**MENISCAL INJURY IN CONJUNCTION WITH ACUTE AND CHRONIC ACL TEARS INCREASE PEAK CARTILAGE STRESSES**

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## INTRODUCTION

Anterior cruciate ligament (ACL) injury is frequently associated with an increased incidence of premature knee osteoarthritis (OA) (Daniel et al. 1994). In conjunction with acute ACL ruptures, meniscal tears are typically found in the lateral meniscus while the medial meniscus usually remains intact. Yet, in chronic cases of ACL ruptures, the medial meniscus shows degeneration in most subjects while damage to the lateral meniscus does not progress (Cipolla et al. 1995). The rotational kinematic changes along with the high loads during the initial phase of the gait cycle have been suggested as one possible mechanism for the initiation of cartilage breakdown after ACL injury (Andriacchi et al. 2004). Yet, the combined effect of these rotational changes with lateral meniscal damage on cartilage stress has not been examined. This study sought to compare the effects of both a typical acute and chronic ACL deficiency on the peak stresses in the tibial articular cartilage in the neutral position of the knee as well as changes in the locations of these peak stresses relative to a healthy knee.

## METHODS

The geometry for the femur, tibia, femoral and tibial articular cartilage, and menisci were derived from segmented magnetic resonance (MR) images to build 3-D models of the bones, cartilage, and menisci. (Koo et al. 2005) and solid meshes were created using MSC Patran (MSC Software, Santa Ana, CA). The model consisted of 30,311 nodes and 15,294 elements. Bone was assumed to be rigid relative to the soft tissue and modeled using rigid 2D elements. The cartilage was discretized into 8-noded brick elements while the meniscus was discretized into 10-noded tetrahedral elements. The cartilage was assumed to behave as a linear elastic, isotropic solid with a modulus of 10 MPa and a Poisson's ratio of 0.46 (Haut Donahue et al. 2002). The menisci were modeled as linearly elastic, transversely isotropic with radial and axial moduli of 20 MPa and a circumferential modulus of 140 MPa (Yao et al. 2006). The in-plane Poisson's ratio was 0.2, while the out of plane Poisson's ratio was 0.3. A previous method was used (Haut Donahue et al. 2002). to model the meniscal horns.

The anterior (ACL) and posterior (PCL) cruciate ligaments and medial (MCL) and lateral (LCL) collateral ligaments were modeled using nonlinear tension-only springs with a constant, k, of 1500. Contact was modeled between the femoral cartilage and the meniscus, the meniscus and the tibial cartilage, and the femoral and tibial cartilage for both the medial and lateral sides resulting in six contact pairs with a coefficient of friction of .00001 between all contact pairs. An ACL-deficient knee was modeled by removing the ACL spring elements and rotating the tibia 5º internally from the normal knee. A lateral longitudinal meniscal tear was introduced by removing elements from the posterior portion of the lateral meniscus. Medial meniscal degeneration was simulated by removing approximately 20%, by volume, of the elements in the posterior third of the medial meniscus. The medial-lateral load ratio at the instant of the first peak adduction moment during gait was used (Schipplein et al.1991). This resulted in a 5.15:1 medial-lateral load ratio, which was distributed as a 700 N (1 body weight) load. The femur was only constrained to maintain full extension while the tibia was held fixed in all six degrees of freedom. Model predictions (ABAQUS 6.5 (HKS Inc., Pawtucket, RI) of stress distributions and contact areas were tested by comparing the location of peak stresses to the location of peak contact pressure found in cadavers using pressure-sensitive film (Ihn et al. 1993).

## RESULTS AND DISCUSSION

The results support the hypothesis that the shift in the location of the contact regions post ACL injury could result in high loads on cartilage that cannot adapt to withstand these loads. Introducing an ACL-deficiency (ACLD) with a rotational offset resulted in increased peak von Mises stresses in the lateral plateaus (Fig 1) in every case. The ACLD in all cases resulted in an anterior shift of the peak stresses on the lateral side and a posterior shift medially (Fig 2). This could result in degenerative changes to the cartilage initiating the early stages of OA.

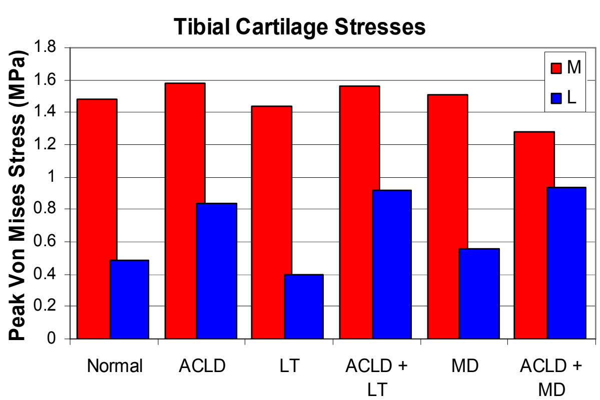


**Fig 1.** Peak von Mises stresses in medial (M) and lateral (L) tibial plateaus.

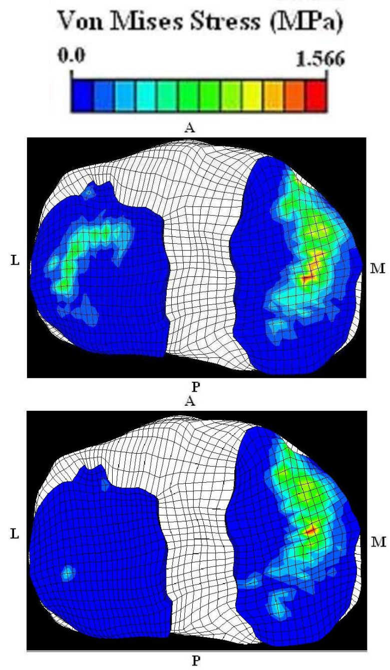


**Fig 2.** Distribution of peak von Mises stresses on the tibial plateau for normal (top) and ACLD+LT (bottom).

While the simplifications in this study likely influenced the magnitudes of stresses predicted by this model, the conclusions regarding the relative increases between the ACL deficient knee and the healthy knee seem consistent with the clinical observation that kinematic changes associated with ACL injury can lead to premature OA in this population.

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