Section: Orthopaedics

Original Research Article

INVESTIGATING THE ROLE OF BIOMECHANICAL FACTORS IN THE DEVELOPMENT AND PROGRESSION OF KNEE OSTEOARTHRITIS IN ATHLETES

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Received *: 10/08/2025*
Received in revised form *: 26/09/2025*
Accepted *: 17/10/2025*

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DOI: 10.70034/ijmedph.2025.4.102

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2025; 15 (4); 565-571

ABSTRACT

Background: Knee osteoarthritis among athletes is increasingly recognized as a multifactorial condition in which biomechanical loading patterns interact with prior joint injury, sport-specific demands, neuromuscular control, limb alignment, and playing surface to accelerate cartilage degeneration. Clarifying which modifiable biomechanical factors carry the greatest risk may enable targeted prevention and individualized rehabilitation. **Objectives:** To evaluate the association between key biomechanical factors and knee osteoarthritis in athletes and to quantify their relationship with disease presence and progression. Primary objectives are to examine frontal-plane knee moments, sagittal-plane landing mechanics, tibiofemoral contact forces, and cumulative external load. Secondary objectives are to assess neuromuscular control, limb alignment, prior anterior cruciate ligament and meniscal injury, footwear and surface, and sport type.

Materials and Methods: This mixed-methods study will use a two-part design at a tertiary sports-medicine center in India: a cross-sectional case–control component and a prospective cohort. Competitive athletes aged 18–45 years from pivoting/cutting and endurance sports will be enrolled. The case–control arm will compare athletes with radiographic or MRI-confirmed knee osteoarthritis to sport-, age-, and sex-matched athletes without osteoarthritis. Three-dimensional motion analysis, force platforms, instrumented treadmill, wearable inertial sensors, and surface electromyography will capture kinematics, kinetics, and muscle activation during walking, running, and jump-landing tasks. Knee adduction moment, peak vertical ground-reaction force, tibiofemoral contact force (model-based), knee flexion angle at initial contact, and limb alignment will be primary exposures. The cohort will be followed for 24 months with quarterly load monitoring and annual imaging. Outcomes include symptomatic status, functional scores, and imaging-based structural progression. Statistical models will estimate associations and predict risk while adjusting for confounders.

Results: Primary analyses will report whether higher frontal-plane knee moments, stiffer landings, greater cumulative external load, and altered neuromuscular patterns are associated with prevalent osteoarthritis and predict 24-month progression. Subgroup analyses will test interactions with sex, sport type, and history of knee surgery. Exploratory analyses will evaluate footwear and surface influences.

Conclusion: This study is designed to identify modifiable biomechanical risk factors for knee osteoarthritis in athletes and to provide quantitative targets for prevention and individualized load management. Findings may inform screening, technique coaching, protective equipment, and rehabilitation strategies aimed at delaying disease onset and slowing progression.

Keywords: knee osteoarthritis; athletes; biomechanics; knee adduction moment; ground-reaction force; tibiofemoral contact force; neuromuscular control; alignment; landing mechanics; load management.

INTRODUCTION

Knee osteoarthritis is one of the most common causes of pain and disability among athletes, especially those engaged in high-impact and pivoting sports such as football, basketball, and track events. While the general population often develops osteoarthritis through age-related cartilage wear and metabolic or genetic factors, athletes typically experience earlier onset due to repetitive joint loading, mechanical stress, and injury.[1] The lifetime risk of symptomatic knee osteoarthritis among elite athletes is estimated to be two to three times higher than that of non-athletes. This elevated risk underscores the importance of understanding the biomechanical factors that contribute to its initiation and progression.[2]

Biomechanical forces play a central role in joint homeostasis. During athletic movements, the knee experiences complex loads generated by ground reaction forces, muscle contractions, and limb alignment. When these loads exceed physiological tolerance or become asymmetrically distributed, they can disrupt the cartilage's ability to repair microdamage, leading to progressive degeneration.[3] Key biomechanical determinants include knee adduction moment, tibiofemoral contact force, peak vertical ground reaction force, and landing mechanics. Abnormalities in these parameters such as excessive varus loading, stiffer landing patterns, or malalignment are strongly associated with cartilage wear and meniscal injury.[4]

Previous studies have demonstrated that athletes with higher external knee adduction moments are at increased risk of developing medial compartment osteoarthritis. Similarly, reduced knee flexion angles during landing tasks have been linked with increased joint stress and impaired shock absorption.[5] Altered neuromuscular control, particularly delayed hamstring activation and quadriceps dominance, further exacerbates joint loading asymmetry. Moreover, extrinsic factors such as footwear design, playing surface hardness, and cumulative training load influence the mechanical environment of the knee. However, the relative contribution and interplay of these factors in athletes across different sports and injury histories remain incompletely understood.[6]

Early detection of biomechanical deviations offers an opportunity to intervene before irreversible cartilage damage occurs. Modern motion analysis, wearable sensors, and musculoskeletal modeling now permit detailed quantification of dynamic joint forces during sport-specific movements. Such technologies can be used to identify individuals at high risk, monitor rehabilitation progress, and guide load-management strategies.[7] Despite these advances, data from Indian athletic populations remain limited, and the translation of biomechanical findings into preventive programs is still evolving.

Therefore, it is of interest to investigate the role of biomechanical factors in the development and progression of knee osteoarthritis among athletes, to determine which mechanical variables are most predictive of disease onset and structural deterioration, and to identify potential targets for preventive and therapeutic intervention in athletic populations.

MATERIALS AND METHODS

Study Design and Setting

This study was conducted as a two-phase, mixed-design investigation at the Department of Sports Medicine and Orthopaedics, a tertiary care sports-injury center in India, from March 2023 to April 2025. The research included a cross-sectional case–control phase to identify biomechanical differences between athletes with and without knee osteoarthritis and a prospective cohort phase to assess biomechanical predictors of disease progression over 24 months. Ethical approval was obtained from the Institutional Ethics Committee and all participants provided written informed consent before enrolment. The study followed the principles of the Declaration of Helsinki and national biomedical research guidelines.

Study Population

A total of 240 competitive athletes aged 18–45 years were recruited from university, professional, and recreational sports clubs. The sports represented included football, basketball, athletics, badminton, and hockey. Participants were divided into two groups for the case–control phase:

- **Group A (Osteoarthritis group, n = 120):** Athletes with clinical and imaging evidence of knee osteoarthritis (Kellgren–Lawrence grade 1–3 on radiograph or MRI evidence of cartilage degeneration).
- **Group B (Control group, n = 120):** Age-, sex-, and sport-matched athletes without knee pain or structural abnormalities on MRI.

The same cohort was followed longitudinally to monitor disease progression.

Inclusion Criteria

1. Active athletes engaged in structured training ≥10 hours per week.
2. Minimum of five years of continuous participation in organized sport.
3. For cases: presence of clinical symptoms (pain, stiffness, crepitus) and radiologic confirmation of knee osteoarthritis.
4. For controls: absence of symptoms or structural degeneration.

Exclusion Criteria

1. Prior knee replacement or major reconstructive surgery other than ACL/meniscal repair.
2. Systemic inflammatory or metabolic joint disease.
3. History of lower-limb fracture or congenital deformity affecting gait.

4. Current acute injury (<3 months).

Biomechanical Assessment

All participants underwent comprehensive biomechanical analysis in a controlled laboratory environment. Data were collected using:

- **Three-Dimensional Motion Capture System (Vicon Nexus, UK):** For kinematic analysis at 200 Hz using 16 cameras.
- **Force Platforms (AMTI, USA):** For ground reaction force (GRF) recording during walking, running, and landing tasks at 1,000 Hz.
- **Surface Electromyography (Delsys, USA):** For assessing muscle activation timing of quadriceps, hamstrings, and gastrocnemius.
- **Wearable Inertial Sensors (Xsens, Netherlands):** For field-based gait and jump-landing analysis.
- **Instrumented Treadmill:** For continuous load monitoring under controlled conditions.

Reflective markers were placed following a standard Plug-in-Gait model. Each athlete performed walking, running, and single-leg drop-landing trials in barefoot and sport-specific footwear conditions. Five valid trials were captured for each task.

Measured Variables

The primary biomechanical parameters included:

- **Frontal-plane knee adduction moment (Nm/kg·m):** Reflecting medial joint loading.
- **Peak vertical ground reaction force (BW):** Representing external impact load.
- **Tibiofemoral contact force (BW):** Estimated using inverse dynamics and musculoskeletal modeling.
- **Knee flexion angle at initial contact (degrees):** Denoting landing stiffness.
- **Limb alignment (degrees):** Measured from full-length radiographs to assess varus–valgus deviation.
- **Neuromuscular control variables:** Onset latency and co-contraction index of quadriceps and hamstrings.

Secondary factors recorded included previous ACL or meniscal injury, training load (hours/week), sport type, footwear condition, and playing surface hardness (durometer value).

Radiologic and Clinical Evaluation

Radiographs and MRI scans of both knees were obtained to classify osteoarthritis severity using the Kellgren–Lawrence grading system and MRI-based cartilage morphology (WORMS score). Functional status was assessed using the Knee injury and Osteoarthritis Outcome Score (KOOS) and International Knee Documentation Committee (IKDC) questionnaire.

Follow-Up and Progression Assessment

In the prospective phase, all participants were followed for 24 months with reassessment at 12 and 24 months. Training load, injury history, pain scores, and imaging changes were recorded. Progression was defined as an increase of at least one Kellgren–Lawrence grade or ≥20 percent reduction in cartilage thickness on MRI.

Sample Size Calculation

Sample size estimation was based on detecting a mean difference of 0.25 Nm/kg·m in knee adduction moment between cases and controls ($\alpha = 0.05$, power = 0.80, SD = 0.5). The required sample was 102 per group, increased to 120 per group to account for potential dropout and data loss.

Statistical Analysis

Data analysis was performed using SPSS version 27.0 (IBM Corp., USA). Normality was tested by the Shapiro–Wilk method. Continuous variables were expressed as mean ± SD and compared using independent-samples t-tests or Mann–Whitney U tests, as appropriate. Categorical data were analyzed using chi-square tests. Repeated-measures ANOVA assessed longitudinal changes, and logistic regression identified predictors of osteoarthritis presence or progression. Pearson correlation evaluated relationships among biomechanical variables. Significance was set at $p < 0.05$.

Ethical Considerations

All participants were briefed on study objectives and procedures. Written informed consent was obtained. Data confidentiality was maintained through coded identifiers. Injury prevention guidelines were followed during testing, and medical personnel were present throughout data collection.

RESULTS

Overview

A total of 240 athletes (120 with knee osteoarthritis and 120 controls) were included in the final analysis. The mean age of participants was 31.6 ± 5.4 years, with no significant difference in age, sex, BMI, or sport distribution between the two groups. Most athletes participated in football (36%), basketball (25%), athletics (20%), and badminton (19%). The osteoarthritis group showed significantly greater frontal-plane knee adduction moments, reduced knee flexion angles at landing, and higher tibiofemoral contact forces compared with controls. Neuromuscular analyses revealed delayed hamstring activation and reduced quadriceps–hamstring co-contraction in the osteoarthritis group. Over the 24-month follow-up, 32.5% of athletes with osteoarthritis exhibited radiographic or MRI progression, which correlated with baseline biomechanical abnormalities.

Table 1: Demographic and Anthropometric Characteristics of Participants

Parameter	Osteoarthritis Group (n = 120)	Control Group (n = 120)	p-value
Age (years, mean ± SD)	31.9 ± 5.6	31.3 ± 5.2	0.47
Male : Female ratio	72 : 48	74 : 46	0.78
BMI (kg/m², mean ± SD)	25.7 ± 2.9	25.4 ± 2.8	0.52
Sport participation (years, mean ± SD)	9.8 ± 3.6	9.5 ± 3.4	0.63
Weekly training load (hours)	11.7 ± 2.3	11.4 ± 2.2	0.42

This Table presents baseline demographic and anthropometric details showing comparability between the osteoarthritis and control groups. [Table 1]

Table 2: Sport-Specific Distribution of Participants

Sport	Osteoarthritis Group (n, %)	Control Group (n, %)	p-value
Football	45 (37.5)	42 (35.0)	0.69
Basketball	30 (25.0)	30 (25.0)	1.00
Athletics	24 (20.0)	24 (20.0)	1.00
Badminton	21 (17.5)	24 (20.0)	0.61

This Table shows the distribution of athletes across different sports categories in both study groups.

Table 3: History of Prior Knee Injury

Type of Injury	Osteoarthritis Group (n, %)	Control Group (n, %)	p-value
ACL injury	35 (29.2)	14 (11.7)	<0.001
Meniscal tear	27 (22.5)	12 (10.0)	0.008
Patellar tendinopathy	16 (13.3)	18 (15.0)	0.71
None	42 (35.0)	76 (63.3)	<0.001

This Table summarizes the frequency of previous knee injuries among participants.

Table 4: Static Alignment Parameters

Alignment Variable	Osteoarthritis Group (mean ± SD)	Control Group (mean ± SD)	p-value
Mechanical axis deviation (degrees)	4.9 ± 1.2 (varus)	2.3 ± 0.9 (varus)	<0.001
Q-angle (degrees)	17.2 ± 2.4	15.8 ± 2.3	0.001
Tibial torsion (degrees)	19.4 ± 3.1	18.8 ± 2.9	0.26

This Table compares limb alignment characteristics between groups.

Table 5: Kinematic Variables during Landing Tasks

Variable	Osteoarthritis Group (mean ± SD)	Control Group (mean ± SD)	p-value
Knee flexion angle at initial contact (°)	18.6 ± 4.3	23.9 ± 5.1	<0.001
Peak knee flexion (°)	49.8 ± 6.9	55.4 ± 7.2	<0.001
Knee valgus angle (°)	11.5 ± 3.2	9.4 ± 2.8	0.002

This Table presents knee joint kinematic parameters observed during single-leg drop landings.

Table 6: Kinetic Parameters during Landing and Running Tasks

Parameter	Osteoarthritis Group (mean ± SD)	Control Group (mean ± SD)	p-value
Peak vertical GRF (BW)	3.01 ± 0.41	2.58 ± 0.38	<0.001
Knee adduction moment (Nm/kg·m)	0.47 ± 0.11	0.34 ± 0.10	<0.001
Tibiofemoral contact force (BW)	2.86 ± 0.55	2.37 ± 0.49	<0.001

This Table shows kinetic measurements reflecting joint loading during functional tasks.

Table 7: Neuromuscular Control Parameters

Variable	Osteoarthritis Group (mean ± SD)	Control Group (mean ± SD)	p-value
Quadriceps activation latency (ms)	54.6 ± 12.1	50.1 ± 11.4	0.01
Hamstring activation latency (ms)	63.9 ± 13.5	56.7 ± 12.7	0.001
Co-contraction index (Q–H ratio)	0.71 ± 0.12	0.82 ± 0.11	<0.001

This Table reports EMG-based muscle activation and co-contraction indices.

Table 8: MRI-Based Cartilage and Meniscal Changes at Baseline

MRI Feature	Osteoarthritis Group (mean ± SD)	Control Group (mean ± SD)	p-value
Cartilage thickness (mm)	2.31 ± 0.35	2.61 ± 0.32	<0.001
WORMS cartilage score	12.8 ± 3.7	4.9 ± 2.8	<0.001
Meniscal signal changes (%)	41.7	12.5	<0.001

This Table shows baseline MRI findings using WORMS scoring.

Table 9: Correlation between Biomechanical Variables and Functional Scores

Variable	KOOS correlation (r)	IKDC correlation (r)	p-value
Knee adduction moment	-0.58	-0.54	<0.001
Tibiofemoral contact force	-0.47	-0.45	<0.001
Knee flexion at landing	0.51	0.48	<0.001
Co-contraction index	0.43	0.39	<0.001

This Table presents correlation coefficients between key biomechanical factors and clinical outcomes.

Table 10: Predictors of Radiographic Progression at 24 Months

Predictor	Adjusted OR (95% CI)	p-value
Knee adduction moment (per 0.1 Nm/kg·m increase)	1.41 (1.15–1.74)	0.001
Tibiofemoral contact force (per 0.5 BW increase)	1.27 (1.08–1.61)	0.01
Reduced knee flexion at landing (<20°)	2.36 (1.22–4.55)	0.009
Prior ACL injury	1.89 (1.03–3.45)	0.04
Male sex	1.22 (0.71–2.10)	0.46

This Table lists significant baseline biomechanical predictors of OA progression in logistic regression analysis.

Table 11: Sport Type and Osteoarthritis Severity

Sport	Mean WORMS Score ± SD	p-value
Football	13.6 ± 4.1	<0.001
Basketball	12.7 ± 3.6	0.002
Athletics	11.1 ± 3.3	0.04
Badminton	9.8 ± 3.1	0.07

This Table compares mean WORMS cartilage degeneration scores across different sports.

Table 12: Longitudinal Changes in Pain and Function Scores

Outcome Measure	Baseline (mean ± SD)	24 Months (mean ± SD)	p-value (within-group)
KOOS Pain (Osteoarthritis group)	66.5 ± 9.1	58.9 ± 8.8	<0.001
KOOS Function (Osteoarthritis group)	69.3 ± 8.4	62.7 ± 8.0	<0.001
IKDC Total (Osteoarthritis group)	71.4 ± 7.9	63.2 ± 7.6	<0.001

This Table compares changes in functional outcomes over the 24-month follow-up.

Table 1 confirms baseline homogeneity between groups. Table 2 illustrates similar sport distribution, eliminating sport-type bias. Table 3 shows significantly higher frequencies of ACL and meniscal injuries among osteoarthritis athletes, reinforcing post-traumatic contribution. Table 4 highlights greater varus alignment and increased Q-angle in affected knees, indicating static malalignment as a structural risk factor. Table 5 demonstrates that osteoarthritis athletes landed with reduced flexion and increased valgus angles, signifying stiffer and asymmetrical movement patterns. Table 6 documents higher ground reaction forces, knee adduction moments, and tibiofemoral contact forces in the osteoarthritis group, confirming increased joint loading. Table 7 shows delayed hamstring activation and reduced co-contraction, suggesting impaired neuromuscular control. Table 8 reveals more severe MRI-detected cartilage and meniscal degeneration in affected athletes. Table 9 identifies strong correlations between elevated joint loading and poorer KOOS and IKDC scores. Table 10 demonstrates that increased adduction moment, higher contact force, reduced flexion, and prior ACL injury independently predict radiographic progression. Table 11 shows higher structural degeneration in football and basketball players, likely due to repetitive pivoting mechanics. Table 12 illustrates worsening pain and function over time among osteoarthritis athletes.

Overall, the results indicate that excessive medial knee loading, stiff landings, reduced neuromuscular coordination, and prior ligamentous injuries are the primary biomechanical factors contributing to both the development and progression of knee osteoarthritis in athletes.

DISCUSSION

This study comprehensively examined the biomechanical factors associated with the development and progression of knee osteoarthritis in athletes. The findings provide robust evidence that increased medial joint loading, altered kinematics during landing, impaired neuromuscular control, and prior ligamentous injuries contribute significantly to both the onset and advancement of degenerative joint disease in athletic populations. By combining motion analysis, force plate kinetics, electromyography, and longitudinal imaging, this study establishes a clear mechanical profile for athletes at high risk of knee osteoarthritis.

Baseline demographic comparability ensured that observed differences were attributable to biomechanical rather than anthropometric or sport-type variations. Athletes with osteoarthritis demonstrated significantly higher frontal-plane knee adduction moments and tibiofemoral contact forces, confirming the established relationship between medial compartment overload and cartilage degeneration.[8] Elevated adduction moments increase compressive stresses on the medial tibiofemoral cartilage, accelerating chondrocyte apoptosis and matrix degradation. This mechanical pattern has been consistently documented as a primary kinetic marker of disease severity and progression, and the present data extend those observations to an Indian athletic cohort.[9]

Reduced knee flexion angles at initial ground contact and at peak flexion during landing tasks were also prominent among athletes with osteoarthritis. These stiffer landing strategies limit energy dissipation and increase impact transmission through the knee joint. Athletes with reduced flexion are less able to utilize

muscular shock absorption, resulting in higher instantaneous ground reaction forces and tibiofemoral contact loads. The correlation between lower flexion angles and higher pain scores, as seen in this study, supports the concept that abnormal sagittal-plane mechanics not only contribute to structural degeneration but also to symptomatic expression.[10]

Static malalignment further amplifies dynamic loading abnormalities. The observed increase in varus mechanical axis deviation and greater Q-angle among affected athletes predispose the medial compartment to disproportionate stress. Malalignment also alters muscle activation patterns, reinforcing maladaptive loading cycles. The combination of alignment deviation and altered motion pattern represents a mechanical feedback loop that accelerates disease progression.[11]

Neuromuscular assessment revealed delayed hamstring activation and diminished quadriceps–hamstring co-contraction in the osteoarthritis group. Timely and balanced activation of these muscle groups is essential for dynamic stabilization and distribution of knee loads.[12] A lower co-contraction index implies inadequate antagonist support, permitting higher shear and compressive forces at the articular surfaces. The latency difference of approximately 7 milliseconds in hamstring activation observed here, although small in absolute terms, is biomechanically significant given the rapid deceleration demands of athletic movements.[13]

The logistic regression analysis identified increased knee adduction moment, higher tibiofemoral contact force, reduced flexion at landing, and previous anterior cruciate ligament injury as independent predictors of radiographic progression over two years. This aligns with prior longitudinal work demonstrating that post-traumatic knees experience altered proprioception, residual laxity, and abnormal contact kinematics even after surgical reconstruction. In this study, nearly one-third of athletes with prior ACL injury developed measurable osteoarthritis progression, highlighting the importance of neuromechanical retraining during rehabilitation.[14]

Sport-specific analyses showed that football and basketball players had the highest structural degeneration scores. Both sports involve repetitive pivoting, sudden deceleration, and high-frequency impacts, which elevate cumulative joint load exposure. In contrast, athletes in badminton and athletics exhibited comparatively lower structural scores, possibly reflecting more linear motion patterns and shorter load cycles. These sport-based distinctions suggest that preventive biomechanical interventions must be discipline-specific.[15]

The strong inverse correlations between knee adduction moment, tibiofemoral contact force, and functional scores (KOOS and IKDC) emphasize that biomechanical abnormalities not only predict structural deterioration but also impair performance and quality of life. Functional decline is likely mediated through pain amplification, altered proprioception, and compensatory movement adaptations that perpetuate mechanical inefficiency. The data collectively support the model of osteoarthritis as a dynamic, load-mediated process rather than a purely degenerative phenomenon.[16]

From a preventive standpoint, these findings reinforce the importance of optimizing movement mechanics early in an athlete's career. Strategies that enhance knee flexion during landing, improve hamstring activation timing, and correct malalignment may reduce cumulative joint loading. Evidence-based interventions such as neuromuscular retraining, eccentric strengthening, real-time biofeedback, and footwear optimization can restore symmetrical mechanics and mitigate risk. Motion-capture–guided screening programs could be incorporated into preseason evaluations to identify high-risk individuals.[17]

The longitudinal follow-up confirmed that athletes exhibiting abnormal baseline biomechanics had a greater likelihood of structural progression. This prospective evidence provides strong support for biomechanical screening as a predictive tool for early osteoarthritis detection. Although the study was conducted at a single tertiary center, the sample size, use of advanced motion analysis, and inclusion of multiple sports enhance its validity and applicability. The absence of significant biochemical or metabolic confounders strengthens the causal interpretation of biomechanical influence.[18]

Limitations include the lack of in-shoe pressure mapping to quantify localized plantar kinetics and the exclusion of female hormonal-phase analysis, which may influence ligament laxity and neuromuscular control. Moreover, while MRI provides superior resolution, its sensitivity to subtle cartilage biochemical changes is limited; incorporation of T2 mapping or dGEMRIC imaging could improve early detection in future work. Despite these limitations, the study provides the most comprehensive biomechanical dataset on athletic knee osteoarthritis from an Indian context.

In summary, this study demonstrates that excessive medial loading, reduced knee flexion, impaired neuromuscular coordination, and prior ligamentous injury are the principal biomechanical factors driving both the development and progression of knee osteoarthritis in athletes. The integration of biomechanical assessment into routine athletic screening and rehabilitation protocols could substantially reduce the burden of degenerative knee disease among sports participants.

CONCLUSION

Biomechanical loading abnormalities are central to the pathogenesis of knee osteoarthritis in athletes. Elevated knee adduction moments, reduced flexion during landings, increased tibiofemoral contact forces, and impaired muscle coordination collectively contribute to early joint degeneration and

symptomatic decline. The findings highlight the potential of biomechanical screening, motion retraining, and alignment correction as key strategies for prevention and management. Incorporating such evidence-based biomechanical interventions into athletic training and rehabilitation programs may reduce the incidence and progression of osteoarthritis, enabling longer, healthier athletic careers.

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