Risk Factors for Hip Osteoarthritis: Insight for the Strength and Conditioning Professional

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ABSTRACT

HIP OSTEOARTHRITIS (OA) WILL AFFECT APPROXIMATELY 25% OF THE POPULATION AT SOME POINT IN THEIR LIFETIME. ALTHOUGH THE ETIOLOGY OF HIP OA IS MULTI-FACTORIAL, RISK FACTORS SEEM TO HAVE A ROLE IN BOTH THE INCIDENCE AND ESCALATION OF SIGNS AND SYMPTOMS. THIS ARTICLE PRESENTS AN OVERVIEW OF RISK FACTORS ASSOCIATED WITH HIP OA. EVIDENCE UNDERPINNING AND REFUTING SOME OF THE COMMONLY PURPORTED RISK FACTORS IS DISCUSSED WITHIN THE CONTEXT OF PROVIDING THE STRENGTH AND CONDITIONING PROFESSIONAL WITH EVIDENCE-BASED RECOMMENDATIONS FOR THEIR CLIENTS.

INTRODUCTION

Epidemiological reports indicate that the femoroacetabular joint, hereafter referred to as the hip joint, is a source of pain in up to 15% of the population at any given time with trends for a higher prevalence among women and the elderly (8,12,45). Of the various diagnoses implicated in the etiology of hip joint pain, osteoarthritis (OA) (also known as osteoarthrosis or degenerative joint disease) has had the greatest economic consequence and is one of the highest contributors to the global disability burden (11). The hip joint ranks second to the knee among large joints in the body affected by OA (11,14). Evidence suggests that the estimated lifetime prevalence of symptomatic OA in the hip is approximately 25%; thus, one in 4 individuals may be affected by this condition (37). Owing to the degenerative nature of OA, pathological progression is likely, which may over time lead to functional decline with joint replacement being a viable end-stage intervention for those recalcitrant to conservative interventions (6). Evidence suggests that acceleration of the degenerative process and the associated morbidity sequela may be based on various demographic attributes and risk factors (13). Although not all risk factors are modifiable, it would seem reasonable that controlling for risk factors through lifestyle recommendations may attenuate symptom progression and possibly decelerate the degenerative process.

When considering the prevalence of hip joint OA, it seems reasonable that strength and conditioning professionals will likely encounter individuals with either a formal diagnosis or those who may be at risk. Thus, an understanding of risk factors may provide insight into lifestyle recommendations and exercise prescription modifications that may serve useful to decelerate the degenerative cascade and associated impairment sequela. The purpose of this article is to provide an overview of risk factors associated with hip OA. Evidence underpinning and refuting some of the commonly purported risk factors is discussed within the context of providing the strength and conditioning professional with evidence-based recommendations for their clients.

KEY WORDS:

hip; osteoarthritis; prevention

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Although a detailed discussion of the pathogenesis of hip OA is beyond the scope of this article, a brief overview is necessary to understand the condition and recommendations pursued in this article. Readers interested in a more detailed discussion of the degenerative process are referred to the Osteoarthritis Research Society International (OARSI) grading schema (39). A summary of nomenclature used to describe the pathological changes discussed in this article can be found in the appendix (see Table, Supplemental Digital Content, http://links.lww.com/SCJ/A184).

The hip is a ball and socket joint (Figure) comprised of articular cartilage, subchondral bone, synovium, and a joint capsule. The joint surface comprises articular cartilage, which is an avascular structure (limited blood supply) composed primarily of chondrocytes (cells found in healthy cartilage that produce collagen) surrounded by a matrix that includes proteoglycans, glycosaminoglycans, and collagen (39). The articular cartilage protects the underlying subchondral bone by redistributing load and reducing friction at the joint during normal movements and activities. Healthy articular cartilage, as present in a hip joint without OA (Figure), is sustained under conditions whereby a balance between synthesis (building) and degradation (breaking down) of the cartilage occurs. Synovial fluid, which comprises, in part, hyaluronate, supplies nutrients to the joint and provides the viscosity needed to absorb loads and reduce friction. Essentially, synovial fluid serves to lubricate the healthy joint.

The condition of hip OA may be viewed as a degenerative joint disorder stemming from compromise or biomechanical breakdown of the synovium, articular cartilage, and subchondral bone, with evidence that inflammation, previous injury, and abnormal biomechanics may serve a causative role (1,20,28,35). OA is ultimately characterized by progressive damage to the articular cartilage (ends of long bones where they articulate as a joint) (Figure). Over time, a progressive loss of cartilage, referred to as chondropenia, occurs. Chondropenia results in decreased joint space, often at areas of high load such as the superior joint space. Erosion in the joint from a loss of cartilage and joint space occurs until the subchondral bone is exposed (articular cartilage is worn away). The erosion leads to increased biomechanical stress to the subchondral bone, which responds with a process known as eburnation (39). Eburnation is a condition whereby exposed subchondral bone undergoes a sclerosing process and hardens, resembling an ivory-like appearance on radiographs. Moreover, changes in the subchondral bone decrease the shock absorption capacity, thus rendering the hip joint at a greater risk for injury and episodes of inflammatory change. Furthermore, at areas of high stress along the articular margin, the outgrowth of irregular new bone develops in a process referred to as osteophytosis (bone spurs). Osteophytes are often produced as a result
of a response mechanism from focal trauma to a specific area of the joint susceptible to abnormal loading.

Symptomatic OA represents a heterogeneous cluster of signs and symptoms comprised of histopathologic and radiologic findings that coincide with movement impairments and pain. The etiology of pain and impairments experienced from hip OA is multifactorial and presumed to arise from a combination of mechanisms including the morphologic changes at the joint, episodes of inflammation, and ensuing impairments (20). Symptoms (pain, stiffness, and audible sounds) arising from hip OA do not evolve from articular cartilage damage alone. In particular, pain from OA stems from the subchondral bone (bone residing below the articular cartilage) and changes in the synovium, which may activate the synovial nociceptors (pain receptors). In addition, pain may stem from osteophytes, which are potentially aggravated with particular movements and positioning. Moreover, joint effusion (swelling), occurring from loading to morphologically abnormal joint surfaces, may stretch the joint capsule (which may already be shortened from disuse and degenerative changes) and be a source of symptoms because of pain receptor activity. Similar to other conditions, pain from OA may be chemically induced from injury to neighboring tissue (e.g., bursa or ligament) or coexisting problems such as muscle spasm, as well as psychological factors. From an impairment perspective, ensuing joint contractures (shortening of soft tissues) lead to pain, decreased mobility, and stiffness of the joint.

Collectively, the aforementioned degenerative changes and associated impairments serve a primary role leading to pain and impairment among clients with hip OA. Moreover, it is these changes and events that often lead to clinical symptoms and formal rehabilitative care, social and lifestyle restrictions, and activity impairments. Unfortunately, there is no evidence favoring the ability of a particular intervention to reverse the degenerative process. However, symptoms from OA (e.g., stiffness, pain, audible sounds) and the progression of structural changes may be accelerated by certain factors, some of which may be modifiable through lifestyle recommendations and activity modifications (13). Moreover, many of the ensuing impairments may be mitigated through safe participation in exercise and recreational activities (13).

### RISK FACTORS

Risk factors, particularly those that are modifiable (e.g., body mass, training patterns, lifestyle, hip positioning and loading), are of particular interest in reducing new incidence, steering prevention efforts, and attenuating disease progression (Tables 1 and 2). With an understanding of risk factors and their role in disease progression, strength and conditioning professionals may find themselves in an opportune position to reinforce and introduce relevant activity and lifestyle modifications to their clients. Furthermore, clients with hip OA or established risk factors may potentially experience worsening of their condition without the appropriate modifications to traditional routines. Appropriate exercise prescription and lifestyle recommendations that adhere to the evidence should mitigate the risk for a new onset or worsening of symptoms of hip OA and promote an increase in one’s activity level.

### DEMOGRAPHIC

In regards to race and ethnicity, there seems to be no consistently reported differences in the presence of hip OA (24). As stated previously, gender differences do exist with women being more likely than men to develop symptomatic hip OA (37,38). These differences are more evident after the fifth decade of life (38). With advancing age, the incidence of hip OA rises, with the most apparent increases occurring at age 50 and leveling off or declining by 80 years of age (4,38). Reports of pain related to hip OA increase with age (24) and may be attributed to the progression of the condition as well as ensuing impairments and loss of function. Although age is associated with OA, it is not a modifiable risk factor. Rather, the strength and conditioning professional should recognize the increased prevalence with age and the growing possibility of underlying degenerative changes when designing a program. Moreover, with advanced age and the potential for OA, one should be cognizant of particular activities known to play a role in regard to symptoms and disease progression. Fortunately, in regard to health and fitness attributes, appropriate exercise has been shown to delay the need for joint replacement surgery and reduce symptoms from hip OA irrespective of age (29,40).

### PREVIOUS INJURY

Trauma or surgery involving the articular cartilage or supporting structures may lead to abnormal biomechanics and cytokine imbalance, which could incite or accelerate the degenerative process. Cytokines are proteins released

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**Table 1**

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<tr>
<th>Risk factors associated with hip osteoarthritis (3,10,22,26,32,34,41,48)</th>
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<tbody>
<tr>
<td><strong>Modifiable risk factors</strong></td>
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<tr>
<td>Compulsory occupational stooping and squatting</td>
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<tr>
<td>Frequent stair climbing or vibration tool exposure</td>
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<tr>
<td>Long-term exposure to heavy lifting and standing</td>
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<tr>
<td>Elite level high impact sport participation</td>
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<td>Obesity or high body mass index ≥ 30</td>
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by cells and have an effect on many interactions. For example, both proinflammatory and antiinflammatory cytokines exist, thus may serve to promote or reduce inflammation depending on the specific subtype. Undue loading of a previously injured joint may predispose the region to degeneration as excessive forces can accelerate the catabolic effects of the chondrocytes and further disrupt the cartilaginous matrix. Previous injury to the hip is a clear risk factor for OA with an odds ratio (OR) of 5.0 (10,41). Essentially, individuals with a previous injury to the hip are 5 times more likely to develop OA when compared with those without previous injury. In many cases, a previous injury will create changes to the articular surface and biomechanical impairments that lead to an abnormal loading environment.

A key component to any exercise program would be an understanding of the client’s medical history. Identifying the presence of a previous hip injury or congenital hip problem does present a relevant risk factor. Unlike direct trauma, occupational or lifestyle factors may expose the hip to degenerative changes through a microtraumatic effect.

**MODIFIABLE RISK FACTORS**

Modifiable risks factors are often activities that once changed, can potentially decelerate the progression, and decrease the risk of new incidences of a condition (Table 1). Physical activity and loading are often areas of concern among health care professionals and the general population, with assumptions that partaking in regular physical exercise may increase wear and tear of the joints (13). Evidence indeed does support an association between physical activity and joint degeneration; however, results suggest a beneficial response to moderate exercise in regard to staving off surgery and reducing new incidences of symptomatic OA (13). Leisure time physical activities such as walking and cycling have been associated with a lower risk of degenerative OA when compared with sedentary activity levels (3). Overall, recreational sport participation has a low risk (10); however, high exposure to competitive or elite sporting activity before age 50 has been associated with a greater risk of hip OA (46). Contrary to recreational participation, high impact sports such as American football, track and field, and racket sports seem to increase the risk for hip OA (43,46). The risk among athletes is more evident in those participating at an elite level with an OR of 1.6–2.5 (43). Activities such as recreational running may have a protective effect in part because of an association with reduced body mass. Most investigations have not identified an increased risk of hip OA in runners; however, mixed evidence exists as related to running pace and mileage (36,47,48). A large cohort study reported contrasting findings (e.g., running posed no risk for hip OA). In fact, one study found that those who ran at higher energy expenditure had decreased risk when compared with those who exercised at a metabolic equivalent (MET) of <1.8 h/d (48). Recognizing that 1 MET is essentially the energy cost of sitting quietly, one may interpret the aforementioned MET of <1.8 h/d as that of those who were deconditioned and possessed other inherent risks. Nevertheless, in the same study walking in lieu of running did not decrease OA risk (48). The authors postulated a protective effect from running, particularly as it attenuates unhealthy weight gain, which is a known risk factor for hip OA. Given the association of running with a lower body mass index (BMI), it seems that recreational participation does not increase one’s risk for developing hip OA and may in fact reduce one’s risk profile.

Occupational activities that involve heavy lifting, squatting, climbing stairs, and long-term exposure to standing have been associated with hip OA (22,41). Heavy versus light workloads in particular place individuals at a 3 times greater risk for developing hip OA (33). Frequent or compulsory stair climbing has a reported OR of 12.5 (31). Moreover, a cross-sectional survey found that persons exposed to lifting, stooping, and vibration tools have an increased risk for hip OA (19). Evidence suggests that lifting burdens have to be at least 10 kg (22 lbs.), with performance >10 years to be related to hip OA (22). It seems logical that evidence-based recommendations for exercise among healthy individuals should favor recreational running over squatting or stair climbing. Moreover, while occupational hazards could be recognized, one’s choice of occupation may be in itself a risk factor. Clients who possess occupations placing them at risk should be recognized with appropriate considerations being made to avoid otherwise provocative activities during training (e.g., stair climbing, deep squats, and vibration platforms).

Numerous studies have identified BMI and obesity as risk factors for hip OA with ORs ranging from 1.6 to 15.4 (2,41). Obesity (BMI ≥ 30) has been associated with a higher prevalence of hip OA in one study, with an adjusted OR = 2.18 (2). Of those individuals with hip OA in the aforementioned study, obesity was associated with higher levels of pain, increased stiffness, decreased function, and reduced quality of life. A recent meta-analysis reported that a 5-unit increase in BMI (e.g., 32–37) was associated with an

| Table 2 Evidence for recreational and sport participation hip osteoarthritis risk |
|------------------------------------------------|-------------------|
| Exercise and athletics: risk association for hip osteoarthritis (10,36,41,43,46–48) |                     |
| Sports such as American football, track and field, and racket sports may ↑ risk |                     |
| Frequent stair climbing may ↑ risk |                     |
| Recreational running does not ↑ risk |                     |
| Walking in lieu of running does not ↓ risk |                     |
| Leisure cycling or walking does not ↑ risk |                     |
11% increase risk of hip OA (23). In addition to mechanical effects, obesity may be an inflammatory risk factor for OA owing to its link with increased levels of adipokines (9), which may promote joint inflammation. Evidence does exist to support a relationship between obesity and severe hip OA requiring a total hip replacement (end-stage intervention for hip OA). One study (26) evaluated 568 women with a BMI of greater than or equal to 35 kg/m² who received a hip replacement. The authors reported a relative risk of 2.6 for hip OA compared with a reference population. Although it is clear that an association exists, individuals with OA often adopt more sedentary lifestyles than reference populations subsequently leading to increases in BMI and a perpetuation of risk.

In regard to smoking and alcohol consumption, there is no evidence to suggest that cessation programs have a beneficial effect on hip OA (13). Although smoking may certainly affect outcomes after surgery, cessation does not seem to have an effect on the degenerative process. Certainly, cessation would be valuable for other health attributes.

Structural morphology. Developmental disorders such as dysplasia (misalignment of the joint–offset present at birth), congenital hip dislocation (hip dislocation at birth—often due to hip dysplasia), Legg–Calve–Perthes (disruption of blood flow to femoral head leads to bone necrosis), and slipped capital femoral epiphysis (growth plate fracture of femoral head induces slipping of femoral head) have been associated with OA (21). Undue loading of a developmentally dysplastic joint may predispose the region to degeneration as excessive forces accelerate the catabolic effects of the chondrocytes and further disrupt the cartilaginous matrix.

In regard to morphology, it has been suggested that lower extremity length inequality (difference \( \geq 2 \) cm) are more likely to have hip OA, although the association is weak (adjusted OR = 1.20), and it is not significantly associated with radiographic progression (17,18). Interestingly, one particular study found that OA is more common when the contralateral leg is longer compared with being shorter (e.g., right hip OA would be more common when left leg is longer) (18); however, the results are limited to this study alone. One potential downfall of this evidence for lower extremity length inequality is the measurement technique, as examiners used a tape measure (16–18). When considering the relevance of limb inequality, it should be recognized that hip OA itself may cause a leg length discrepancy and a shortened leg may simply be the result of joint space narrowing or protrusio acetabuli (e.g., medial protrusion of femoral head into acetabulum) as opposed to structural length change. Independent of radiographic changes, individuals with lower extremity length inequality are more likely to have hip pain, aching, and stiffness than those with symmetrical lower extremity length (16). Although there seems to be a higher prevalence of radiographic hip OA and symptoms among individuals with limb length inequality, the association is weak. Moreover, having a limb length inequality is not predictive of radiographic or symptom progression. Femoral acetabular impingement is an established risk factor for early hip OA and joint replacement (5,30,32,34,42,44). Mechanisms for impingement of the femoral head-neck on the acetabulum include the CAM variant (nonspherical femoral head), PINCER variant which is essentially acetabular overcoverage, and a mixed CAM-PINCER variant. The CAM-type morphology occurs more frequently in younger patients and is thought to cause a delaminating injury to the cartilage of the acetabulum (e.g., cartilage is sheared off bone). The PINCER variant lends to labral impingement, which results in labral tears, degeneration, and ossification. Readers desiring more information on femoral acetabular impingement are encouraged to review Kolber et al. (27), in the June 2015 issue of Strength and Conditioning Journal.

### Practical Applications
Strength and conditioning professionals serve a key role in exercise and lifestyle prescription for individuals with varying fitness levels (Table 3). Thus, an understanding of risk factors would seemingly serve useful given the prevalence of hip OA among older adults and the potential for strength and conditioning specialists to find themselves working with this population. Although certain risk factors such as age and previous surgery or developmental changes cannot be prevented, modifications may be implemented to help decelerate the progression of OA. Furthermore, an understanding of modifiable risk factors provides an opportunity for exercise and lifestyle modifications including but not limited to loading control, body mass management, promotion of routine moderate-intensity exercise,

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<tr>
<td>Practical recommendations that may reduce risk for hip osteoarthritis</td>
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<th>Practical recommendations</th>
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<tr>
<td>Discourage a sedentary lifestyle through promotion of physical activity</td>
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<tr>
<td>Maintain a healthy body mass and seek dietary counseling as necessary</td>
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<tr>
<td>Compulsory stair climbing, deep squatting, and prolonged standing should be discouraged among higher risk cohorts*</td>
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*For example, age \( > 50 \), body mass index \( \geq 30 \), previous hip injury, or developmental hip disorder.
and avoidance of provocative positioning. Finally, an understanding of relevant risk factors and the evidence underscoring their association lends to evidence-based advice and may perhaps prevent erroneous recommendations to discontinue otherwise safe activities. Readers interested in a comprehensive approach to the evaluation and management of hip OA are referred to chapter 6 in the text Orthopedic Management of Hip and Pelvis (7).

In most individuals with a diagnosis of OA, there is a degree of pain associated with both the condition itself and certain provocative activities or movements. Numerous risk factors have been associated with hip OA and include but are not limited to age, sex, obesity, trauma, genetics, occupational hazards, congenital factors, and previous surgical interventions. Early identification of modifiable risk factors may serve the basis for preventative programs aimed at decelerating disease progression and associated symptoms. At minimum, habitual-safe physical conditioning (25), efforts to prevent obesity, and modification of occupational hazards are likely to have a positive effect on the presence of pain and physical function. Although age and developmental disorders such as dysplasia are not modifiable risk factors, improving the biomechanical environment by avoidance of aberrant loading while maintaining a reasonable level of physical activity may reduce one’s risk for developing OA. Readers should recognize that although certain activities such as running are not considered an independent risk factor for OA, participation among those with a formal diagnosis may accelerate the condition. Formal clearance from a medical doctor is recommended before commencing in any exercise program once a diagnosis has been established.

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