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Competitive Running and the Risk of Hip, Knee, and Ankle Osteoarthritis: Cartilage Adaptation, Degeneration, and Joint Replacement Outcomes — A Narrative Review

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ABSTRACT

Introduction and purpose. Running provides well-established cardiovascular and metabolic benefits, but its long-term influence on articular cartilage and osteoarthritis (OA) remains debated, particularly in competitive athletes exposed to high cumulative loading. This narrative review aimed to synthesize current evidence regarding the influence of competitive running on cartilage health, OA risk, and long-term outcomes in the hip, knee, and ankle joints.

Brief description of the state of knowledge. Current evidence suggests that recreational running is not consistently associated with increased OA prevalence and may even be protective compared with sedentary behavior. In contrast, competitive and elite running may be associated with higher OA risk, especially when combined with previous injury, excessive training volume, malalignment, elevated body mass index, or biomechanical abnormalities. MRI studies indicate that many exercise-induced cartilage changes are transient and may reflect physiological adaptation rather than irreversible degeneration.

Conclusions. Running should not be regarded as a universal independent cause of osteoarthritis. Joint degeneration in runners appears to result from an interaction between cumulative mechanical loading, injury history, biomechanics, recovery capacity, and individual susceptibility. Recreational running is generally compatible with joint health, whereas prolonged high-intensity competitive running may increase degeneration risk in predisposed individuals.

Keywords: ankle joint; cartilage degeneration; hip joint; knee joint; osteoarthritis; running

1. Introduction

Running is one of the most accessible forms of physical activity and is associated with cardiovascular, metabolic, musculoskeletal, and psychological benefits [4,9,12]. It is practiced both recreationally and competitively, ranging from occasional jogging to structured high-volume endurance training and professional sport [1,4,12]. Despite these benefits, concerns persist that repetitive lower-limb loading during running may accelerate articular cartilage degeneration and contribute to osteoarthritis (OA), particularly in the hip, knee, and ankle joints [1,7,12].

Articular cartilage is an avascular, aneural, highly specialized connective tissue that distributes load, reduces friction, and protects subchondral bone within synovial joints [10,14]. Its limited intrinsic regenerative capacity has historically supported the concern that repetitive impact loading could cause progressive cartilage damage [1,25,29]. However, contemporary mechanobiological evidence indicates that cartilage requires physiological loading to maintain extracellular matrix integrity and that moderate exercise may support cartilage homeostasis [10,18,24].

The relationship between running and OA is therefore not linear. Recreational running has not been consistently associated with increased hip or knee OA and may be associated with lower OA prevalence than sedentary behavior [1,5,31,34]. In contrast, competitive and elite running may be associated with a higher prevalence of OA, probably due to greater cumulative load, higher training intensity, and a higher frequency of previous joint injuries [1,12,19,29]. A recent Quality in Sport literature review similarly concluded that recreational running appears neutral or beneficial for joint health, whereas high-level competitive and Olympic running may increase OA risk [12].

Osteoarthritis is currently understood as a whole-joint disease involving cartilage, subchondral bone, synovium, menisci, ligaments, periarticular muscles, and inflammatory and metabolic pathways [11,14]. Mechanical factors remain central to OA initiation and progression, but they interact with age, body mass index, genetic susceptibility, sex, previous injury, malalignment, and neuromuscular control [2,11,14,23]. Therefore, the orthopedic consequences of running should be interpreted through a multifactorial model rather than through the simplistic statement that “running destroys joints” [1,9,12].

The purpose of this narrative review was to synthesize current evidence regarding the influence of competitive running on articular cartilage, OA risk, and long-term outcomes involving the hip, knee, and ankle joints, with particular emphasis on cartilage adaptation, degeneration, injury-related risk, and arthroplasty-related outcomes.

A narrative literature search was performed using PubMed, Scopus, and Google Scholar databases. The search included combinations of the keywords “running”, “osteoarthritis”, “cartilage”, “knee”, “hip”, “ankle”, “athletes”, “MRI”, and “joint replacement”. Priority was given to systematic reviews, longitudinal cohort studies, MRI-based investigations, and clinically relevant observational studies published in English. Due to substantial heterogeneity in study design, imaging protocols, athlete populations, and OA definitions, a narrative rather than systematic review methodology was considered more appropriate.

2. Biomechanical Load and Cartilage Adaptation During Running

Running exposes the lower limb to repetitive compressive, tensile, and shear forces transmitted through the ankle, knee, and hip joints [2,13,22]. Ground reaction forces during running may reach several times body weight and vary according to speed, cadence, stride length, surface, footwear, fatigue, and individual running mechanics [13,22]. These forces are not inherently pathological; rather, cartilage and periarticular tissues respond dynamically to mechanical stimuli through mechanotransduction pathways [10,18].

Physiological cyclic loading can stimulate chondrocyte activity, promote nutrient exchange, and maintain extracellular matrix properties [10,18,24]. Roos and Dahlberg demonstrated that moderate exercise may positively influence glycosaminoglycan content in knee cartilage, supporting the concept that cartilage can adapt favorably to appropriate loading [24]. Conversely, insufficient loading, excessive loading, or inadequate recovery may disturb cartilage metabolism and contribute to degeneration [10,16,24].

MRI studies have demonstrated that long-distance running may produce acute changes in cartilage thickness, T2 relaxation times, delayed gadolinium-enhanced MRI of cartilage parameters, and cartilage hydration patterns [8,17,27,32]. Importantly, many of these changes appear transient and reversible after recovery, suggesting physiological deformation and fluid redistribution rather than irreversible structural damage. Elevated T2 relaxation times do not necessarily indicate irreversible degeneration and may reflect temporary alterations in hydration, collagen fiber orientation, or physiological adaptation to mechanical loading [8,17,27,32]. A 2025 comprehensive review in the Journal of Education, Health and Sport noted that MRI studies have shown temporary decreases in knee cartilage thickness after acute exercise, while long-term effects of endurance running remain inconclusive [4].

Recent quantitative MRI evidence has refined this interpretation. Jandacka et al. reported that regular running distance and frontal-plane knee biomechanics were related to medial femoral cartilage T2 values in healthy adults [16]. In that study, runners covering 6–20 km per week showed more favorable medial central femoral cartilage structure than nonrunners and highly active individuals, whereas runners exceeding 41 km per week had higher T2 values, suggesting a possible dose-dependent or U-shaped relationship [16]. Similarly, Zhang et al. reported that long-term running may be associated with regional increases in knee cartilage thickness and volume, indicating that repeated mechanical exposure can produce structural adaptation rather than uniform degeneration [35].

However, very high loads may exceed adaptive capacity. Della Rosa et al. found that ultra-trail running was associated with knee cartilage ultrastructural modifications persisting for at least one month after the event, which suggests that extreme endurance exposure may require prolonged recovery and may not be directly comparable with recreational running [8]. These data support a model in which cartilage response depends on the dose, intensity, recovery interval, tissue condition, and individual biomechanics [8,16,35].

3. Knee Joint: Cartilage Degeneration and Osteoarthritis Risk

The knee is the most extensively studied joint in research on running and OA. This is clinically relevant because the knee is frequently affected by OA and is highly exposed to repetitive loading during running [1,14,23]. Nevertheless, the available evidence does not support a simple causal relationship between running and knee OA [1,5,9,20,31].

Alentorn-Geli et al. reported that recreational runners had a lower prevalence of hip and knee OA than sedentary individuals and competitive runners [1]. Their meta-analysis indicated that OA prevalence was lowest among recreational runners and highest among competitive runners, supporting a nonlinear dose-response relationship [1]. Chakravarty et al. similarly found that long-distance running was not associated with accelerated radiographic knee OA progression in older runners over long-term follow-up [5]. Williams reported that running and walking were associated with reduced risk of OA and hip replacement, largely mediated by lower body mass index and health-related selection factors [34].

More recent evidence remains consistent with this interpretation. Dhillon et al. concluded that, in the short term, running was not associated with worsening patient-reported outcomes or radiological signs of knee OA and may be protective against generalized knee pain [9]. Lo et al. similarly reported that running did not increase symptoms or structural progression in individuals with knee OA [20]. These findings are important clinically because they argue against advising all patients or athletes to stop running solely because of fear of OA progression [9,20].

In competitive athletes, the situation is more complex. Kujala et al. demonstrated knee OA in former elite runners, soccer players, weight lifters, and shooters, with sport-specific differences suggesting that both cumulative loading and injury exposure influence later degeneration [19]. Spector et al. found that long-term participation in weight-bearing sports was associated with OA risk, particularly when exposure was intensive [29]. Competitive running may therefore be associated with increased OA risk in some athletes, but this risk is probably mediated by total training dose, injury history, joint morphology, and biomechanics [1,12,19,29].

Previous knee injury is one of the strongest predictors of OA development in athletes [9,14,23]. Meniscal tears, anterior cruciate ligament rupture, chondral lesions, osteochondral injury, and post-surgical changes substantially increase long-term OA risk [9,14,23]. This is crucial when interpreting elite sport cohorts because professional runners and other high-level athletes may continue training despite pain, accumulate microtrauma, and sustain traumatic injuries at higher rates than recreational runners [12,13,19].

Biomechanics also modifies risk. Abnormal frontal-plane mechanics, malalignment, altered knee adduction moments, quadriceps weakness, impaired hip control, and excessive joint loading can influence regional cartilage stress [2,16,22,28]. Jandacka et al. found that frontal-plane knee biomechanics were related to medial femoral cartilage structure, suggesting that running mechanics may be a modifiable factor in cartilage health [16]. Miller et al. proposed that per-unit-distance joint loading, tissue adaptation, and load distribution may help explain why most runners do not develop knee OA despite repetitive loading [22].

MRI-based studies support the distinction between transient adaptation and pathological degeneration. Kessler et al. reported recovery of meniscus and cartilage changes after long-distance running [17]. Shen et al. used MRI to analyze knee injuries and related factors in amateur marathon runners, further supporting the relevance of imaging-based assessment in running populations [27]. However, extreme

endurance events, such as ultra-trail running, may induce more persistent cartilage changes and should be interpreted separately from moderate running [8].

Overall, knee OA risk in runners appears most strongly associated with excessive cumulative exposure, prior joint injury, high training intensity, malalignment, and abnormal mechanics rather than with running as an isolated activity [1,9,12,16,20].

4. Hip Joint: Degeneration and Arthroplasty Risk

The hip joint is less extensively studied than the knee in relation to running, but available evidence suggests that elite athletic exposure may increase the risk of hip OA and total hip arthroplasty in selected populations [1,21,30,33,34]. During running, the hip transmits repetitive forces through the acetabulofemoral articulation, and joint contact forces increase with running speed, stride mechanics, and fatigue [2,22,28].

Vingård et al. reported an association between participation in certain sports and later hip osteoarthrosis, suggesting that high cumulative loading may contribute to hip degeneration [33]. Michaëlsson et al. found that severe hip and knee OA risk differed according to physical exercise level, supporting the idea that very high activity exposures may increase risk in some individuals [21]. Spector et al. also reported OA risk associated with long-term weight-bearing sports [29].

However, hip OA in athletes cannot be attributed to running alone. Structural predispositions such as cam morphology, pincer morphology, acetabular dysplasia, labral pathology, and femoroacetabular impingement may increase focal chondrolabral stress during repetitive motion [14,28]. In such athletes, running may accelerate degeneration in an already predisposed joint rather than initiate OA independently [14,28,33].

The literature also suggests that recreational running does not carry the same risk profile as professional sport. Alentorn-Geli et al. found that recreational runners had lower hip and knee OA prevalence than sedentary individuals and competitive runners [1]. Williams reported that running was associated with a lower risk of hip replacement, although the relationship was influenced by body mass index and other health-related factors [34]. These findings support the view that moderate running is not inherently harmful to the hip joint [1,34].

Long-term arthroplasty outcomes are particularly relevant for clinical counseling. In former elite athletes, increased rates of total hip arthroplasty have been reported, but causality remains difficult to establish because high-level sport involves selection bias, injury burden, morphological predisposition, and training exposure [21,30,33]. Therefore, hip OA risk in runners should be framed as multifactorial and individualized rather than inevitable.

5. Ankle Joint: A Distinct Biomechanical Environment

The ankle joint differs from the knee and hip because primary ankle OA is much less common and most ankle OA is post-traumatic [3,25,31]. Saltzman et al. reported that ankle arthritis has a distinct epidemiology compared with knee and hip OA, with trauma playing a dominant etiological role [25]. Valderrabano et al. similarly found that ankle OA is most often related to previous trauma, including fractures and chronic instability [31].

Ankle cartilage is thinner than knee cartilage but has high congruity and distinct mechanical properties, which may partly explain the lower frequency of primary ankle OA [3,25,31]. In runners, ankle

degeneration is therefore more plausibly linked to recurrent sprains, osteochondral lesions, instability, fractures, and altered biomechanics than to repetitive running alone [3,13,25,31].

The direct evidence linking running itself to ankle OA remains limited. Most available studies on ankle OA emphasize trauma, instability, and post-traumatic degeneration rather than endurance running as an independent cause [3,25,31]. This creates an important limitation in any review addressing the hip, knee, and ankle together: the evidence base is strongest for the knee, moderate for the hip, and weakest for the ankle.

Clinically, this means that ankle-joint preservation in runners should focus on prevention and adequate rehabilitation of ankle sprains, restoration of proprioception, management of chronic instability, and correction of training errors [3,13,25,31].

6. Recreational Versus Competitive Running

The distinction between recreational and competitive running is essential. In this review, recreational runners are defined as individuals participating in running primarily for health, fitness, or leisure purposes without structured elite-level competition. Competitive runners refer to athletes participating in organized endurance events with higher training volume and performance-oriented goals, whereas elite or professional runners represent individuals exposed to long-term high-intensity training and substantial cumulative running loads [1,12,19]. Recreational running generally involves lower weekly mileage, lower intensity, more flexible recovery, and lower cumulative lifetime exposure than elite sport [1,12,16]. In contrast, competitive runners may accumulate high mileage over many years, train at higher intensity, and continue training during pain or after incomplete recovery [12,13,19].

Several studies suggest that recreational running is not associated with increased OA risk and may be protective compared with sedentary behavior [1,5,9,20,31,34]. This may be explained by lower body mass index, improved metabolic health, enhanced muscular support, and favorable cartilage mechanoadaptation [10,16,24].

Competitive running, however, may involve a different biological and mechanical environment. High-volume running, particularly above individual recovery capacity, may be associated with increased cartilage stress and overuse injury risk [8,12,16]. The 2024 study by Jandacka et al. supports a possible U-shaped relationship, in which moderate running was associated with more favorable medial femoral cartilage structure, whereas very high weekly running distance was associated with higher T2 values [16]. This finding is highly relevant because it provides recent imaging-based support for the distinction between beneficial loading and potential overload.

Thus, the clinical message should not be that running is harmful, but that the dose, context, recovery, and injury history matter [1,9,12,16].

7. Risk Modifiers and Confounding Factors

The effect of running on cartilage and OA risk is strongly modified by individual and sport-related factors. Previous joint injury is among the most important risk factors, particularly meniscal injury, anterior cruciate ligament rupture, osteochondral injury, and intra-articular fracture [9,14,23]. Such injuries can alter joint congruity, load distribution, inflammatory pathways, and neuromuscular control, accelerating OA development independent of running exposure [9,14,23].

Body mass index is another major modifier. Higher body mass increases absolute joint loading during running and is independently associated with OA risk [14,23]. Williams suggested that part of the

apparent protective association between running and hip replacement risk may be mediated by lower body mass among runners [34].

Alignment and biomechanics also influence risk. Varus or valgus alignment, abnormal knee adduction moments, excessive hip internal rotation, impaired hip abductor function, and altered trunk control can change regional loading patterns across the lower limb [2,16,28]. Souza and Powers demonstrated that hip mechanics are relevant during running and may influence downstream knee loading [28].

Training characteristics are also relevant. Abrupt increases in mileage, insufficient rest, hard surfaces, high-intensity sessions, fatigue, and inadequate strength preparation may contribute to overuse injury [13]. Since injury is a major pathway toward OA, training-load management is indirectly central to joint preservation [9,12,13].

Age, sex, genetics, and joint morphology further affect susceptibility. Jandacka et al. found that age and sex were associated with medial femoral cartilage T2 values, indicating that biological factors influence cartilage response to running exposure [16]. Women may demonstrate different OA susceptibility profiles due to hormonal influences, pelvic and lower-limb biomechanics, differences in ligamentous laxity, and sex-related injury patterns, particularly regarding anterior cruciate ligament injury prevalence [14,23].

8. Clinical Implications and Prevention Strategies

Clinicians should avoid advising complete cessation of running solely because of generalized fear of OA. Current evidence supports recreational running as generally safe for joint health in individuals without major risk factors or uncontrolled symptoms [1,5,9,20,31,34]. In many cases, running may support body-weight control, cardiometabolic health, muscle function, and cartilage homeostasis [4,9,10,24].

In competitive athletes, prevention should focus on modifiable risk factors. These include progressive load management, adequate recovery, treatment of pain and injury, correction of major biomechanical abnormalities, strength training, neuromuscular conditioning, and individualized monitoring of athletes with previous joint trauma [9,12,13,16].

The knee should receive particular attention because it is the joint with the strongest evidence base and the most frequent concern among runners [1,9,16]. Hip evaluation should be considered in athletes with groin pain, limited range of motion, suspected femoroacetabular impingement, or labral pathology [14,28,33]. Ankle prevention should prioritize recurrent sprain prevention, proprioceptive training, and management of chronic instability, because ankle OA is predominantly post-traumatic [3,25,31].

Overall, counseling should be individualized. For recreational runners, the message should emphasize safe continuation and gradual progression. For competitive runners, the message should emphasize dose control, injury prevention, adequate recovery, and early response to persistent joint symptoms [9,12,13,16].

9. Limitations of the Current Evidence

The literature has several limitations. First, most available studies are observational and therefore demonstrate associations rather than direct causality. Consequently, caution is required when interpreting relationships between long-term running exposure and OA progression.

Second, many studies differ in definitions of running exposure, OA diagnosis, imaging modality, follow-up duration, and athlete classification [1,9,12]. Third, cross-sectional designs are common and cannot establish causality [16,29]. Fourth, selection bias and survivorship bias may influence long-term runner cohorts. Individuals capable of maintaining running over many years may represent a healthier subgroup with greater musculoskeletal resilience, whereas runners developing severe pain or early degeneration may discontinue activity and become underrepresented in athletic cohorts.

Fifth, previous injury is a major confounder because elite athletes are more likely to have experienced joint trauma, surgery, and recurrent overuse injury [9,14,19,23].

Sixth, imaging abnormalities do not always correlate with symptoms or long-term clinical outcomes [9,14]. MRI can detect early biochemical and structural changes, but the clinical meaning of transient T2 or cartilage-thickness changes remains uncertain [8,16,17,27]. Seventh, evidence is uneven across joints: knee data are relatively abundant, hip data are moderate, and ankle-specific running data remain limited [1,3,25,31].

Future research should prioritize prospective longitudinal studies using standardized MRI protocols, objective training-load assessment, injury-history stratification, and clinically meaningful endpoints such as symptomatic OA and arthroplasty [8,16].

9.1. Practical Summary of Current Evidence

Current evidence supports a continuum model of cartilage response to running exposure. Moderate physiological loading may promote cartilage maintenance and favorable adaptation, whereas excessive cumulative loading combined with injury, malalignment, inadequate recovery, or abnormal biomechanics may contribute to degeneration [1,10,16,24]. Recreational running appears generally compatible with long-term joint health, while elite-level cumulative exposure may be associated with increased OA prevalence in selected populations [1,12,19,29,34].

Key modifiers influencing OA risk in runners include:

- previous joint injury,
- cumulative training volume,
- recovery capacity,
- body mass index,
- lower-limb biomechanics and alignment,
- neuromuscular control,
- genetic and biological susceptibility.

Thus, current evidence does not support the simplified statement that “running destroys joints,” but rather indicates that cartilage adaptation and degeneration exist along a spectrum determined by load, tissue resilience, and injury history [1,9,12,16].

10. Conclusions

Running should not be considered a universal independent cause of osteoarthritis. Recreational running appears generally compatible with joint health and may be associated with lower OA prevalence compared with sedentary behavior [1,5,9,20,31,34].

Competitive and elite running may be associated with increased degeneration risk in selected individuals, particularly when high cumulative loading is combined with prior injury, malalignment, excessive training volume, inadequate recovery, or unfavorable biomechanics [1,8,12,16,19,29].

The knee has the strongest evidence base and appears to be influenced by both running dose and injury history. Hip OA risk in athletes appears multifactorial and may be amplified by structural predisposition and high cumulative exposure. Ankle OA is more strongly associated with trauma and instability than with repetitive running alone [3,25,31].

The most accurate model is therefore not that running destroys cartilage, but that cartilage adaptation and degeneration exist on a continuum determined by mechanical dose, tissue recovery, injury history, and individual biological susceptibility.

Disclosure:

Author Contributions

Conceptualization: K.P.

Methodology: K.P., W.P.

Data curation: E.B., A.T., A.B.

Writing: K.P., J.S.

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Conflicts of Interest

The authors declare no conflicts of interest.

AI

During the preparation of this work, the authors used OpenAI ChatGPT
for the purpose of basic data analysis and to identify linguistic patterns associated with specific logical fallacies. It is important to emphasize that the AI tool was used strictly as an assistive instrument under human supervision. Human experts in clinical medicine and formal logic determined the final interpretation of results, classification of errors, and conclusions, and take full responsibility for the substantive content of the publication.

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