A COMPUTATIONAL KNEE MODEL FOR PRODUCING FAILURE STRAIN IN THE ACL
A POSSIBLE MECHANISM FOR NON-CONTACT ACL RUPTURE

Guess, TM, PhD[1] Cook, JL, DVM PhD[1], Willis BW, PT[1], Ma, R, MD[1], Echelmeyer, D, PT[1], Gray, AD, MD[1]

[1] University of Missouri, Columbia, MO

Missouri Orthopaedic Institute
University of Missouri Health Care

MAC
MIZZOU MOTION ANALYSIS CENTER

Background: Non-contact rupture of the anterior cruciate ligament (ACL) is common, especially in adolescent female athletes. Increased knowledge of injury biomechanics, particularly regarding their relation to anatomical, kinematic, and neuromuscular risk factors would advance development of effective personalized injury prevention strategies.

Hypothesis/Purpose: It is generally accepted that non-contact injury often occurs during planting, cutting and pivoting maneuvers that produce anterior tibial translation, internal tibia rotation and valgus moments at the knee. But, the combination of key biomechanical components yielding failure strain in ACL fibers, and the contribution of known anatomical risk factors, has not been fully elucidated. Given the envelope of kinematic constraint provided by articulating surfaces and soft tissue structures of the knee, we hypothesize that the ACL is vulnerable to an injury mechanism whereby the lateral tibial plateau significantly shifts anteriorly relative to the lateral femoral condyle.

Methods: With knee flexion at 20 degrees, an internal tibia moment (12Nm), valgus moment (6Nm), vastus (450N) and gastrocnemius muscle forces (412N lateral, 733N medial) were applied to a validated computational knee model (Fig. 1).[1, 2] The multibody model was comprised of knee geometries from a 29 year old female and included the medial and lateral menisci with horn and peripheral attachments, collateral ligaments, the posterior cruciate ligament and an ACL represented by 8 fiber bundles.[3] The tibiofemoral motions and tissue forces resulting from the applied moments and muscle forces were then analyzed. To determine the effect of increased knee laxity, simulations were run with a 5% increase in ACL laxity.

Contact Information: Trent M. Guess, Ph.D.
Email: guesstr@health.missouri.edu

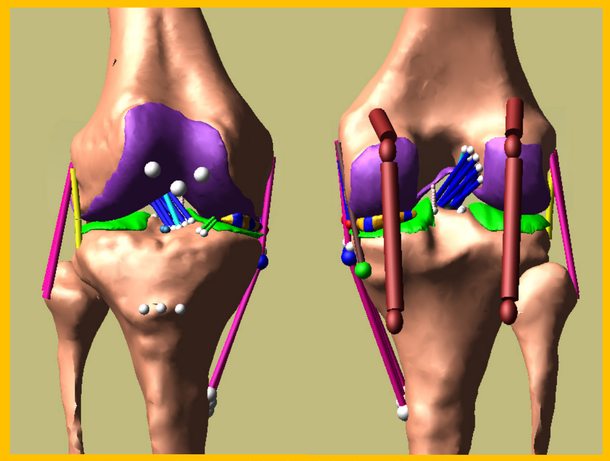

Figure 1: Computational model of a right knee including contact between articulating surfaces, the menisci, ligaments, and vastus and gastrocnemius muscle forces. Note that some geometries (e.g. patella) have been hidden in this figure to facilitate viewing the ACL.

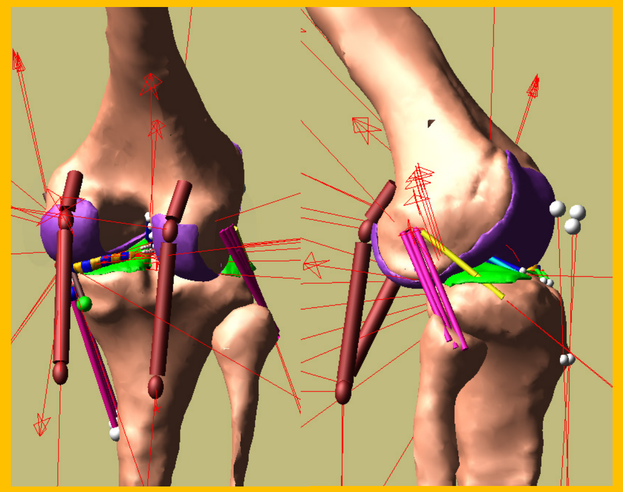

Figure 2: Simulation showing knee response to the applied loads and the proposed ACL injury mechanism. The posterior edge of the lateral tibial plateau has shifted past the lateral femoral condyle. In addition to producing high ACL strain, the anterolateral ligament (yellow ligament in the figure) also experiences high strain.

Results: The combination of internal tibia rotation moment, valgus moment, and vastus and gastrocnemius muscle forces, caused the posterior edge of the lateral tibial plateau to shift anteriorly past the lateral femoral condyle with the knee slightly flexed (Fig. 2). This sequence of events created an increase in ACL strain (Fig. 3), providing a plausible mechanism for non-contact ACL rupture.

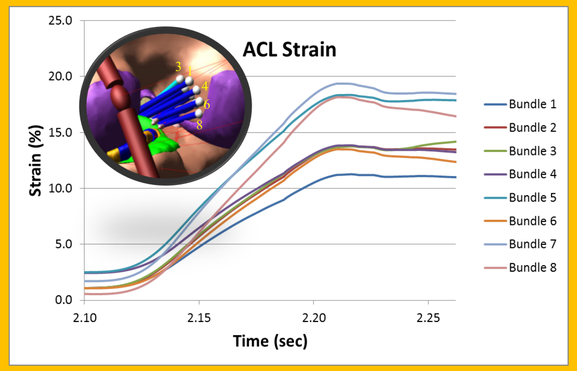

Figure 3: Strain in the 8 ACL bundles in response to knee loading.

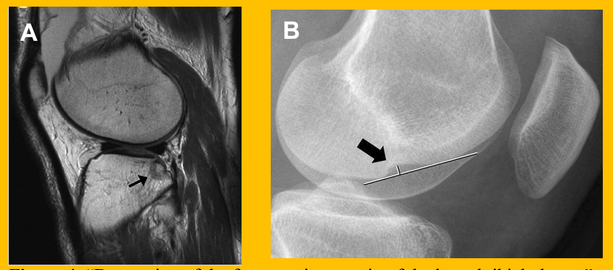

Figure 4: "Depression of the far posterior margin of the lateral tibial plateau" as reported by Potter et al. [4] (A) and location of the lateral femoral notch sign on the lateral femoral condyle as reported by Lodewijks et al. [5] (B).

This injury mechanism is consistent with bone bruise locations [4] and the femoral notch sign.[5] The simulation produced high contact forces and strain in the posterior horn of the medial meniscus and high strain in the lateral meniscus. The posterior cruciate ligament was unloaded, while the medial and lateral collateral ligaments were moderately loaded. The anterolateral ligament experienced high strain in this biomechanical model, consistent with Segond fractures and involvement of the anterolateral complex that often occur with non-contact ACL injury. [6]

This injury mechanism is also consistent with anatomical ACL injury risk factors including increased posterior slope of the lateral tibia plateau and decreased femoral notch width.[7] Elevated knee laxity has also been identified as a risk factor for ACL injury[8], but how increased knee laxity manifests in higher injury risk is unknown. For the same knee loads, our simulation with a 5% increase in ACL laxity resulted in greater internal tibia rotation (Fig. 5a) and an increase in anterior movement of the lateral tibial plateau relative to the lateral femoral condyle. This resulted in an increase in knee abduction (Fig. 5b) as the posterior femur shifted further beyond the eccentric posterior edge of the lateral plateau.

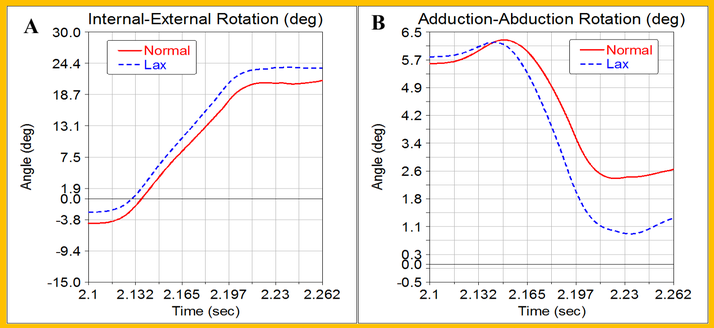

Figure 5: Internal-external (A) and adduction-abduction (B) rotation of the tibia relative to the femur in response to knee loading. Shown are simulations for the unmodified knee (Normal) and with a 5% increase in ACL laxity (Lax).

Conclusion: This model reinforces that strain capable of ACL rupture may occur when the posterior lateral edge of the lateral tibial plateau shifts past the lateral femoral condyle during plant, cut, and pivot maneuvers. External rotation of the body and upper leg on a planted foot, producing internal rotation of the tibia, is a primary contributor to this injury mechanism. As the lateral femoral condyle moves beyond the eccentric posterior margin of the lateral tibia plateau, compressive forces from the lateral gastrocnemius muscle accelerates this motion, further straining the ACL. This motion also increases engagement of ACL fibers with the femoral notch and engagement would increase with decreased notch spacing. Increased posterior slope and elevated knee laxity facilitate internal rotation and anterior translation, increasing the probability of the proposed injury mechanism. As non-contact ACL injury mechanisms are multifaceted, this model further elucidates the precise role of neuromuscular, kinematic, and anatomical risk factors culminating in ACL rupture, which offers healthcare providers insight into methods of reducing such occurrences.

References: [1] Guess TM, et al (2018) *J Knee Surg.* [2] Guess TM and Razu S. (2017) *Med Eng Phys.* [3] Hara K, et al (2009) *Am J Sports Med.* [4] Potter HG, et al (2012) *Am J Sports Med.* [5] Lodewijks P, et al (2019) *Knee Surg Sports Traumatol Arthrosc.* [6] Ferretti A, et al (2019) *Int Orthop.* [7] Sturnick DR, et al (2015) *Am J Sports Med.* [8] Vacek PM, et al (2016) *Am J Sports Med.*