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Does Limited Internal Femoral Rotation Increase Peak Anterior Cruciate Ligament Strain During a Simulated Pivot Landing?

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Abstract

Background: Many factors contributing to anterior cruciate ligament (ACL) injury risk have been investigated. Recently, some ACL-injured individuals have presented with a decreased range of hip internal rotation compared with controls. The pathomechanics of why decreased hip range of motion increases risk of ACL injury have not yet been studied.

Purpose: To test the hypothesis that peak relative strain of the anteromedial bundle of the ACL (AM-ACL) during a simulated single-leg pivot landing is inversely related to the available range of internal femoral rotation.

Study Design: Controlled laboratory study.

Methods: A series of pivot landings were simulated in 10 female and 10 male human knee specimens with a testing apparatus that applied a 2-bodyweight impulsive load, inducing knee compression, flexion moment, and internal tibial torque. The range of internal femoral rotation was (1) locked at ~0°, (2) limited with a hard stop to ~7°, (3) limited with a hard stop to ~11°, or (4) free, with rotation resisted by 2 springs to simulate the resistance of the active hip rotator muscles to stretch. The AM-ACL strain was quantified with a differential variable reluctance transducer. A linear mixed model was used to determine whether a significant linear relation existed between peak AM-ACL relative strain and range of internal femoral rotation.

Results: Peak AM-ACL relative strain was inversely related to the available range of internal femoral rotation ($R^2 = 0.91$; $P < .001$), with strain increasing 1.3% for every 10° decrease in rotation; this represented a 20% increase in peak relative strain, given an average range of femoral rotation of 15° upon landing in healthy athletes.

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Conclusion: Peak AM-ACL relative strain was inversely proportional to the available range of internal femoral rotation during simulated single-leg pivot landings.

Clinical Relevance: Decreased range of internal femoral rotation results in greater ACL strain and may therefore increase the susceptibility to ACL rupture with athletic cutting and pivoting activities. Screening for a limited range of hip internal rotation should therefore become a component of not only ACL injury prevention programs but also evaluation protocols for those with ACL injuries and/or reconstructions.

Keywords

anterior cruciate ligament; strain; knee; hip; femoroacetabular impingement

Injuries to the anterior cruciate ligament (ACL) continue to occur at significant rates,^{21,39} especially in younger females.⁴¹ Given that knee osteoarthritis is an expensive sequela of ACL injuries,^{29,30,33} in terms of both financial and health costs, insights are needed to better prevent ACL injuries.

While many factors contributing to injury risk have been investigated,³⁸ attention has focused on the knee joint.^{15,37,40,46} The mechanics of the hip, however, may also contribute to injury risk. For example, a restricted passive range of internal rotation at the hip, mostly associated with abnormal proximal femoral or acetabular anatomy,⁷ has been correlated with ACL ruptures and reruptures in soccer players.^{6,8} *Restricted hip internal rotation* was defined as being from $<30^{\circ}$ to 35° , with ACL-injured and ACL-reinjured soccer players having an average range of 26° and 18° , respectively, compared with 39° in the control group.^{6,8} A similar restriction in range of hip internal rotation has been identified as a contributing factor to ACL injury risk in professional American football athletes.² Specifically, players at the 2012 National Football League Combine with a restricted range of internal rotation at the hip were more likely to have had sustained an ACL injury that required surgical reconstruction.² Also, as compared to uninjured controls, a group of ACL-injured individuals were found to have a larger cam-type femoroacetabular deformity of the femoral head, as measured by the alpha angle,³¹ as well as a greater prevalence of acetabular dysplasia.⁴⁷ Abnormal hip anatomy therefore may play a critical role in increasing ACL injury risk given its prevalence in healthy and pathologic populations. For example, femoroacetabular impingement (FAI) is present in 6% to 24% of asymptomatic individuals,^{10,14,16,23,32} in addition to 63% of patients with an intra-articular hip disorders.¹

The underlying mechanism for an increased risk of ACL injury with restricted terminal hip range of motion, however, remains unexplored. We theorized that limiting the available range of hip internal rotation will cause an increase in peak ACL strain during athletic maneuvers via compensatory axial tibial rotation at the knee joint.²⁷ If hip internal rotation is limited by a bony impingement between the acetabular rim and the femoral head-neck junction, then rotation at an adjacent joint may have to increase to achieve the desired athletic outcome. Hence, the purpose of this study was to quantify the effect of limiting the range of internal femoral rotation on peak ACL strain during a simulated single-leg pivot landing. We also examined whether sex modulated this peak strain.

We used a custom-built in vitro knee testing apparatus^{17,18,26–28,42–44} to simulate a single-leg pivot landing under a 2-bodyweight (2×BW) impulsive load. To titrate the available range of internal femoral rotation, a new femoral rotation device was added to the apparatus whereby the experimenter could set the range of available internal femoral rotation to restrict axial hip motion. Such a restriction can occur because of bony contact in hips with abnormal anatomy, such as FAI, as well as axial hip motion resisted by pretensed muscles. We used the apparatus to test the primary hypothesis that peak strain in the anteromedial bundle of the ACL (AM-ACL) would be inversely related to the available range of internal femoral rotation during the landing. We also tested the secondary hypothesis that female knee specimens would exhibit greater peak AM-ACL strain, in comparison with male knee specimens, regardless of the range of internal femoral rotation.

MATERIALS AND METHODS

Specimen Procurement and Preparation

To determine the sample size needed to reveal statistically significant differences in peak AM-ACL strain among femoral rotation conditions as well as between sexes, 2-sided paired and unpaired 2-sample *t*-test models ($\alpha = 0.05$; $1-\beta = 0.80$)³⁴ were applied to pilot data. These a priori power analyses revealed required total sample sizes between 6 and 18 knee specimens. A total of 20 unembalmed knee specimens (10 female, 10 male specimens) were therefore harvested from 6 female and 6 male human donors (Table 1) acquired from the University of Michigan Anatomical Donations Program, Anatomy Gifts Registry and Research for Life. All knee specimens were free of scars indicative of knee surgery, free of evidence of joint degeneration, and free of joint deformity. Knee specimens were stored in a freezer at -20°C , and each specimen was thawed at room temperature 48 hours before dissection. The specimens were dissected until only the joint capsule remained, including the ligaments and the tendons of the quadriceps, hamstrings (medial and lateral), and gastrocnemii (medial and lateral). The dissected knee was stored in a freezer (-20°C) until testing. Each specimen was removed from the freezer and thawed at room temperature 24 hours before testing. Immediately before testing, the femur and tibia were cut 20 cm proximal and distal of the joint line, respectively, to standardize the length of the specimen. Then, each bone extremity was potted in polymethylmethacrylate as previously described.¹⁸

Experimental Design and Protocol

A cross-sectional repeated-measures design (block order, A-B-C-D-E-A) (Table 2) was used to test the hypotheses. Each testing session began with 5 trials for which only an impulsive compression force and knee flexion moment were applied (ie, no axial tibial torque) to precondition the knee specimen. After these preconditioning trials, the repeated-measures design, which consisted of 6 blocks of 6 trials, was executed. The first trial of each block served to precondition the knee, while the subsequent 5 trials were used for analysis. In block B, the femoral rotation device was locked, allowing minimal axial femoral rotation. In blocks C and D, the range of internal femoral rotation was limited by a hard stop to $\sim 7^{\circ}$ and $\sim 11^{\circ}$, respectively. In block E, internal femoral rotation was not limited and achieved $\sim 15^{\circ}$. A block of nonpivot trials (block A) was included both before and after the main testing sequence (blocks B-C-D-E) (Table 2), the data from which were used to ensure that the

integrity of the knee specimens was not compromised during the testing protocol. The sequence of the main testing blocks (B-C-D-E) was randomized.

Knee Testing Apparatus

The single-leg pivot landings were simulated with a modified Withrow-Oh apparatus,²⁶ which impacted the distal end of the tibia of an inverted knee specimen (Figure 1). Specifically, a weight (Figure 1, *W*) was dropped onto the distal end of the knee specimen in 15° of flexion from a height that would simulate an impulsive ground-reaction force of 2×BW (±10%). This height was determined by trial and error during the preconditioning trials. The impact of the weight on the distal end of the knee specimen induced an impulsive compression force and knee flexion moment, with and without internal tibial torque (pivot and nonpivot trials, respectively), that were measured by the proximal and distal 6-axis load cells (Figure 1, *L*). Internal tibial torque was developed by means of a tibial torsion device (Figure 1, *T*), which could be locked (without tibial torque) or unlocked (with tibial torque). When the tibial torsion device was unlocked, the apparatus applied a compressive force, flexion moment, and axial torque to the knee via the distal tibia. A novel addition to the apparatus was a proximal femoral rotation device (Figure 1, *R*) able to limit the range of axial femoral rotation. This device comprised a circular plate that rotated in the transverse plane on a tapered-roller bearing. Two pretensioned springs were attached tangentially to the perimeter of the plate via aircraft cables to represent the tensile resistance of active external hip rotator muscles to rapid stretch during a pivot landing. To limit internal femoral rotation, a steel stop was inserted into a hole on the femoral rotation device to lock it (Table 2, block B) or abruptly halt rotation after ~7° (Table 2, block C) or ~11° (Table 2, block D) of internal femoral rotation. The goal of the stop was to mimic the bone-on-bone restriction in terminal hip motion secondary to FAI. The stop was removed from the device for the trials in block E (Table 2), during which femoral rotation was resisted by only the springs of the femoral device to represent the resistance of active pretensed hip muscles to axial femoral rotation without any bone-on-bone contact; hence, no hard stop was present. To simulate dynamic muscle tension and tensile resistance to stretch during the landing, tendons of the quadriceps (Figure 1, *Q*), medial and lateral hamstrings (Figure 1, *H*), and gastrocnemii (Figure 1, *G*) were attached via cryoclamps to elastic structures made of woven nylon cord. The tendon-muscle unit of the quadriceps was pretensioned to 180 N, while those of the hamstrings and gastrocnemii were pretensioned to 70 N before every trial.²⁶ Individual muscle tensions were measured at 2 kHz with 5 uniaxial load cells (Transducer Techniques) attached, in series, to the woven nylon cord and cryoclamps.

The 3-dimensional (3D) motions of the femur and tibia were quantified via infrared-emitting diodes tracked by an optoelectric imaging system (Optotrak Certus; Northern Digital) at 400 Hz. Three diodes were affixed to the femur segment and to the tibia segment such that they defined the 3 anatomic planes of the knee (sagittal, coronal, and transverse). The 3D coordinates of the diodes were used to calculate 3D angles and translations of the knee joint during each landing trials. Three-dimensional forces and moments produced at the distal tibia and proximal femur were quantified via two 6-axis force sensors (Advanced Manufacturing Technology Inc) (Figure 1, *L*) at 2 kHz. Finally, a 3-mm differential variable reluctance transducer (DVRT; MicroStrain Sensing Systems) was affixed to the distal third

portion of the AM bundle of the ACL to measure ligament elongation. Displacement data were recorded at 2 kHz.

Data Processing

Three-dimensional marker coordinates acquired from the motion capture system were low-pass filtered with a Butterworth filter (4th order, 20-Hz cutoff frequency). From the 3D coordinates of the 6 markers as well as those of the knee's origin (roof of the femoral notch digitized before the landing trials), 3D angles and translations were calculated via the method established by Grood and Suntay.⁹ Femoral rotation was defined as rotation of the femur relative to the testing apparatus, whereas tibial rotation was defined as rotation of the tibia relative to the femur. Data acquired from all load cells, as well as the elongation data acquired from the DVRT, were also low-pass filtered with a Butterworth filter (4th order, 70-Hz cutoff frequency). The AM-ACL relative strain (ϵ) was quantified as $\epsilon = (L - L_0) / L_0 \times 100$, where L_0 is the reference interbarb distance of the DVRT and L is the instantaneous interbarb distance of the DVRT. The reference length (L_0) was defined as the interbarb distance of the DVRT at the beginning of each trial.

From each trial, peak AM-ACL relative strain was extracted as the main dependent variable of interest. Range of internal femoral rotation was set as the independent variable. Last, femoral rotational stiffness, range of anterior tibial translation, range of internal tibial rotation, peak internal tibial deceleration, and difference between time-to-peak internal tibial and femoral rotations were obtained to gain further insight into differences in peak AM-ACL relative strain between femoral rotation conditions. A positive value for difference between time-to-peak rotations indicated that peak internal femoral rotation occurred before peak internal tibial rotation, whereas a negative value indicated that peak internal tibial rotation occurred first.

Statistical Analysis

The hypotheses were tested with a linear mixed model, with *range of internal femoral rotation*, *sex of donor*, and *age* treated as fixed effects and *knee specimen* and *knee donor* as random effects. Age was included in the model to account for differences in this variable between the male and female donors given that the tensile properties of the ACL (eg, linear stiffness) have been reported to decrease significantly with age.⁴⁵ Knee donor was included in the model to account for the correlation between paired specimens. The model determined whether a significant inverse (linear) relation existed between peak AM-ACL relative strain and range of internal femoral rotation. The proportion of variance explained by the full model, R^2 , was quantified with a method recommended by Nakagawa and Schielzeth.²⁴ The model also compared peak AM-ACL relative strain between the female and male knee specimens. An alpha level above 0.05 indicated statistical significance.

Lower Limb Computational Model

To help interpret our findings from a biomechanical perspective, we developed a simple computer simulation of an axial impulsive torque applied distally to a lower extremity having segmental inertias (see the Appendix, available online at <http://ajsm.sagepub.com/supplemental>). Briefly, the lower limb, consisting of a foot, tibia, and femur, was represented

by 3 rigid bodies connected by torsional springs to represent the axial rotational stiffnesses of the ankle, knee, and hip passive structures and active muscles. To represent the transverse-plane mechanics of a pivot landing, an axial impulsive torque of 10 N·m was applied in the transverse plane, over 80 milliseconds, orthogonal to the longitudinal axis of the foot to create foot angular momentum (see Results). The stiffness of the spring representing the hip was then systematically increased from 0.9 to 9.4 N·m/deg in separate trials to cause a systematic decrease in femoral rotation. For each trial, the magnitude and timing of peak intersegmental rotations were then calculated over the first 200 milliseconds. Tibial rotation, relative to the femur, in the transverse plane was then calculated as an outcome measure.

RESULTS

The temporal behavior of the variables from a representative trial for each axial femoral rotation condition is presented in Figure 2. For the axial femoral rotation conditions where rotation was abruptly limited to $\sim 7^\circ$ and $\sim 11^\circ$, the slope of the axial femoral torque curves revealed that the femoral rotation device adequately modeled a hard stop with a bilinear stiffness response (Figure 2, B-1 and C-1). Specifically, there was low rotational stiffness within the available range of motion, with a sudden increase in stiffness at the limit of motion (ie, when the femoral rotation device hit the stop). In contrast, the conditions during which axial femoral rotation was either locked or free, a linear stiffness response was observed, with the free rotation condition presenting with a more compliant response (Figure 2, A-1 and D-1).

Peak AM-ACL relative strain was inversely proportional to internal femoral rotation during the simulated single-leg pivot landings ($P < .001$) (Figure 3). Peak AM-ACL relative strain was generally largest when the range of internal femoral rotation was abruptly arrested after $\sim 7^\circ$ and 28.4% larger when femoral rotation was locked than when it was free. Furthermore, strong positive relations were also found between peak ACL relative strain and femoral rotational stiffness ($P < .001$) (Figure 4A), range of anterior tibial translation ($P < .001$) (Figure 4B), range of internal tibial rotation ($P < .001$) (Figure 4C), peak internal tibial deceleration ($P < .001$) (Figure 4D), and the difference between time-to-peak internal tibial and femoral rotations ($P < .001$) (Figure 4E). Peak ACL relative strain increased as each of these variables increased with decreasing femoral internal rotation.

The transverse-plane mechanics of the lower limb computational model revealed that as femoral rotation decreased, tibial rotation and torque (relative to the femur) increased (Figure 5C). The model also showed an increase in the difference in time-to-peak tibial rotation (relative to the femur) and time-to-peak femoral rotation with decreasing hip resistance (k_{Hip}), as found in the in vitro data (Figure 5D).

The female ACL experienced greater peak strain, in comparison to the male ACL, during the pivot landings. On average, peak AM-ACL relative strain was 45% larger in the female knee specimens than the male specimens, regardless of axial femoral rotation condition ($8.69\% \pm 3.46\%$ vs $6.00\% \pm 3.35\%$; $P = .003$) (Figure 3). However, restricting femoral range of rotation did not appear to differentially affect the female knee specimens more than the male knee specimens (Figure 3).

Last, no differences in peak AM-ACL relative strain were found between the 2 nonpivot blocks of trials (Table 2, block A), which occurred before and after the experimental blocks (before: $3.78 \pm 2.03\%$ vs after: $3.52 \pm 1.90\%$; $P = .106$). That result confirmed that the integrity of the knee specimens was not compromised during the testing protocol.

DISCUSSION

This study presents a cause-and-effect relation between limiting internal femoral rotation and ACL strain during dynamic 3D knee loading. The literature reveals a relation between range of passive axial hip motion and ACL injury risk.^{2,6} It is not clear, however, whether a lack of range of hip motion leads to an increase risk of ACL injury or whether an ACL injury leads to a decrease in range of hip motion.⁶ Furthermore, previous work did not show, or even speculate, how decreased hip internal rotation may increase ACL injury risk. Our results suggest that when the range of hip rotation is abruptly limited by a hard stop (modeling FAI), peak ACL strain increases and thus ACL injury risk during athletic maneuvers such as single-leg pivot landings.

Our primary hypothesis—that peak AM-ACL relative strain would increase as the range of internal femoral rotation decreased—was supported. According to our statistical model, peak ACL relative strain increased 1.3% with every 10° decrease in femoral rotation with a hard stop. Given an average range of internal femoral rotation of 15° upon landing,¹¹ this amounts to a 20% relative increase in peak ACL strain. For example, an athlete presenting with FAI with a 10° deficiency in internal femoral rotation would systematically experience 20% more peak ACL strain during a landing than a healthy athlete.

Because we limited the range of internal femoral rotation by adding a hard stop to our in vitro landing to simulate the most commonly observed scenario of FAI or femoral retroversion, we believe that it is the result of the increase in femoral rotation stiffness (via the stop, which abruptly decreased axial femoral rotation), the increase in peak internal tibial deceleration, and the increase in the timing difference between peak internal tibial and femoral rotations that caused the increase in peak ACL strain. As illustrated in Figure 4A, peak ACL relative strain increased as femoral rotational stiffness increased. Also, abruptly arresting internal femoral rotation appears to cause internal femoral rotation to reach its terminal range of motion sooner in relation to internal tibial rotation and peak internal tibial deceleration to increase, thereby increasing internal tibia rotation and coupled anterior tibial translation, and thus peak ACL relative strain (Figure 4, B-E). This explains why peak AM-ACL relative strain was generally largest when the range of internal femoral rotation was abruptly arrested after ~7°, not when it was locked. When femoral rotation was limited to ~7°, peak internal tibial deceleration and the difference in time-to-peak internal tibial and femoral rotations had, on average, the greatest values.

The increase in anterior tibial translation with decreasing internal femoral rotation was most likely due to coupled tibial motion of this translation with axial tibial rotation,^{20,28} as well as an increase in peak quadriceps force. In part because of the geometry of the tibial plateau, including the larger posteriorly directed slope of the lateral plateau in comparison with the medial plateau,¹² the center of axial rotation is located in the knee's medial compartment.

^{22,25} As the tibia rotates internally in relation to the femur under a compressive load and an internal tibial torque, the lateral plateau and geometric center of the plateau translate anteriorly, thus producing both internal tibial rotation and anterior tibial translation. In addition to this coupled tibial motion, the quadriceps force, whose peak increased 148 N, or 12% on average from the free to the locked femoral internal rotation condition, may have played a role in the increase in anterior tibial translation via the patellofemoral reaction force. Given that anterior tibial translation and internal tibial rotation are known to strain the ACL, it is likely that this increase in axial rotation and translation at the knee joint caused the increase in measured peak ACL strain. We acknowledge that, in vivo, various combinations of forces and torques can contribute to an increase in peak ACL strain and to ACL injury and that the mechanism simulated herein represents one sequence of events.

Our interpretation of the in vitro knee mechanics during the pivot landings was supported by the lower limb model simulations (Figure 5 and online Appendix). The 3-rigid body computational model clearly illustrates how decreasing axial femoral rotation, by increasing the torsional spring stiffness representing the hip joint, increased peak tibial rotation in relation to the femoral segment. With a compliant spring, the femur reached the terminal range of motion long after the tibia did in relation to the femur. As spring stiffness was increased, time to terminal range of axial motion of the femur approached that of the tibia, thereby producing greater rotation at the torsional spring representing the knee.

Our secondary hypothesis that the female ACL would exhibit greater peak strain than the male ACL, regardless of the range of internal femoral rotation, was also accepted. Differences in peak ACL relative strain under similar relative loads (ie, %BW) between sexes are likely a contributing factor to the higher ACL injury rate in women.⁴¹ These sex-based differences in ACL strain were likely attributed to sexual dimorphism in ACL size and/or knee joint structure given that the initial knee kinematics and knee loading conditions, including muscle preloads, were not simulated differently for the female and male knee specimens. In a recent study, Lipps et al¹⁷ found the smaller cross-sectional area of the female ACL and the steeper posteriorly directed slope of the female lateral tibial plateau to be mainly responsible for sex differences in peak AM-ACL relative strain in knee specimens subjected to a similar impulsive loading scenario. In addition to its smaller size, the female ACL has a lower strain to failure, stress at failure, and modulus of elasticity,⁵ which are most likely due to ultrastructural differences.¹³ Without accounting for sex differences in neuromuscular control, it appears that the female ACL is at greater risk of injury owing, in part, to greater peak ACL strain than the male ACL.

The 4 conditions of internal femoral rotation were selected to include values ranging from minimal femoral rotation (Table 2, block B) to free femoral rotation (Table 2, block E). The range of internal femoral rotation of this latter condition was based on Hart et al,¹¹ who reported an average of 14.9° of hip internal rotation in female and male collegiate soccer players during a single-leg landing. The values selected for blocks B through D allowed for a relation between range of internal femoral rotation and peak AM-ACL strain to be established without compromising the integrity of the ACL by executing too many loading trials. The values were selected to provide a spectrum of limited ranges of rotation.

Several limitations of the present study are acknowledged. First, knee specimens from older donors were tested. Results therefore cannot necessarily be generalized to younger populations, in which ACL injuries occur most frequently.³⁶ We believe, however, that similar qualitative results would be found in younger specimens, even if the quantitative results would most likely differ owing to changes in structural and mechanical properties of the ACL with age.⁴⁵ Second, relative strain was measured only in the distal third of the AM bundle of the ACL. It was not possible to attach an additional DVRT to the posterolateral bundle without compromising the integrity of the knee joint, especially the posterior joint capsule, or causing measurement error because of contact between the strain transducer and the knee structures. Previous work has demonstrated, however, that strain measured in the AM bundle of the ACL is representative of that in the entire ligament.^{3,4,19} Third, absolute ACL strain could not be measured, but rather relative strain was quantified and compared between conditions. Given that the preloading condition and initial knee specimen position were the same for all internal femoral rotation conditions, we believe that peak AM-ACL relative strain did indeed allow for valid intracondition peak strain comparison. In addition, the sequence of the internal femoral rotation conditions was randomized, which eliminated any effect that previous conditions may have had on the reference interbarb distance of the DVRT. Fourth, an ankle joint was not included in our cadaveric model. The loads induced at the distal tibia by the simulated landing may therefore be overestimated because the ankle joint can absorb energy during the landing.³⁵ In our opinion, the lack of an ankle joint actually strengthens this study because it allows one to minimize confounding variables in the cadaveric model. Fifth, although tension of the major knee muscles and their tensile resistance to stretch during the landing were represented in the in vitro model, only their monoarticular actions were simulated (along with the monoarticular actions of the hip external rotator muscles). Sixth, this work addresses only the effect of limited range of internal femoral rotation because of a sudden stop, as someone with FAI may experience because of bone-on-bone contact. It does not simulate limited rotation because of a volitional increase in muscle tension during the rotation, for example, which may cause a smaller peak internal tibial deceleration as the femur encounters increasing resistance to internal rotation. Last, an interaction term was not included in our statistical model to evaluate whether the effect of the range of femoral internal rotation on peak AM-ACL strain was dependent on sex of the donor. We did not want to decrease the statistical power of our model by including such a term, because this was beyond the scope of this study. Also, data presented in Figure 3 provide no qualitative evidence that such an interaction exists.

There are 2 clinical implications from our study. First, it matters where the femur is in its range of internal femoral rotation when ground contact occurs during a landing or cut maneuver. The closer the femur is to its terminal range of internal rotation, the more likely it is that bone-on-bone contact will occur between the femur and the acetabular rim, thereby decreasing femoral rotation and increasing peak ACL strain. This may offer justification for physical therapy for FAI and rehabilitation to improve the functional range of motion available at the hip even in the absence of surgical correction of the deformity. Second, screening for restricted internal rotation at the hip is advisable for ACL injury prevention programs, as well as for evaluation protocols in individuals with ACL injuries and/or reconstructions. With a simple examination of passive internal hip range of motion before

preseason training, at-risk athletes could be identified and targeted for injury prevention interventions. Future research might translate our in vitro findings in vivo by prospectively confirming limited hip internal rotation as a risk factor for ACL injury.

CONCLUSION

Peak AM-ACL relative strain during in vitro pivot landings was inversely related to the available range of internal femoral rotation.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Sagittal-plane diagram (left) of the in vitro testing apparatus that simulated a single-leg pivot landing, with a top view (right) of the femoral rotation device, *R*. The solid portions represent the starting position of the specimen and device; meanwhile, the transparent portions represent their end position during a trial for which terminal internal femoral rotation was set to ~7° (block C of the testing protocol). *B*, position of steel stop for block B of the repeated-measures protocol (locked); *C*, position of steel stop for block C of the repeated-measures protocol (hard stop at ~7°); *D*, position of steel stop for block D of the

repeated-measures protocol (hard stop at $\sim 11^\circ$); *G*, gastrocnemii tendons; *H*, hamstring tendons; *L*, 6-axis load cell; *Q*, quadriceps tendon; *R*, femoral rotation device; *T*, tibial torsion device; *W*, weight dropped. Note: positions of steel stops are not to scale, to allow better visualization.

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Figure 2.

Sample temporal plots of a representative trial for each axial femoral rotation condition: (A) femoral rotation locked; (B) hard stop at ~7° of rotation; (C) hard stop at ~11° of rotation; and (D) free femoral rotation. Subplots display (1) compressive force (CF), internal femoral torque (IFT), quadriceps force (QF), and knee flexion angle (KFA) and (2) anterior tibial translation (ATT), internal femoral rotation (IFR), internal tibial rotation (ITR), and relative strain of the anteromedial bundle of the anterior cruciate ligament during a cadaver-simulated single-leg pivot landing. Insets in B-1 and C-1: Portions of the internal femoral torque curve showing the bilinear stiffness response because of the hard stop. All data are

from 1 knee specimen (ID No. 20686R). Data are normalized to their peak values (values in parentheses).

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Figure 3.

Scatter plot of peak relative strain of the anteromedial bundle of the anterior cruciate ligament (AM-ACL) versus range of internal femoral rotation quantified for 20 knees under 4 axial femoral rotation conditions (rotation locked, hard stop at $\sim 7^\circ$ of rotation, hard stop at $\sim 11^\circ$ of rotation, no sudden stop in rotation) during the pivot landings. Data points from the same knee specimen are connected with a dashed line. The solid line represents the line of best fit of the full linear mixed model, with a slope (β_{IFRot}) of -0.132 , which explained 91% of the variance in the peak AM-ACL relative strain data. This model predicted peak ACL relative strain with range of hip internal rotation, sex, and age of knee donor as fixed effects and with knee specimen and knee donor as random effects. IFRot, internal femoral rotation.

Figure 4.

Scatter plots of peak relative strain of the anteromedial bundle of the anterior cruciate ligament (AM-ACL) versus (A) femoral rotational stiffness (B) range of anterior tibial translation, (C) range of internal tibial rotation, (D) peak internal tibial deceleration, and (E) difference between time-to-peak internal tibial and femoral rotations quantified for 20 knees under 4 axial femoral rotation conditions (rotation locked, hard stop at ~7° of rotation, hard stop at ~11° of rotation, no hard stop in rotation) during the pivot landings. In plot E, a negative value indicates that peak internal tibial rotation is occurring before peak internal

femoral rotation, and vice versa. The solid lines represent the lines of best fit of the full linear mixed models, including their corresponding R^2 values. IR, internal rotation.

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Figure 5.

Simulation of axial impulsive torque applied distally to a simple lower limb model, including resulting mechanics. (A) The lower limb was modeled with 3 rigid bodies (femur, tibia, foot) attached to each other by springs (k_{Knee} , k_{Ankle}). The end of a third spring (k_{Hip}) was attached to the femur, with its other end fixed. The torsional stiffness of k_{Hip} was systematically varied, while that of k_{Knee} and k_{Ankle} remained constant. An axial torque of 10 N·m was applied to the foot. (B) With a compliant spring (ie, low k_{Hip} stiffness), larger peak femoral rotation and smaller peak tibial rotation were observed. Femoral rotation

peaked shortly after tibial rotation, as shown in D (dark gray shaded area). In comparison, a stiff spring (ie, high k_{Hip} stiffness) resulted in smaller peak femoral rotation and larger tibial rotation. Femoral rotation peaked much longer after tibial rotation, as shown in D (light gray shaded area). This difference in time-to-peak tibial and femoral rotation is shown in D as the gray shaded areas between the vertical lines, which represent time of peak rotation for each segment and condition. (C) As peak femoral rotation increased, peak tibial rotation decreased. Note: femoral rotation is defined as the absolute angle of the femur, whereas tibial rotation is defined as the angle of the tibia relative to the femur.

TABLE 1

Demographic Data of the Donors of Knee Specimens Tested

Sex	Age, y	Height, m	Mass, kg	BMI, kg/m ²
Female (n = 10 ^b)	55.2 ± 10.5	1.67 ± 0.06	60.5 ± 8.3	21.6 ± 3.1
Male (n = 10 ^b)	59.9 ± 6.6	1.77 ± 0.05	81.3 ± 8.2	25.9 ± 2.3

^aValues are represented as average ± 1 SD. BMI, body mass index.

^bThere were 10 knee specimens harvested from 6 donors.

TABLE 2

Testing Protocol—Including Knee Loading Condition and Status of Internal Femoral Rotation—for Each Block of Trials

Protocol Block	Loading Condition	Femoral Rotation
A	comp + flex m	N/A
B ^b	comp + flex m + int tib trq	locked
C ^b	comp + flex m + int tib trq	hard stop at ~7°
D ^b	comp + flex m + int tib trq	hard stop at ~11°
E ^b	comp + flex m + int tib trq	free
A	comp + flex m	N/A

^a comp, compression force; flex m, flexion moment; int tib trq, internal tibial torque; N/A: not applicable.

^b Randomized sequence.