

Knee Contact Biomechanics are Altered at Sites of Articular Cartilage Damage in Femoral Condyles of Adult Rabbits with Post-Traumatic Osteoarthritis

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INTRODUCTION: Altered knee joint mechanics occur after anterior cruciate ligament (ACL) rupture and may contribute to post-traumatic osteoarthritis (PTOA). ACL-deficient knees exhibit abnormal tibial movement relative to the femur, including excessive anterior translation and internal rotation.¹ Such macroscopic joint mechanics may cause changes in tissue-level contact biomechanics and incipient mechanical or mechanobiological damage to articular cartilage (AC) and meniscus (MEN) tissues.² In the rabbit ACL transection (rACLT) model of PTOA, mild surface AC damage patterns occur at specific sites on femoral condyles (FCs),³ MEN exhibit gradual damage,⁴ and *ex vivo* imposed gait-like loading of ACLT rabbit knees induces similar site-specific AC damage as well as abnormal kinematics.⁵ Thus, more detailed *ex vivo* loading analysis of joint contact may clarify the extent of abnormal AC and MEN mechanics at sites where FCs exhibit early AC damage. The aims of this study were to subject rabbit knees *ex vivo* to loading to mimic knee extension within a micro-computed tomography (μ CT) instrument to allow multi-scale assessment of joint biomechanics, and to determine if ACLT knees, relative to intact knees, exhibit abnormal tibia and MEN movement.

METHODS: Hindlimbs from NZW adult rabbits ($n=3$) were used. Biomechanics were assessed by μ CT imaging knees under tare load or extension-simulating load, at one of two positions, for intact or ACL-transected (ACLT) states. Loading simulated knee extension in the swing phase of normal activity. The two knee States (S) were (1) non-operated, normal (NL) and (2) *ex vivo* transected (ACLT). The two knee Positions (P) were flexion angles of (A) 90° and (B) 135°. Knees were imaged in S-P 1A and 1B under tare load, and in S-P 1A, 1B, 2A, and 2B under load. **Contrast-enhanced μ CT.** Joints were stained with 2% phosphotungstic acid in 70% ethanol (3days), then rehydrated with 70% H₂O (2d) and PBS (1d). During loading, imaging was performed at (35 μ m)³ voxel resolution. **Knee Loading.** Loading was applied via lines through osseous tunnels to mimic loading by quadriceps (F^Q) and gastrocnemius (F^G) muscle groups (Fig 1). A tare load of $F^Q=10$ N for and $F^G=4$ N were applied. Physiological loads for F^Q 35 N at 90° and 41 N at 135° and for F^G of 10 N were applied.⁶ **μ CT Image Analysis.** Knee images at different S-P combinations were co-registered referencing FC anatomical coordinate system. **Knee kinematics** including tibia internal-external (I-E), adduction-abduction (Ad-Ab), and A-P translation were determined, relative to tare state.⁷ **TP sliding** relative to FC, $D_{FC,TP}^{A-P}$, was quantified in 2D sagittal images from **outer**, **central**, and **inner** subsites of medial and lateral compartments (MFC-MTP, LFC-LTP). $D_{FC,TP}^{A-P}$ between tare and S-P combinations was determined by computing the difference between the distance from reference (most distal point on FC) to target (most posterior aspect of TP) landmark along the TP surface. **Meniscus kinematics**, A-P displacement relative to TP, $d_{TP,ACM}^{A-P}$, was assessed by localizing menisci centroid displacement. In 2D mid-sagittal images, centroids of A and P regions of medial and lateral menisci (MM, LM) were obtained by averaging the coordinates of triangular vertex positions. Repeatability was assessed from two trials. **Statistics.** NL and *ex vivo* ACLT knees were compared at each knee flexion for TP and meniscus kinematics, and TP sliding at subsite for metrics using student's paired t-test. Significance was taken as $p<0.05$.

RESULTS: Repeatability. Across-trial standard deviations (SD) for TP rotations and translation measures were relatively small, averaging 0.05° for I-E rotation, 0.15° for Ad-Ab, and 0.18 mm for A-P translation. SD averaged 0.03 mm for all TP sliding measures, and 0.02 mm for meniscus vertex positions. **Knee Kinematics.** At 135°, TP internal rotation was higher in ACLT knees ($\Delta=-3.39^\circ$), and abduction trended higher ($\Delta=-0.60^\circ$), compared to NL (Fig. 1). **TP Sliding.** At 135°, ACLT knees demonstrated higher TP anterior sliding at OUT ($\Delta=0.67$ mm), CENT ($\Delta=0.65$ mm), and INN ($\Delta=0.38$ mm) subsites of LFC-LTP in ACLT knees compared to NL (Fig. 2). **Meniscus Kinematics.** At 135°, ACLT knees showed higher posterior displacement of posterior LM ($\Delta=-0.26$ mm), and anterior displacement of posterior MM ($\Delta=0.32$ mm) compared to NL (Fig. 3).

DISCUSSION: With ACLT, the increased TP internal rotation and excessive TP sliding between LFC-LTP are self-consistent. This ACLT-associated difference suggests a localized site of increased sliding velocity and stress concentrations between FC AC with TP AC and meniscus that could cause AC damage at OUT, CENT, and INN subsites. Altered meniscal kinematics in the posterior region may herald meniscal weakening and tear with repeated loading and also potentiate injurious loading of FC AC.

SIGNIFICANCE: The established link between biomechanics at the joint and tissue scales defines the local kinematics and tissue strains that may cause direct biomechanical damage or abnormal mechanobiology leading to articular cartilage and meniscus damage in ACL-deficient knees.

REFERENCES: [1]Andriacchi T+, *Ann Biomed Eng*, 2004.[2]Nelson F+, *Osteoarthritis Cartilage*, 2006.[3]Jelvani, *Trans ORS*, 2020.[4]Hellio Le Graverand M+, *Osteoarthritis Cartilage*, 2001.[5]Caffrey J+, *Osteoarthritis Cartilage*, 2020.[6]Gushue D+, *J Orthop Res*, 2005.[7]Grood E+, *Biomech Eng*, 1983.

FIGURES AND TABLES:

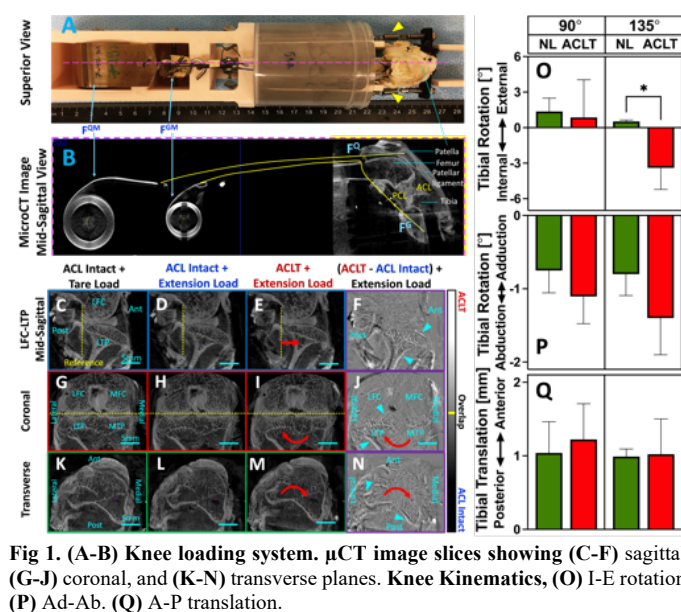


Fig 1. (A-B) Knee loading system. μ CT image slices showing (C-F) sagittal, (G-J) coronal, and (K-N) transverse planes. **Knee Kinematics**, (O) I-E rotation. (P) Ad-Ab. (Q) A-P translation.

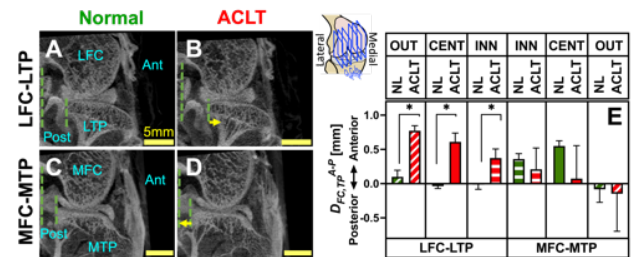


Fig 2. Tibial sliding. * $p<0.05$.

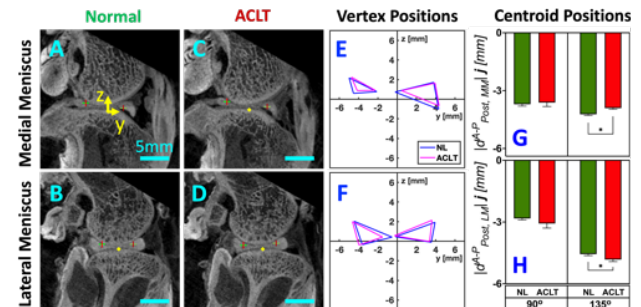


Fig 3. Meniscus kinematics. * $p<0.05$.