–387 million	Not reported	43 million	Occupational exposure, high body mass index, smoking	Establishes low back pain as a long-standing global disability burden requiring broader biomechanical assessment beyond the lumbar spine alone.
2020	619 million	843 million	69 million	Aging, population growth, occupational exposure, high body mass index	Supports the need to identify modifiable musculoskeletal contributors, including lower-limb alignment, knee range of motion, and muscle weakness.
2021	629 million	Not reported	70 million	Age, sex, occupational loading, body mass index, regional and demographic factors	Reinforces the importance of integrated preventive and rehabilitative approaches for spine, pelvis, and lower-limb function.

Knee posture also appears to influence lumbar mechanics during functional tasks such as lifting, squatting, landing, and sport-specific impact activities. During lifting with restricted knee range of motion, pelvic rotation and lower-limb muscle activation changed even when trunk flexion, extension, and side bending were not substantially altered (16). This indicates that compensatory strategies may occur through the pelvis and lower limb before obvious trunk motion changes are detected. Hamstring flexibility also appears relevant, as limited hamstring flexibility may restrict anterior pelvic rotation and

increase trunk flexion during stoop lifting (17). In athletes, reduced knee flexion at front-foot contact in fast bowlers was associated with increased lumbar and lower-limb loading, suggesting that insufficient knee flexion may reduce shock absorption during high-impact movement (13).

The evidence linking knee muscle strength to low back pain is also clinically relevant. A systematic review and meta-analysis found that patients with low back pain demonstrated reduced lower-limb muscle strength, including knee flexor and extensor deficits, with knee extensor torque deficits reported at approximately 0.31 Nm/kg at 60–120°/s (18). Other clinical studies support an association between quadriceps weakness, pain severity, and reduced mobility in low back pain populations (19). These findings suggest that knee extensor strength may contribute to lumbar function by supporting shock absorption, sagittal alignment, and controlled force transfer during gait and functional tasks.

*Table 2. Anatomical and Biomechanical Pathways Linking the Knee Joint to Lumbar Function*

Pathway	Main Structures Involved	Proposed Mechanism	Lumbar / Lumbopelvic Consequence	Strength of Evidence
<b>Closed kinetic chain force transmission</b>	Foot, ankle, tibia, knee, femur, hip, pelvis, lumbar spine	Ground reaction forces pass proximally through linked joints during standing, walking, squatting, lifting, and landing	Altered knee mechanics may change pelvic orientation, trunk posture, lumbar muscle demand, and spinal loading	Moderate biomechanical support
<b>Sagittal alignment compensation</b>	Knee joint, hip joint, pelvis, lumbar spine	Knee flexion posture or restricted extension shifts center of mass and requires compensatory pelvic and trunk adjustments	Reduced lumbar lordosis, increased trunk flexion, altered sagittal balance, increased lumbar moments	Moderate clinical and biomechanical support
<b>Frontal-plane malalignment</b>	Genu varum, genu valgum, hip abductors, pelvis, trunk	Varus or valgus alignment alters lower-limb loading and may induce pelvic or trunk compensation	Asymmetric lumbar loading, altered trunk lean, potential aggravation of low back symptoms	Emerging to moderate support
<b>Myofascial continuity</b>	Thoracolumbar fascia, iliotibial band, hamstrings, gluteal complex, paraspinal muscles	Tension may be transmitted across fascial chains linking lower limb, pelvis, and lumbar region	Altered stiffness, altered lumbopelvic control, symptom propagation across regions	Emerging mechanistic support
<b>Neuromuscular control</b>	Quadriceps, hamstrings, gluteus medius, multifidus, trunk stabilizers	Impaired proprioception or delayed muscle activation may disrupt coordinated load transfer	Poor postural control, compensatory trunk activation, altered movement patterns	Moderate support for association; causal pathways remain less certain
<b>Shock absorption failure</b>	Quadriceps, hamstrings, ankle-knee-hip complex	Weak or poorly coordinated knee musculature reduces attenuation of ground reaction forces	Greater proximal force transmission to pelvis and lumbar spine	Moderate support, especially in low back pain and athletic populations

Neuromuscular control represents an additional mechanism linking knee dysfunction and lumbar symptoms. Individuals with chronic low back pain frequently demonstrate impaired balance and altered postural control, suggesting disruption of sensorimotor integration beyond isolated lumbar muscle weakness (20). Knee pathology may contribute to this process through altered proprioceptive input, quadriceps inhibition, impaired lower-limb coordination, and compensatory activation of proximal musculature (14,15). The evidence is strongest for association and mechanistic plausibility, while direct causal pathways between knee sensorimotor dysfunction and lumbar pain remain less established.

Frontal-plane knee alignment and trunk compensation provide another clinically important pathway. Genu varum, genu valgum, varus thrust, and altered hip-knee coordination may influence pelvic control and trunk lean during walking, squatting, sit-to-stand movement, and landing tasks (21). A systematic review of trunk biomechanics across knee disorders reported that individuals with knee osteoarthritis showed greater trunk flexion during squatting and trunk lean during ambulation and sit-

to-stand tasks, while trunk flexion was also associated with patellofemoral pain and anterior cruciate ligament reconstruction (11). These findings suggest that trunk adaptation may function as a compensation for knee pain, weakness, instability, or altered joint loading.

**Table 3. Evidence Summary: Knee Posture, Range of Motion, and Lumbar Loading**

Evidence Source	Design / Context	Knee Variable	Spinopelvic Variable	Key Finding
<b>Harato et al.</b>	Gait analysis using simulated knee flexion contracture	Knee flexion contracture	Knee–spine compensatory mechanics	Simulated knee flexion contracture altered gait and helped explain knee–spine syndrome mechanisms
<b>Shimizu et al.</b>	Longitudinal spinal deformity-related evaluation	Limited knee extension	Lumbar lordosis and lower-extremity deviation	Lumbar lordosis below 30° was associated with limited knee extension range
<b>Teraguchi et al.</b>	Spine–pelvis–lower extremity alignment study	Knee extension limitation greater than 5°	Back-related disability and sagittal imbalance	Knee extension limitation was associated with sagittal imbalance and back-related disability
<b>Nakatsuji et al.</b>	Biomechanical gait study	Unilateral knee extension restriction	Lumbar moment, paraspinal activity, L4–L5 loading	Knee extension restriction increased lumbar extension moment 2.2-fold and increased anterior vertical joint force at L4 and L5 by up to 36.5% and 20%, respectively
<b>Kotaki et al.</b>	3D musculoskeletal analysis in older adults with knee osteoarthritis	Restricted knee extension	Thoracolumbar and upper lumbar disc compression force	Restricted knee extension increased compression force at T12–L1, L1–L2, L2–L3, and L3–L4 during walking
<b>Storari et al.</b>	Lifting biomechanics with knee range-of-motion restriction	Restricted knee ROM	Trunk, pelvis, and muscle activity during lifting	Knee ROM restriction did not substantially change trunk flexion, extension, or side bending, but significantly affected pelvic rotation and increased gastrocnemius lateralis and biceps femoris activity
<b>Patterson et al.</b>	Stoop-lift biomechanics	Hamstring flexibility and lumbar extensor strength	Lumbar and pelvic kinematics	Limited hamstring flexibility was associated with increased trunk flexion and restricted anterior pelvic rotation during lifting
<b>Arun et al.</b>	Systematic review in fast bowlers	Knee flexion below 30° at front-foot contact	Lumbar and lower-limb loading	Reduced knee flexion at front-foot contact increased lumbar and lower-limb loading and was associated with higher injury risk

The clinical concept of knee–spine syndrome is supported by evidence showing that knee osteoarthritis, knee flexion contracture, lumbar degenerative disease, and sagittal malalignment often coexist, particularly in older adults (22). In such patients, symptoms may not arise from a single anatomical region; rather, pain and disability may reflect combined impairment across the knee, pelvis, and lumbar spine. This has practical implications for diagnosis, rehabilitation, and surgical planning. For example, correction of knee alignment or flexion contracture after total knee arthroplasty may influence standing posture and spinopelvic alignment, while persistent lumbar deformity may affect postoperative function and symptom relief (22,23).

**Table 4. Evidence Summary: Knee Muscle Strength, Neuromuscular Control, and Lumbar Function**

Evidence Source	Population / Context	Knee or Lower-Limb Factor	Functional Outcome	Key Finding	Evidence Strength
<b>De Sousa et al.</b>	Systematic review and meta-analysis of patients with low back pain	Knee flexor and extensor strength	Low back pain status	Patients with low back pain showed reduced lower-limb muscle strength, including knee extensor torque deficits of approximately 0.31 Nm/kg at 60–120°/s	Stronger evidence for association
<b>Kato et al.</b>	Older women	Knee extensor weakness	Low back pain, mobility limitation, malnutrition	Low back pain was associated with muscle weakness and reduced mobility function	Moderate clinical evidence
<b>Kocaman et al.</b>	Chronic nonspecific low back pain	Quadriceps isometric strength	Pain severity measured by visual analogue scale	Quadriceps strength showed an inverse relationship with pain severity	Moderate clinical evidence
<b>Cai and Kong</b>	Recreational runners with chronic low back pain	Knee extensor performance	Shock absorption during running	Reduced knee extensor performance may impair eccentric control at ground contact and transmit greater forces proximally	Moderate biomechanical-clinical evidence
<b>Edwards et al.</b>	Golf biomechanics review	Quadriceps inhibition linked with erector spinae fatigue	Spinal endurance and golf-related low back pain	Erector spinae fatigue was associated with quadriceps inhibition, supporting functional trunk–lower-limb coupling	Emerging sport-specific evidence
<b>Berenshteyn et al.</b>	Systematic review of chronic low back pain	Balance and postural control deficits	Standing balance	Individuals with chronic low back pain frequently	Moderate evidence

Evidence Source	Population / Context	Knee or Lower-Limb Factor	Functional Outcome	Key Finding	Evidence Strength
Almansoof et al.	Narrative review of kinetic-chain mechanisms	Neuromuscular coordination and kinetic-chain dysfunction	Injury risk and performance	demonstrated altered standing balance Impaired neuromuscular control in one segment may affect coordinated movement across the kinetic chain	Conceptual and emerging evidence
Criss et al.	Multidisciplinary review after ACL injury	Sensorimotor and CNS adaptations	Proximal motor planning and movement control	ACL injury and reconstruction may be associated with central nervous system adaptations affecting movement control beyond the knee	Emerging mechanistic evidence
Sherman et al.	Review of knee pathology and efferent pathway dysfunction	Arthrogenic muscle inhibition	Muscle force and motor pathway function	Knee pathology may inhibit quadriceps activation through pathways extending from motor cortex to muscle force production	Emerging mechanistic evidence

Table 5. Knee Pathology, Knee–Spine Syndrome, and Clinical Implications

Scenario	Knee-Related Mechanism	Trunk Adaptation	Clinical Implication	Evidence Basis
Knee osteoarthritis	Pain, flexion contracture, altered femoral tilt, restricted extension	Loss of lumbar lordosis, trunk flexion, altered sagittal balance	Assess lumbar alignment and function in patients with symptomatic knee osteoarthritis	Moderate evidence from clinical and radiographic studies
Patellofemoral pain	Altered knee loading and hip–knee mechanics	Possible spinal imbalance and trunk movement adaptation	Include lumbopelvic control and trunk mechanics in evaluation	Emerging to moderate evidence
ACL injury or reconstruction	Sensorimotor disruption, altered proprioception, quadriceps inhibition	Modified trunk control, altered landing or squatting mechanics	Rehabilitation should integrate knee control with trunk and hip stabilization	Moderate mechanistic and clinical evidence
Knee–spine syndrome in older adults	Coexisting knee osteoarthritis and lumbar degenerative disease	Combined sagittal malalignment, pain overlap, functional impairment	Determine dominant pain generator and assess both regions before planning treatment	Moderate clinical evidence
Total knee arthroplasty in patients with spinal disease	Correction of knee flexion or alignment may alter global posture	Potential changes in spinopelvic alignment and low back symptoms	Surgical sequencing should consider knee, hip, pelvis, and spine alignment together	Emerging to moderate surgical evidence
Lifting, squatting, and landing tasks	Knee ROM limitation, poor shock absorption, altered knee valgus control	Increased trunk compensation, altered lumbar loading, increased paraspinal demand	Rehabilitation should combine lower-limb strength, mobility, and trunk control	Moderate biomechanical evidence

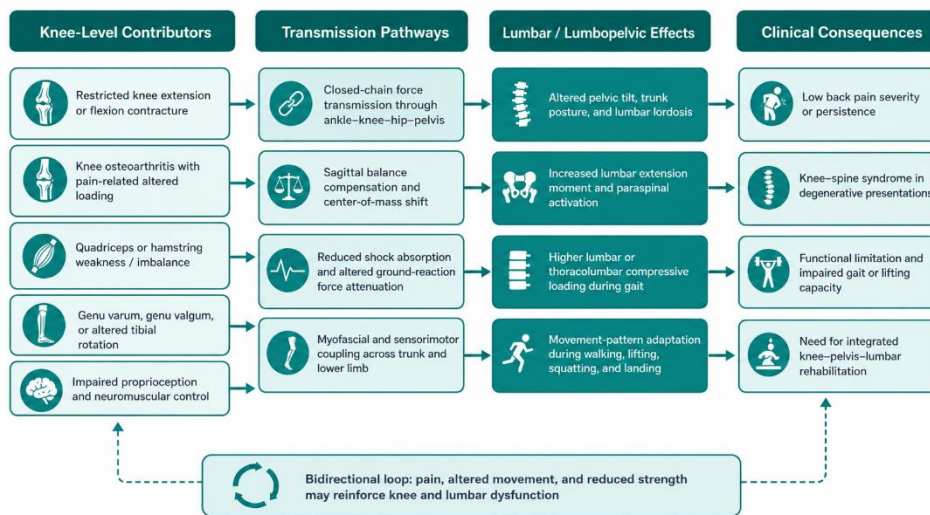


Figure 1. Conceptual Synthesis of Knee–Lumbar Biomechanical Interactions

The figure presents an integrated kinetic-chain model showing how knee-level contributors may influence lumbar and lumbopelvic mechanics through four principal pathways: closed-chain force transmission, sagittal balance compensation, reduced shock absorption, and myofascial or sensorimotor coupling. The strongest evidence supports links among restricted knee extension, sagittal compensation, and increased lumbar loading, while moderate evidence supports the contribution of quadriceps or hamstring weakness to impaired shock absorption and low back pain. Myofascial and neuromuscular pathways remain clinically plausible but comparatively emerging, particularly in relation to

proprioception, altered movement control, and bidirectional reinforcement between knee dysfunction and persistent low back symptoms.

Overall, the synthesis indicates that the knee and lumbar spine interact through several overlapping mechanisms: sagittal alignment compensation, closed-chain force transmission, altered shock absorption, myofascial continuity, neuromuscular control, and clinical coexistence of degenerative knee and spinal disorders. The strongest evidence supports associations between knee extension limitation, knee osteoarthritis, altered sagittal balance, and increased lumbar loading. Moderate evidence supports relationships between knee muscle weakness, especially quadriceps weakness, and low back pain or impaired function. Emerging evidence supports neuromuscular and myofascial mechanisms, although these pathways require cautious interpretation because many findings are indirect or based on mechanistic inference. Clinically, these results support a broader assessment model for patients with low back pain. Evaluation should include knee range of motion, sagittal and frontal knee alignment, quadriceps and hamstring strength, hamstring flexibility, gait mechanics, trunk posture, and functional movement tasks such as squatting, lifting, stair negotiation, and landing when relevant. Rehabilitation strategies should address both local lumbar impairments and lower-limb contributors, particularly knee extension limitation, quadriceps weakness, poor shock absorption, and impaired trunk–pelvis–knee coordination.

## DISCUSSION

This narrative review synthesized biomechanical and clinical evidence on the relationship between knee posture, knee muscle strength, lumbopelvic alignment, and low back pain. The principal finding is that the knee and lumbar spine should not be interpreted as mechanically independent regions, particularly during closed-chain activities such as standing, walking, lifting, squatting, stair negotiation, and landing. Instead, the available evidence supports an integrated lower-limb–pelvis–lumbar model in which restricted knee motion, altered knee alignment, knee osteoarthritis, muscle weakness, and impaired neuromuscular control may influence pelvic orientation, trunk posture, lumbar muscle activation, and spinal loading. The strongest evidence appears to concern sagittal-plane mechanisms, particularly knee extension limitation and flexion contracture, whereas evidence for myofascial and sensorimotor pathways remains clinically plausible but less directly established.

A consistent theme across the reviewed evidence is that knee extension limitation can disturb sagittal balance and increase lumbar mechanical demand. Restricted knee extension may shift the body's center of mass and require compensatory trunk or pelvic adjustments to maintain upright posture and forward progression during gait. Biomechanical findings showing increased lumbar extension moment and elevated anterior vertical joint forces at L4 and L5 during unilateral knee extension restriction provide a particularly important mechanistic explanation for how a distal joint restriction can increase lumbar loading (16). Similarly, evidence from older adults with knee osteoarthritis indicates that limited knee extension may increase thoracolumbar and upper lumbar compression forces during walking (20). These findings support the view that knee extension deficit is not merely a local knee impairment but a potential contributor to multisegment postural compensation and lumbar overload.

The relationship between knee posture and lumbopelvic alignment is most clinically apparent in patients with knee osteoarthritis, flexion contracture, sagittal imbalance, and degenerative spinal disease. In these populations, knee pain and restricted motion may encourage compensatory trunk flexion, altered pelvic position, reduced lumbar lordosis, or abnormal global sagittal alignment. This provides a biomechanical basis for the clinical concept of knee–spine syndrome, in which knee and lumbar disorders coexist and may amplify one another (19,22). However, the relationship should be interpreted as bidirectional rather than strictly causal. Knee pathology may contribute to altered spinal loading, but lumbar dysfunction may also influence lower-limb movement strategies, pain distribution, gait, balance,

and knee loading. This bidirectionality is important for clinical reasoning because treating only the most symptomatic region may fail to address the mechanical contributors sustaining pain or disability.

Knee muscle strength also appears to play an important role in lumbar function. The quadriceps and hamstrings contribute to knee stability, shock absorption, and controlled transfer of ground reaction forces through the lower-limb kinetic chain. Reduced knee extensor or flexor strength in individuals with low back pain suggests that lower-limb weakness may impair force attenuation and increase compensatory demand on the hip, pelvis, and lumbar musculature (23). The association between quadriceps weakness and pain severity further supports the clinical relevance of knee muscle assessment in low back pain populations (24). This relationship is especially important during gait, running, lifting, and landing, where inadequate eccentric control or poor shock absorption at the knee may increase proximal loading. Nevertheless, most available evidence demonstrates association rather than definitive causation, and it remains unclear whether knee weakness is a cause, consequence, or perpetuating factor in low back pain.

Neuromuscular control provides an additional explanatory pathway linking knee dysfunction and lumbar symptoms. Chronic low back pain is frequently associated with impaired postural control, altered proprioception, delayed trunk muscle activation, and maladaptive movement strategies (25). Knee pathology may contribute to similar sensorimotor disturbances through altered afferent feedback, quadriceps inhibition, and compensatory activation of proximal muscles (25,26). These mechanisms are particularly relevant after anterior cruciate ligament injury, in patellofemoral pain, and in movement tasks involving dynamic valgus, trunk lean, or poor landing control. Established evidence supports the presence of altered motor control in both knee and lumbar conditions; however, the precise neural pathways linking knee proprioception to lumbar motor control remain incompletely defined. Therefore, neuromuscular explanations should be presented as emerging mechanistic interpretations rather than settled causal pathways.

The reviewed evidence also suggests that trunk compensation is common across several knee disorders. Individuals with knee osteoarthritis, patellofemoral pain, and anterior cruciate ligament reconstruction may demonstrate increased trunk flexion, ipsilateral or contralateral trunk lean, altered hip-knee coordination, and modified movement strategies during gait, squatting, sit-to-stand tasks, and landing (27). These adaptations may serve short-term protective functions by reducing knee joint load or avoiding pain, but they may also increase mechanical demand on the lumbar spine when repeated over time. This interpretation is particularly relevant for rehabilitation because correcting knee mechanics without addressing trunk and pelvic control may leave important compensatory patterns unresolved.

The findings have several implications for clinical assessment. Patients presenting with low back pain should be evaluated not only for lumbar mobility, paraspinal function, and pain provocation but also for knee extension range, flexion contracture, frontal-plane alignment, quadriceps and hamstring strength, hamstring flexibility, gait mechanics, balance, and functional movement quality. Similarly, patients with knee osteoarthritis, patellofemoral pain, anterior cruciate ligament injury, or persistent knee stiffness should be assessed for lumbar posture, pelvic control, trunk compensation, and back-related disability. This integrated assessment model is especially relevant in older adults, athletes, and patients with persistent symptoms despite localized treatment.

The rehabilitation implications are equally important. For patients with low back pain and coexisting knee impairments, treatment may need to include knee extension restoration, quadriceps and hamstring strengthening, hip and trunk stabilization, gait retraining, balance training, and movement-pattern correction. For patients with knee pathology, rehabilitation should consider whether trunk stiffness, poor lumbopelvic control, reduced spinal endurance, or altered landing and lifting mechanics are contributing to symptoms or functional limitation. Interventions should be individualized because restricting knee movement during activities such as squatting or lifting may reduce local knee stress in some contexts but increase compensatory loading at the hip or lumbar spine in others (21,22). Therefore,

rehabilitation should emphasize coordinated movement across the knee, hip, pelvis, and trunk rather than isolated strengthening alone.

These findings also have implications for surgical decision-making in patients with combined knee and spinal pathology. In knee–spine syndrome, determining whether knee disease or lumbar disease is the dominant pain generator can be difficult because symptoms, posture, and function may overlap. Evidence suggesting that total knee arthroplasty can influence spinopelvic alignment and posture indicates that correction of knee deformity or flexion contracture may alter lumbar mechanics in selected patients (24–26). Conversely, persistent sagittal spinal malalignment may limit functional recovery after knee surgery. Surgical planning should therefore consider global alignment, flexion contracture severity, neurological symptoms, pain distribution, and functional priorities rather than treating the knee and spine as unrelated problems.

Several limitations of this review should be acknowledged. Because the synthesis is narrative, it does not provide pooled effect estimates, formal risk-of-bias assessment, or graded certainty of evidence. The evidence base is heterogeneous, including biomechanical modeling studies, gait analyses, cross-sectional clinical studies, systematic reviews, surgical outcome studies, and sport-specific investigations. Populations also vary widely, including older adults with knee osteoarthritis, individuals with chronic nonspecific low back pain, athletes, post-surgical patients, and healthy volunteers. These differences limit direct comparison across studies and make it difficult to define a single causal pathway. In addition, several proposed mechanisms, particularly myofascial force transmission and long-range sensorimotor coupling, remain partly inferential and require more direct experimental confirmation.

Future research should prioritize prospective longitudinal studies to determine whether knee extension limitation, knee malalignment, or knee muscle weakness precede the development or persistence of low back pain. Standardized biomechanical protocols are needed to measure knee range of motion, sagittal alignment, pelvic posture, trunk motion, lumbar loading, and muscle activation across comparable populations. Randomized controlled trials should examine whether interventions targeting knee extension, quadriceps strength, hamstring flexibility, or knee–trunk movement coordination improve low back pain outcomes. Future studies should also investigate whether specific subgroups, such as older adults with knee osteoarthritis, athletes exposed to repetitive impact loading, or patients with combined degenerative knee and lumbar disease, respond differently to integrated rehabilitation strategies. Finally, patient-reported outcomes should be combined with biomechanical measures to clarify whether improvements in alignment or loading translate into meaningful reductions in pain, disability, and functional limitation.

## CONCLUSION

The available evidence indicates that knee posture, knee range of motion, muscle strength, and neuromuscular control are clinically relevant contributors to lumbopelvic biomechanics and low back pain through their effects on sagittal alignment, closed-chain force transmission, shock absorption, trunk compensation, and spinal loading. Restricted knee extension, knee flexion contracture, and knee osteoarthritis appear most consistently associated with altered pelvic posture, reduced or compensatory lumbar curvature, increased lumbar loading, and functional limitation, while quadriceps and hamstring weakness may further impair force attenuation and movement control. These findings support an integrated clinical approach in which assessment and rehabilitation of low back pain include knee alignment, knee mobility, lower-limb strength, gait mechanics, and functional movement patterns, and in which patients with knee pathology are also evaluated for lumbopelvic compensation. Future research should prioritize longitudinal and interventional studies to clarify causal pathways and determine whether targeted correction of knee impairments can reduce low back pain and improve function.

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