



# The relationship between peak knee extension at heel-strike of walking and the location of thickest femoral cartilage in ACL reconstructed and healthy contralateral knees



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## ABSTRACT

Reports that knee cartilage health is sensitive to kinematic changes, combined with reports of extension loss following ACL reconstruction, underscores the importance of restoring ambulatory knee extension in the context of preventing premature osteoarthritis. The purpose of this study was to test the relationship between individual variations in peak knee extension at heel-strike of walking and the anterior–posterior location of thickest cartilage in the medial and lateral femoral condyles of healthy contralateral and ACL reconstructed knees. In vivo gait analysis and knee MR images were collected from 29 subjects approximately 2 years after unilateral ACL reconstruction. Knee extension was measured at heel-strike of walking and 3-D femoral cartilage thickness models were reconstructed from MR images. The ACL reconstructed knees had significantly reduced knee extension ( $-1.5 \pm 4.2^\circ$ ) relative to the contralateral knees ( $-4.6 \pm 3.4^\circ$ ) at heel-strike of walking but did not have side-to-side differences in the anterior–posterior location or magnitude of thickest medial and lateral femoral cartilage. The anterior–posterior location of the thickest medial femoral cartilage was correlated with knee extension at heel-strike in both the healthy contralateral ( $R^2=0.356$ ,  $p<0.001$ ) and reconstructed ( $R^2=0.234$ ,  $p=0.008$ ) knees. These results suggest that ACL reconstruction can impair terminal extension at periods of ambulatory loading known to be related to cartilage morphology in healthy joints. The fact that the femoral cartilage thickness distribution had not changed at 2 years post-op, even in the subset of subjects with extension loss, suggests that loads may be shifted to thinner cartilage regions, which could have important implications on long-term joint health.

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## 1. Introduction

There is increasing evidence that the mechanics of walking play a critical role in both the maintenance and breakdown of knee articular cartilage (Andriacchi et al., 2004, 2006, 2009; Chaudhari et al., 2008; Dye, 1996; Seehom, 2006). Specifically, healthy cartilage becomes conditioned to the individual loading variations during walking, as observed in studies correlating medial–lateral tibiofemoral cartilage thickness ratio to the adduction moment during walking (Andriacchi et al., 2004; Koo and Andriacchi, 2007). Kinematics during walking can also influence inter-subject regional variations in the femoral cartilage thickness in healthy subjects, as it has been reported that variations in knee extension at heel-strike were associated with the anterior–posterior location of the thickest femoral cartilage (Koo et al., 2011). Taken together, these findings

suggest that healthy cartilage biologically and structurally adapts over time to both the location and magnitude of ambulatory loads by increasing the thickness in the frequently loaded regions.

The fact that mature cartilage has limited capacity to adapt to changes (Mankin, 1982), combined with the observation that healthy cartilage is conditioned to normal patterns of walking, may render cartilage sensitive to abrupt changes in knee kinematics. Specifically, it is proposed that a degenerative pathway can be initiated when changes in walking kinematics shift loading to regions of cartilage that cannot adapt to the new mechanical environment (Andriacchi et al., 2009). This theory has been motivated by observations of ACL deficient subjects, where both computational (Andriacchi et al., 2006) and experimental (Andriacchi et al., 2009) studies have linked kinematic alterations during walking to specific patterns of cartilage thinning. Such findings have led to increasing scrutiny of knee kinematics following ACL reconstruction to better understand its failure to prevent the high incidence of post-injury osteoarthritis (Lohmander et al., 2004; Meuffels et al., 2009; von Porat et al., 2004).

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Loss of passive knee extension following ACL reconstruction, occurring in 11–25% of knees (Harner et al., 1992; Mauro et al., 2008; Sachs et al., 1989; Shelbourne and Gray, 2009), is one of the most common deficits after reconstruction. The clinical observation of extension loss is consistent with reports that ACL reconstructed knees are generally more flexed during walking (Ferber et al., 2002; Gokeler et al., 2003; Hunt et al., 2010; Scanlan et al., 2010; Webster et al., 2012), with some studies specifically observing an extension loss at heel-strike of walking (Hunt et al., 2010; Scanlan et al., 2010; Webster et al., 2012) where the normal knee is typically near full extension (Benoit et al., 2007; Besier et al., 2003; Chao et al., 1983; Dyrby and Andriacchi, 2004; Koo et al., 2011). While the short-term functional deficits of knee extension loss are well known (Sachs et al., 1989), recent clinical literature indicates that post-operative loss of extension is also predictive of an increased risk of osteoarthritis in the ACL reconstructed knee (Shelbourne and Gray, 2009). Given that in healthy joints the individual variations in the location of thickest cartilage are conditioned to variations in knee extension specifically at heel-strike of walking (when the knee is typically near full extension) (Koo et al., 2011), these findings raise the question of whether the relationship between kinematics and cartilage morphology is disrupted after ACL reconstruction.

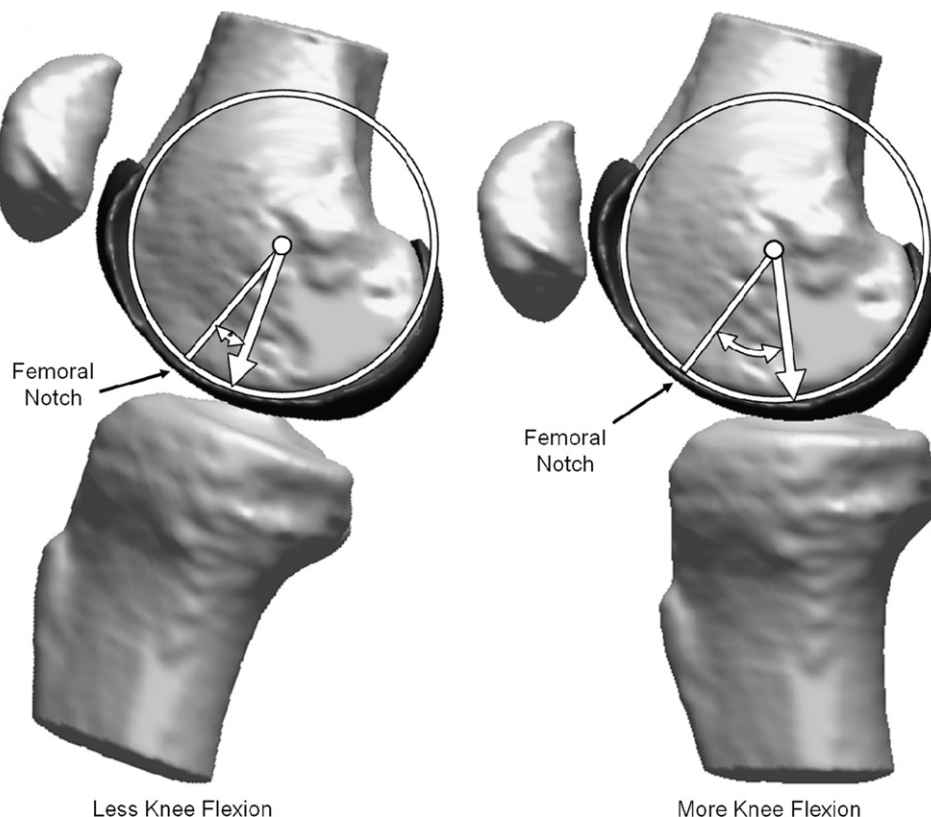
The purpose of this study was to test the relationship between peak knee extension at heel-strike of walking and the anterior–posterior (A–P) location of thickest cartilage in the medial and lateral femoral condyles in healthy contralateral and ACL reconstructed knees of subjects at approximately 2 years post-surgery. Specifically, it was hypothesized that side-to-side differences in extension would not correspond to side-to-side differences in thickest cartilage location, providing new evidences to support the potential pathway to knee osteoarthritis described above.

## 2. Methods

Twenty-nine subjects with unilateral transtibial ACL reconstruction and no other history of serious lower limb injury were enrolled in this IRB-approved study (15 male; age:  $28.7 \pm 6.3$  years; height:  $170.7 \pm 8.2$  cm; weight:  $72.9 \pm 12.1$  kg; injured knee: 17 right). The subjects underwent ACL reconstruction at  $2.6 \pm 1.9$  months (range, 0.4–7.9 months) after their index injury and were examined at a mean follow-up of  $26.9 \pm 3.7$  months (range, 21.9–35.8 months). Exclusion criteria included clinical instability (self-reported history of giving-way episodes, KT-1000 manual maximum side-side difference  $\geq 5$  mm), removal of more than 25% of the meniscus, a history of other serious ligamentous injury to either lower limb, or a history of surgical procedures performed on either lower limb. All reconstructed patients had undergone single-bundle transtibial reconstruction with Achilles tendon allografts and interference screw fixation.

All subjects performed level walking trials at their self-selected walking speed until three successful trials were collected per side (Dyrby and Andriacchi, 2004). Data were collected for analysis during the middle stride of several consecutive strides. Therefore, six total strides were analyzed per subject, three left strides and three right strides. An eight-camera opto-electronic system for 3-D motion analysis (Qualisys, Gothenburg, Sweden) was used to collect lower limb kinematics, while a multi-component force plate (Bertec, Columbus, OH) recorded the ground reaction force. Marker motion data and force data was collected at 120 Hz and custom software was used to calculate the knee kinematics during the gait cycle using the previously-described point-cluster technique (Andriacchi et al., 1998; Dyrby and Andriacchi, 2004). The heel-strike portion of gait is of particular interest for this study because large knee contact forces ( $> 1$  body weight) occur with the knee near full extension (Schipplein and Andriacchi, 1991; Zhao et al., 2007) and heel-strike knee extension is correlated with spatial variations in cartilage thickness in the femoral condyles of healthy subjects (Koo et al., 2011). Moreover, heel-strike knee extension has been used as a surrogate measure of the anterior–posterior location of contact on the femoral condyles (Fig. 1) (Li et al., 2005; Koo et al., 2011). Therefore, for each trial peak heel-strike knee extension was measured as the local minimum knee flexion angle (maximum knee extension) achieved at, or within 40 ms, of foot contact (Fig. 2) as determined by the force plate. The values of the three trials were then averaged to obtain one mean peak knee extension angle per subject per knee. The average self-selected walking speed of the subjects was  $1.39 \pm 0.15$  m/s.

All subjects underwent an MRI of both knees on a 1.5 T MR scanner (GE Signa 1.5 T; GE Medical Systems, Milwaukee, WI). High contrast MR images were acquired using a sagittal-plane fat-saturated 3-D spoiled gradient recalled echo



**Fig. 1.** Given the shape of the femorotibial joint and that flexion is the primary motion of the knee, knee flexion angle is a surrogate of the anterior–posterior location of sagittal-plane contact on the femoral condyles (white arrow).

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