Although scientists are only starting to understand its contribution, numerous companies are marketing direct-to-consumer genetic tests for common injuries, including ACL ruptures. These tests are, however, premature since the genetic data is incomplete and not interpreted together with clinical indicators and lifestyle factors to identify an altered risk for injury by an appropriately qualified healthcare professional. Although there is no specific genetic test, clinicians are currently in a position to consider if a patient is potentially predisposed to ACL ruptures. A positive family history of ACL ruptures, a history of other tendon and/or ligament ruptures, and participation in sports associated with ACL ruptures need to be considered. Personalized prehabilitation exercises to reduce the risk of ACL ruptures could be prescribed.

CONCLUSION

ACL rupture risk is most likely underpinned by a complex interaction of a number of biological and nonbiological factors. There is evidence suggesting that there is in part a genetic contribution to an individual's risk of sustaining an ACL rupture. To date, several gene variants (polymorphisms) at known genetic loci have been implicated in risk. These associated variants may contribute to the interindividual variation in the structure, and by implication mechanical properties, of the ACL and surrounding tissues, as well as its responses to mechanical loading and other stimuli. Furthermore, many of the anatomical, structural, and other risk factors are also in their own right multifactorial phenotypes determined, to a lesser or greater extent, by both genetic and environmental factors.[28] It is therefore unlikely, based on the current evidence, that the identified genetic variants are independent risk factors, but rather modulate risk through their complex interactions with other genetic and nongenetic factors.

SELECTED READINGS

Collins M, Posthumus M, Schwellnus MP. The *COL1A1* gene and acute soft tissue ruptures. *Br J Sports Med*. 2010;44:1063–1064.

El Khoury L, Posthumus M, Collins M, et al. *ELN* and *FBN2* gene variants as risk factors for two sports-related musculoskeletal injuries. *Int J Sports Med*. 2015;36:333–337.

Ficek K, Stepien-Slodkowska M, Kaczmarczyk M, et al. Does the A9285G polymorphism in collagen type XII α1 gene associate with the risk of anterior cruciate ligament ruptures? *Balkan J Med Genet*. 2014;17:41–46.

Mannion S, Mtintsilana A, Posthumus M, et al. Genes encoding proteoglycans are associated with the risk of anterior cruciate ligament ruptures. *Br J Sports Med*. 2014;48:1640–1646.

O'Connell K, Knight H, Ficek K, et al. Interactions between collagen gene variants and risk of anterior cruciate ligament rupture. *Eur J Sport Sci*. 2015;15:341–350.

O'Connell K, Saunders CJ, Collins M. Collagen gene sequence variants in exercise-related traits. *Cent Eur J Sport Sci Med*. 2013;1:3–17.

Posthumus M, Collins M, van der Merwe L, et al. Matrix metalloproteinase genes on chromosome 11q22 and the risk of anterior cruciate ligament (ACL) rupture. *Scand J Med Sci Sports*. 2012;22:523–533.

Posthumus M, September AV, O'Cuinneagain D, van der Merwe W, Schwellnus MP, Collins M. The association between the *COL12A1* gene and anterior cruciate ligament ruptures. *Br J Sports Med*. 2010;44:1160–1165.

Posthumus M, September AV, O'Cuinneagain D, van der Merwe W, Schwellnus MP, Collins M. The *COL5A1* gene is associated with increased risk of anterior cruciate ligament ruptures in female participants. *Am J Sports Med*. 2009;37:2234–2240.

Rahim M, Gibbon A, Hobbs H, et al. The association of genes involved in the angiogenesis-associated signaling pathway with risk of anterior cruciate ligament rupture. *J Orthop Res*. 2014;32:1612–1618.

Stępień-Słodkowska M, Ficek K, Maciejewska-Karłowska A, et al. Overrepresentation of the *COL3A1* AA genotype in Polish skiers with anterior cruciate ligament injury. *Biol Sport*. 2015;32:143–147.

Stępien-Słodkowska M, Ficek K, Eider J, et al. The +1245G/T polymorphisms in the collagen type I alpha 1 (*COL1A1*) gene in polish skiers with anterior cruciate ligament injury. *Biol Sport*. 2013;30:57–60.

A complete reference list can be found online at ExpertConsult.com.

3 Association Between Anterior Cruciate Ligament Tear and Femoroacetabular Impingement

Marc J. Philippon, MD, Jorge Chahla, MD, Karen Briggs, MPH

INTRODUCTION

In the United States, approximately 100,000 anterior cruciate ligament (ACL) injuries per year are reported, resulting in an expense of billions of dollars for the health system.[1] Therefore prevention and identification of risk factors for ACL tears is fundamental. Several ACL injury-prevention programs have been developed.[2–10] Many of these programs focus on males or females[5] and specific sports, such as handball, soccer,[2,4] basketball,[3] volleyball,[6] and other sports. While these prevention programs have been widely studied, a recent meta-analysis showed that programs for soccer players did reduce injury risk for the knee, but they did not find a statistically reduction in ACL injuries.[4] Recent studies have begun to look at how the hip may contribute to risk of ACL injuries.[11–21]

FEMOROACETABULAR IMPINGEMENT

In the past 20 years, the understanding of hip pathology had grown. Ganz et al. described femoroacetabular impingement (FAI) as a cause of hip pain, chondrolabral damage, and osteoarthritis of the hip.[22,23] Two types of FAI have been described. Cam impingement results from abnormal femoral head-neck offset, and excessive coverage of the acetabular rim causes pincer impingement. Pincer type impingement is due to abnormal bony features of the acetabulum (Fig. 3.1). This can result in either global overcoverage of the acetabulum with coxa profunda or protrusio acetabuli, or focal overcoverage with acetabular retroversion. This overcoverage results in impingement between the bone of the acetabular rim and the femoral head-neck junction. This results in crushing of the labrum, resulting in a bruised labrum and damage to chondrolabral

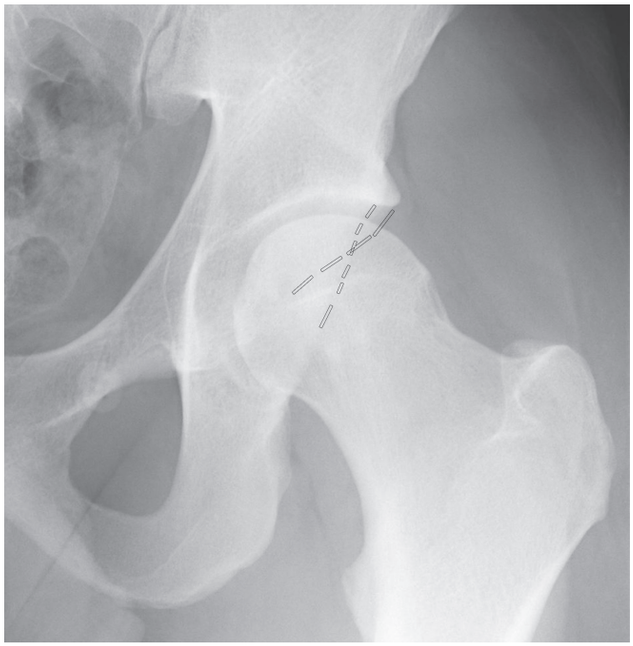

Fig. 3.1. Pincer impingement as shown by a crossover sign on antero-posterior radiograph.

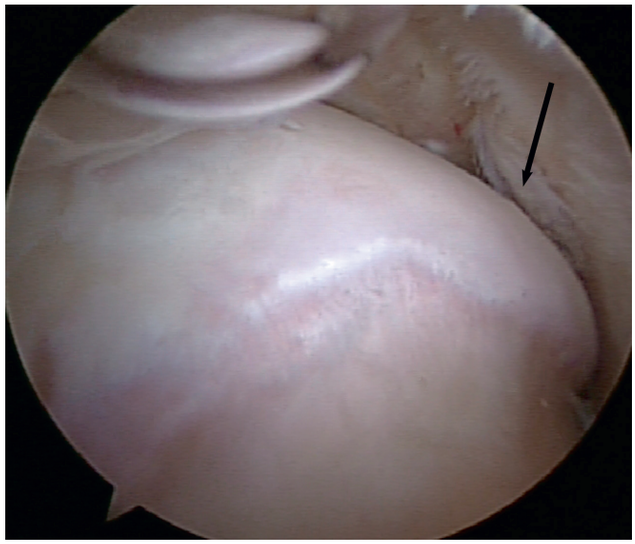

Fig. 3.2. Bony abnormality on the femoral head-neck junction as viewed from peripheral compartment during hip arthroscopy. *Arrow* shows bony abnormality on femoral neck impinging on other tissue.

junction.[24] Cam type impingement results from abnormal bone on the femoral head-neck junction (Fig. 3.2). The aspherical bone at the head-neck impinges with the acetabulum during motion. The resulting shear forces cause damage to the acetabular cartilage and labrum.[24] Often the labrum will detach and the cartilage will peel away from the subchondral bone. Cam impingement is commonly diagnosed using the alpha angle measurement on magnetic resonance imaging (MRI).[25] Alpha angle measures the point where the femoral head loses it sphericity. A line from the center of the femoral heal is extended to the midpoint of the femoral neck, and a second line is extended from the center head to the point where bone on the femoral neck goes outside the circle drawn around the femoral head (Fig. 3.3).[26] A combination of combined cam and pincer impinge is seen most often; however, isolated cases of cam and pincer do exist.

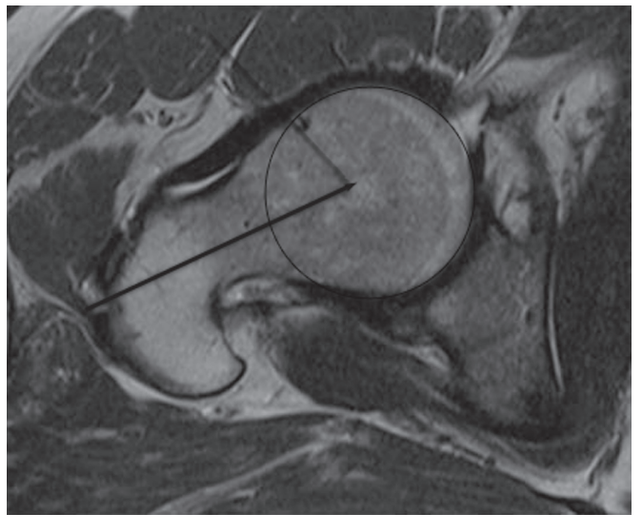

Fig. 3.3. Measurement of the alpha angle on MRI with a circle around the femoral head with a line drawn to the center of the femoral head and a line drawn where the bone leaves the circle.

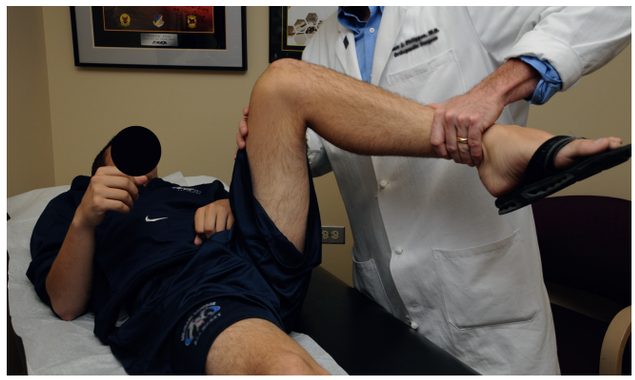

Fig. 3.4. The anterior impingement test is performed in the supine position with the hip flexed to 90 degrees, with forced adduction and internal rotation.

Patients with FAI present with pain, decreased function, and physical limitations.[25] In addition to radiographic findings, several specific tests are performed to assist with diagnosis. The anterior impingement test is very specific for FAI (Fig. 3.4). This test is based on pain so it is less reliable in the asymptomatic patient.[25] The Flexion, Abduction and External Rotation (FABER) distance test measures hip motion with the knee flexed and the foot placed above the patella of the other knee. The distance from the table to the knee is compared between each leg. A difference in distance between legs greater than 3 mm is considered a positive test for cam impingement in patients with unilateral FAI (Fig. 3.5).[25] Both of these tests, in addition to loss of internal hip rotation, can diagnose hips with FAI.

With the intra-articular damage caused by FAI, this is most commonly seen in patients with hip and/or groin pain. However, many studies have shown that these bony abnormalities associated with FAI can be seen in asymptomatic individuals.[27–31] A recent systematic review identified 26 studies that documented findings in asymptomatic individuals.[29] The prevalence of cam impingement ranged from 7% to 100%, and the prevalence of pincer impingement was 61% to 76%.[29] The prevalence of pincer may be difficult to determine, given the different definitions for diagnosis. Several studies have shown that the pincer prevalence differs based on which definition is used;[27] however, cam

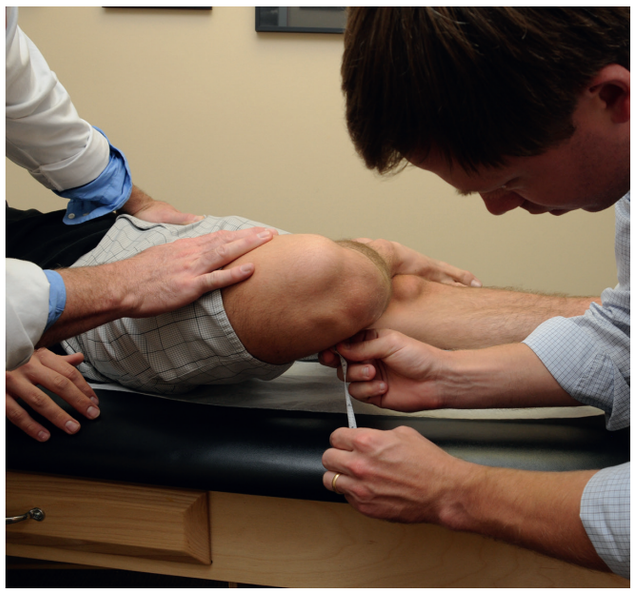

Fig. 3.5. The FABER distance test is performed in the supine position with the leg in a figure-four position of flexion, abduction, and external rotation.

impingement is commonly seen in asymptomatic hips.[30,31] With the high prevalence of asymptomatic FAI, it is possible that FAI is present but not recognized in individuals who suffer from ACL injuries.

ANTERIOR CRUCIATE LIGAMENT INJURIES

The mechanism for ACL injuries is still misunderstood, and research continues to determine critical factors for prevention.[32–38] The mechanism for ACL injuries can be direct (trauma or physical contact) or indirect. Commonly, ACL tears occur with an abrupt landing, associated with a rapid change of direction with the knee in a semiflexed position with valgus or varus stress and internal or external rotation.[18,33] In the vast majority of cases, this results from an indirect mechanism.

External and internal risk factors of ACL lesions have been identified. Most of the studies have focused on the intrinsic factors of the knee. Reported intrinsic factors include impingement on the intercondylar notch,[37] aggressive quadriceps contraction,[35,36] the quadriceps-hamstring force balance, and axial compressive forces on the lateral aspect of the joint. Female athletes are also at an increased risk of injury. Potential explanations for this include increased knee valgus or abduction moments, generalized joint laxity,[36] knee recurvatum,[1] a comparatively smaller ACL,[34] and the hormonal effects of estrogen on the ACL.[34]

FEMOROACETABULAR IMPINGEMENT: ANTERIOR CRUCIATE LIGAMENT INJURY

The mechanics of the hip may contribute to risk of ACL injury.[11] Both femoral (decreased femoral head-neck offset) and acetabular bone deformities could place the ACL at risk.[19,21] Yamazaki et al.[21] reported that the center edge angle of the ACL-injured patients group was significantly smaller than that of the uninjured control group. Therefore ACL-injured patients might have a high prevalence of acetabular dysplasia. In a study of soccer players with noncontact ACL injuries, 56% of the players had abnormal radiographic findings in the hip.[15] The senior author reported that patients with a decreased femoral head-neck offset (alpha angle greater than 60 degrees) were at increased risk of having an ACL injury.[19] Furthermore, these increased odds were seen in both males and females; however, the odds were higher in males. ACL injury cohort had a mean alpha angle of 86 degrees and 79 degrees in males and females, respectively, which are markedly higher than previously reported limits of normal[31] and would be considered a positive diagnosis of FAI.[19] Beaulieu performed a simulated single-leg pivot landing study to assess for the peak relative strain of the anteromedial bundle of the ACL in relation to the available range of internal femoral rotation.[11] In their statistical model, peak ACL relative strain increased by 1.3% with every 10-degree decrease in femoral rotation with a sudden stop. From their data, they extrapolated that an athlete presenting with FAI with a 10-degree deficiency in internal femoral rotation would experience 20% more peak ACL strain during landing than a healthy athlete.[11]

Available literature supports a strong association between decreased hip motion in patients with ACL injuries—predominantly loss of internal rotation of the hip.[12–14,16–18,20] This suggests that ACL injury has not only an intrinsic knee etiology but also an adjacent joint origin. In a group of 44 ACL injuries aged 13–17, limited hip rotation was identified as a possible factor related to the injury.[20] Specifically, when total hip rotation is less than 80 degrees, the risk of ACL injury is extremely high. When rotation is between 80 degrees and 100 degrees, the risk is moderate, and in patients with more than 100 degrees of rotation, the risk of rupture is highly unlikely.[20] Benjaminse et al. studied videos of noncontact ACL injuries in professional basketball players.[3] The study showed that on initial landing, hip flexion was greater in the injured knee compared with the control. In women basketball players, they found greater hip flexion through the entire landing sequence compared with controls. No differences were found in abduction. Although many predisposing factors have been identified, such as age, sex, anthropometric measures, psychological, and anatomical factors, the relationship of mobility of the hip (which may acts as a *buffer* in forced external rotation of the knee) in patients with ACL injury requires further study.

Decreased hip range of motion is commonly found in patients with FAI, similar to that seen in individuals who suffer ACL injury. In a study of 50 soccer players who sustained ACL injury, there was a strong association between hip range of motion, specifically internal rotation, and the ACL injury.[17] This was also seen in a group of professional football players. Bedi et al. found that decreased internal rotation was associated with increased odds of ACL injury.[13] They calculated 4–5 times greater odds of ACL injury with a 30-degree reduction in hip internal rotation.

Patients with abnormally elevated alpha angles, or cam impingement, may have diminished capacity at the hip to accommodate overall lower extremity internal rotation moments, potentially predisposing the knee and the ACL to a greater rotational stress. Girard et al. suggested that improving the femoral head-neck offset could result in an improved range of motion in the hip, specifically flexion.[39] While correction of the head-neck offset in the asymptomatic hip has not been advocated, labral tears are commonly seen in asymptomatic hips with large alpha angles. In a study of hockey players, ages 16–19 with an average alpha angle of 65 degrees, 93% had labral tears.[30] It is unclear if and when these may become symptomatic. In addition, the longer individuals played sports, the higher the alpha angle became.[30] More research is needed to determine the timeline of the development of the cam impingement, the labral tear, and the loss of motion in the hip.

A screening program has been developed to identify the individual with the hip *at risk* for FAI.[30] This screening has been implemented over 5 years in a youth hockey program. The goal of the program is to identify factors associated with increased risk of hip injury and provide programs to address the risk. Individuals are screened prior to beginning their sport. Players undergo a physical examination. The exam consists of

a FABER distance test that was described earlier in this chapter. This is followed by an anterior impingement test, posterior impingement test, hip dial test, and a Trendelenburg gait test. Range of motion is measured with a goniometer, including flexion, abduction, and adduction in supine position and extension, internal rotation, and external rotation in prone position. The screening program was validated by performing 3T MRIs to determine alpha angle and confirm the presence of cam impingement. Individuals with positive exams had increased alpha angles on MRI. For screening of young individuals, functional, passive internal rotation of the hip is a safe and inexpensive intervention[30] that could help athletes at risk of ACL injury, and programs could be developed to prevent lesions through neuromuscular and proprioceptive training.

The Prevent Injury and Enhance Performance program introduced by Mandelbaum et al.[5] emphasizes the importance of having a proper landing technique; engaging knee and hip flexion on landing and lateral maneuvers; avoiding excessive genu valgum at the knee on landing and squatting; increasing hamstring, gluteus medius, and hip abductor strength; and addressing proper deceleration techniques. In a cohort study, they demonstrated an 88% and 74% decrease in ACL injury in the intervention group subjects compared with the control group in the first and second year, respectively.[5]

In a recent study, Swart et al. compared the effectiveness and cost-effectiveness of two approaches to lower the risk of ACL injury in young athletes: training everyone or training high-risk athletes identified by screening.[10] They reported that the universal neuromuscular training strategy was cost-effective in virtually all situations, as ACL injury is common and often associated with a large cost, while neuromuscular training programs have a relatively low cost coupled with a large demonstrable risk reduction.[10]

In a systematic review, the authors concluded that there is moderate evidence to support the use of multifaceted training interventions, consisting of stretching, proprioception, strength, plyometric, and agility drills with additional verbal and visual feedback on proper landing technique to decrease the rate of ACL injuries.[9]

CONCLUSION

The success of ACL injury-prevention programs may be improved with screening for FAI and loss of hip motion. Programs can be developed to address loss of hip motion while allowing individuals to adapt their activities to deal with the bony abnormalities of FAI.

SELECTED READINGS

Frank JM, Harris JD, Erickson BJ, et al. Prevalence of femoroacetabular impingement imaging findings in asymptomatic volunteers: a systematic review. *Arthroscopy*. 2015;31(6):1199–1204.

Ganz R, Parvizi J, Beck M, et al. Femoroacetabular impingement: a cause for osteoarthritis of the hip. *Clin Orthop Relat Res*. 2003;417:112–120.

Nötzli HP, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J. The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. *J Bone Joint Surg Br*. 2002;84:556–560.

Philippon M, Dewing C, Briggs K, Steadman JR. Decreased femoral head–neck offset: a possible risk factor for ACL injury. *Knee Surg Sports Traumatol Arthrosc*. 2012;20(12):2585–2589.

Philippon MJ, Ho CP, Briggs KK, Stull J, LaPrade RF. Prevalence of increased alpha angles as a measure of cam-type femoroacetabular impingement in youth ice hockey players. *Am J Sports Med*. 2013;41(6):1357–1362.

Philippon MJ, Maxwell RB, Johnston TL, Schenker M, Briggs KK. Clinical presentation of femoroacetabular impingement. *Knee Surg Sports Traumatol Arthrosc*. 2007;15:1041–1047.

Stojanovic MD, Ostojic SM. Preventing ACL injuries in team-sport athletes: a systematic review of training interventions. *Res Sports Med*. 2012;20(3–4):223–238.

Swart E, Redler L, Fabricant PD, Mandelbaum BR, Ahmad CS, Wang YC. Prevention and screening programs for anterior cruciate ligament injuries in young athletes: a cost-effectiveness analysis. *J Bone Joint Surg Am*. 2014;96(9):705–711.

A complete reference list can be found online at ExpertConsult.com.