# MCL INSERTION SITE AND CONTACT FORCES IN THE ACL-DEFICIENT KNEE

*Dalton, M S; *Ellis, B J; *Lujan, T J; +*,**Weiss, J A;
+*Department of Bioengineering and Scientific Computing and Imaging Institute, University of Utah, Salt Lake City, UT
jeff.weiss@utah.edu

**INTRODUCTION:** It is unclear how ACL deficiency affects the mechanical function of other knee ligaments. This is important because even knees with reconstructed ACLs often exhibit abnormal knee kinematics [1]. The ACL is a primary restraint to anterior tibial translation and a secondary restraint to valgus rotation. Since the medial collateral ligament (MCL) is the primary restraint to valgus rotation and a secondary restraint to anterior tibial translation, MCL mechanics may be altered after ACL injury. This could have important implications for MCL injury subsequent to ACL injury as well as for combined MCL/ACL injuries. Our previous study demonstrated that ACL deficiency increased MCL strains during anterior-posterior (A-P) tibial translation but not during varus-valgus (V-V) rotation [2]. However, locally large strains do not necessarily translate into large insertion site and contact forces and thus ligament contribution to joint function. The objective of this study was to determine the effect of ACL injury on MCL insertion site forces and contact forces between the MCL and the bones under A-P and V-V loading.

**METHODS:** Five human male knees were tested (59±4.5 yrs). All periarticular soft tissue was removed with the exception of the posterior capsule. Black spheres (2.3 mm dia.) were affixed to the MCL along the local collagen direction. A 3 x 7 grid formed 18 gage lengths for strain measurement (Fig. 1, left).

Each knee was mounted in custom fixtures on a material testing machine that allowed application of V-V rotation and A-P translation at fixed flexion angles with constrained or unconstrained tibial axial rotation. Joint distraction and medial-lateral translation were unconstrained. Results in this study focused on a subset of tests that were performed on each knee: V-V torque/rotation and A-P force/displacement tests (limits of ±10 N-m and ±100 N, respectively), normal vs. ACL-deficient knee, two flexion angles (0 and 30 degrees) with tibial rotation constrained. Ten cycles of either V-V or A-P loading were applied for each test case. MCL strains were measured during the 10th loading cycle using a 3D motion analysis system consisting of two digital cameras (Pulnix TM-1040, 1024x1024x30 fps, Sunnyvale, CA) and analysis software (DMAS, Spica Technology Corp, Maui, HI).

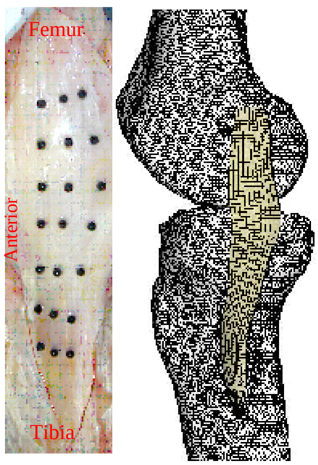


**Fig. 1:** Left – MCL strain markers. Right – FE mesh of femur, MCL and tibia for one knee.

After testing, the MCL was dissected from its femoral, tibial, and meniscal attachments for measurement of the reference lengths [3]. The isolated ligament was placed on a saline covered glass plate and allowed to assume its stress-free configuration. The motion analysis system was used to record the stress-free position of the surface markers. After the marker positions were found for both the stress-free configuration and during anterior, posterior, and valgus loadings, the strain between marker pairs was calculated for each test case.

Subject-specific finite element (FE) models were constructed for each knee using our published/validated procedures [4] (Fig. 1, right). Experimental kinematics were used to drive the motion of the tibia with respect to the femur. FE model validation was performed by comparing regional strains predicted by the FE model to those measured experimentally. Contact forces (between femur-MCL and tibia-MCL) and insertion site forces (femoral and tibial insertions) were determined for each condition (ACL intact and ACL-deficient) for each knee. The effect of ACL state (intact, cut), and flexion angle (0, 30) were assessed for insertion site and contact forces at the femur and tibia using two-way r/m ANOVAs.

**RESULTS:** There was a significant correlation between experimental and FE MCL fiber strains (Fig. 2, $R^2$ = 0.77, p<0.001). The FE models tended to under-predict experimental strain slightly.

There was a significant increase in MCL insertion forces at the femur and tibia under anterior tibial translation after ACL transection (p=0.05 for both insertions) (Fig. 3, left). Insertion site forces were significantly higher at 0 degrees (p=0.035 for both insertions). However there was no effect of ACL transection or flexion angle on insertion site forces for the case of valgus rotation (Fig. 3, right). ACL transection significantly increased tibial (p=0.04) but not femoral contact forces under anterior tibial translation (Fig. 4).

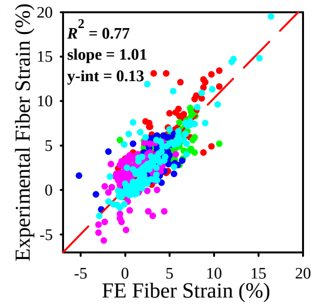


**Fig. 2:** FE vs. exp fiber strain for all knees, test conditions and measurement regions (N=720). Colors indicate different knees.

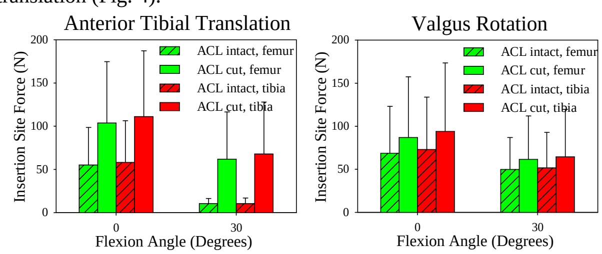


**Fig. 3:** FE predictions of insertion site force for femoral and tibial insertion sites as a function of flexion angle and ACL state.

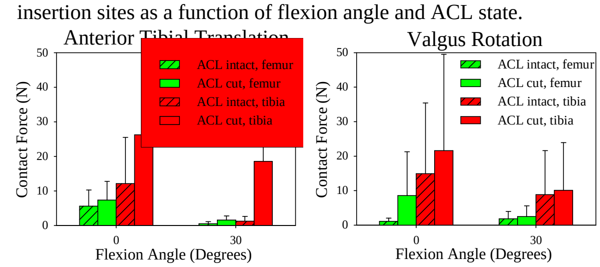


**Fig. 4:** FE predictions of contact forces between the MCL-tibia and MCL-femur as a function of flexion angle and ACL state.

**DISCUSSION:** The results of this study demonstrate that ACL deficiency increases MCL insertion forces and thus vulnerability to injury during anterior tibial translation. In contrast, ACL transection had little effect on MCL insertion site and contact forces during valgus rotation. Although it has been reported that the ACL is a secondary restraint to valgus rotation, Fig 3 (right) shows that the ACL does not contribute to valgus joint stability in knees that have an intact MCL.

Under anterior tibial translation, the largest insertion forces occurred in the normal and ACL-deficient knees at 0 degrees flexion, but the largest percent change occurred at 30 degrees. FE predicted strains as well as experimental measurements [2] indicated that the highest strains in the MCL of the ACL-deficient knee were in the posteromedial corner near the femoral insertion. Taken together, these results highlight the potential for MCL injury in the ACL-deficient knee.

**REFERENCES:** [1] Robins AJ, et al.: Am J Sports Med, 21:20-5, 1993. [2] Lujan et al.: Proc 50th ORS, 29:1272, 2003 (in review, AJSM). [3] Gardiner JC, et al.: Clin Orthop, 391:266-74, 2001. [4] Gardiner JC and Weiss JA: J Orthop Res, 21:1098-1106, 2003.

** Department of Orthopedics, University of Utah, SLC, UT

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