Published in final edited form as:
J Biomech. 2013 February 1; 46(3): 478–483. doi:10.1016/j.jbiomech.2012.10.031.

In vivo measurement of ACL length and relative strain during walking

K A Taylor[1], **H C Cutcliffe**[1,4], **R M Queen**[1,2], **G M Utturkar**[1], **C E Spritzer**[3], **W E Garrett**[1], and **L E DeFrate**[1,4]

[1]Sports Medicine Center, Department of Orthopaedic Surgery, Duke University, Durham NC

[2]Michael W. Krzyzewski Human Performance Lab, Department of Orthopaedic Surgery, Duke University, Durham NC

[3]Department of Radiology, Duke University Medical Center, Duke University, Durham NC

[4]Department of Biomedical Engineering, Duke University, Durham NC

Abstract

Although numerous studies have addressed the effects of ACL injury and reconstruction on knee joint motion, there is currently little data available describing in vivo ACL strain during activities of daily living. Data describing in vivo ACL strain during activities such as gait is critical to understanding the biomechanical function of the ligament, and ultimately, to improving the surgical treatment of patients with ACL rupture. Thus, our objective was to characterize the relative strain in the ACL during both the stance and swing phases of normal level walking. Eight normal subjects were recruited for this study. Through a combination of magnetic resonance imaging, biplanar fluoroscopy, and motion capture, we created in vivo models of each subject's normal walking movements to measure knee flexion, ACL length, and relative ACL strain during gait. Regression analysis demonstrated an inverse relationship between knee flexion and ACL length (R^2=0.61, p<0.001). Furthermore, relative strain in the ACL peaked at 13±2% (mean± 95%CI) during mid-stance when the knee was near full extension. Additionally, there was a second local maximum of 10±7% near the end of swing phase, just prior to heel strike. These data are a vital step in further comprehending the normal in vivo biomechanics experienced by the ACL. In the future, this information could prove critical to improving ACL reconstruction and provide useful validation to future computational models investigating ACL function.

Keywords

anterior cruciate ligament; biomechanics; gait; knee; model

Corresponding Author: Louis E. DeFrate, Ph.D., Assistant Professor, Orthopaedic Research Laboratories, Department of Orthopaedic Surgery, Box 3093, Duke University Medical Center, Durham, NC 27710.

Conflict of Interest

The authors have no conflicts of interest to declare with regards to this study.

1. Introduction

Despite encouraging clinical outcomes after surgery, a number of studies have reported an inability of ACL reconstruction to reproduce normal knee motion (Abebe et al., 2011b; Gao and Zheng, 2010; Tashman et al., 2007). These findings are important because abnormal knee motion potentially predisposes the joint to degenerative changes (Andriacchi et al., 2004; Griffin and Guilak, 2005; Hosseini et al., 2012), which remain a concern after ACL reconstruction (Lohmander et al., 2007; Salmon et al., 2006; Shelbourne et al., 2012). Therefore, in order to improve surgical interventions such that they more closely restore normal ACL function, data describing the forces and displacements the ACL experiences during activities of daily living is needed (Herfat et al., 2012; Wu et al., 2010). Additionally, information on the biomechanical role of the ACL during in vivo activities is important to the design of rehabilitation protocols (Fleming et al., 2001a), the development of injury prevention programs (Shin et al., 2011), and the validation of mathematical models of ACL function (Shelburne et al., 2004).

Currently, however, there is limited data describing the *in vivo* function of the ACL during gait (Herfat et al., 2012; Wu et al., 2010), an important activity of daily living. A number of investigations quantified joint motion during gait in normal subjects (Andriacchi et al., 1998; Lafortune et al., 1992) and subjects with ACL injuries (Andriacchi and Dyrby, 2005; Chen et al.; Gao and Zheng, 2010; Gardinier et al., 2012). Other groups have utilized implantable strain gauges to measure local ACL strains during several common athletic and rehabilitation movements (Beynnon et al., 1997b; Cerulli et al., 2003; Fleming et al., 1998). Recently, Wu et al. (Wu et al., 2010) reported in vivo ACL kinematics during treadmill gait using biplanar fluoroscopic and MR imaging. However, this analysis was restricted to only the stance phase of treadmill gait.

Therefore, the purpose of this study was to characterize the length and relative strain (Fleming and Beynnon, 2004) of the ACL during a complete over-ground gait cycle (both swing and stance phases of gait) using a combination of marker-based motion capture, MR imaging, and biplanar fluoroscopy (Taylor et al., 2011). We hypothesized that the ACL would experience significant relative strain during both the swing and stance phases of gait. In particular, based on previous studies reporting inverse relationships between ACL elongation and flexion during weightbearing and non-weightbearing loading (Beynnon et al., 1997b; Taylor et al., 2011; Wu et al., 2010), we expected to observe local maxima in the relative strain of the ACL in portions of the gait cycle where the knee was most extended.

2. Methods

2.1 Recruitment of Normal Subjects

Eight (7M, 1F) healthy subjects (mean age: 26, range: 22-32 years, body mass index: 22.8, range: 21.5-25.8) were recruited to participate in this study. All volunteers were physically active, participating in recreational sports or exercise a minimum three days a week for at least one hour each day. Potential subjects were disqualified if they had a history of previous knee injury or other condition that would alter their natural gait. Institutional Review Board approval was obtained for this protocol before enrollment of participants and all subjects gave informed consent prior to beginning any study activities.

2.2 MR Imaging and Model Creation

Coronal, sagittal, and axial MR images of each subject's knee were acquired using a 3T magnet (Trio Tim, Siemens Medical Solutions USA, Malvern, PA). Lying in a supine and relaxed position, subjects were imaged with a double-echo steady state (DESS) sequence (flip angle: 25°, TR: 17 ms, TE: 6 ms) using an eight-channel receive-only knee coil (Invivo,

Orlando, FL). A 15×15cm field of view, a matrix of 512×512 pixels, and a 1mm slice thickness were used, resulting in a voxel resolution of 0.3×0.3×1.0mm. From these images, 3D models of each tested knee were constructed by compiling manually traced outlines of the cortical bone and ACL attachment site using solid-modeling software (Rhinoceros, Robert McNeel and Associates, Seattle, WA) as described previously (Abebe et al., 2011a).

2.3 Validation of Model Creation

In order to further quantify our ability to measure the geometry of the ACL attachment sites accurately, a validation study was performed using three cadaveric knee specimens. Similar to a previous validation performed in our lab (Abebe et al., 2009), each knee was scanned using MR imaging so that 3D models could be created as described above. After imaging, the knees were dissected down to the bone, leaving their respective ACL attachment sites intact. Using a stylus (Immersion Technologies, San Jose, CA) with an accuracy of 0.1mm, the attachment of the ACL was digitized into the solid modeling software. In addition to the ACL attachment, the stylus was used to record points on the bony surface to complete the joint model. An iterative closest point technique was used to align the bony models created using the MR and the digitizing stylus (Figure 1). The difference in the location of the centroids of the attachment sites using MR and the digitizing stylus was 0.3±0.2mm (mean ±SD). These data were similar to our previous validation study (Abebe et al., 2009) and demonstrate that our 3D joint models can accurately recreate the native anatomy and position of the ACL attachment in the knee.

2.4 Motion Capture

Subjects' normal gait patterns were recorded using motion capture while wearing standardized form-fitting athletic gear and footwear. First, an overconstrained marker set, designed to help minimize skin motion artifact during post-processing, was placed unilaterally on each subject's dominant leg (Figure 2) (Taylor et al., 2011). Our motion capture protocol included an initial static standing trial, immediately followed by four successful walking trials. In all trials, three-dimensional kinematic data were collected using an eight-camera system (Motion Analysis Corporation, Santa Rosa, CA) with a sampling rate of 240 Hz. The static standing trial was captured for one second as the subject stood in a relaxed position with their feet shoulder width apart. Subjects then completed the walking trials at a self-selected pace across a 30m walkway (Figure 2). Four embedded force plates (AMTI, Boston, MA, USA) within the walkway were used to collect ground reaction force data at 2400Hz.

2.5 Biplanar Fluoroscopy

Immediately following motion capture, biplanar fluoroscopic images of the knee were taken with the markers still in place, as previously described (Taylor et al., 2011). For the imaging, subjects were postured to replicate the static standing trial taken prior to motion capture (Figure 2). By maintaining the position of the markers throughout testing, the position of the markers relative to the underlying bone could be defined. To accomplish this, the in vivo joint environment was first recreated from the manipulation of individual subject's MR-based 3D bone models in a virtual reproduction of their respective fluoroscopic test environment(Abebe et al., 2011b). The models were positioned so that their projections matched the bony silhouettes on the fluoroscopic images when viewed from the perspective of the orthogonal x-ray sources. Previous validation of this technique has shown that we can measure 3D translations and rotations to within 0.1mm and 0.3°, respectively (DeFrate et al., 2006). Positions of the markers were calculated from the area centroids of each marker on the orthogonal image set.

2.6 Numerical Optimization and Dynamic Model Creation

With the relationship of the markers to the underlying bones and ligament attachment sites quantified, numerical optimization was then utilized to reproduce the dynamic knee motion using 3D models(Taylor et al., 2011). First, the fluoroscopic marker data were registered to those from the static standing trial obtained during motion analysis by rigidly transforming one bone at a time. After this initialization, similar optimizations using subsets of the total marker count that correspond to each individual bone were performed to reduce the error associated with soft tissue motion during the gait trials. To reproduce the 3D dynamic in vivo knee joint environment, the optimized gait kinematics were then superimposed onto the knee model (Figure 2). From this model, kinematic measures of knee flexion angle and ACL length were calculated. Flexion was measured as the angle between axes fitted to the tibial and femoral long shafts about the transepicondylar line of the femur (Taylor et al., 2011). ACL length was defined as the distance between the area centroids of each ligament attachment site. A prior validation demonstrated that, during a lunge activity, length measurements between this technique and that measured directly with biplanar fluoroscopy were strongly correlated, with a root mean square difference of 0.5mm (Taylor et al., 2011). Relative strain was defined as ACL length measurements normalized to each subject's ACL length standing with the leg in full extension during the fluoroscopic imaging.

2.7 Outcome Measures

Data comparison across subjects was achieved by normalizing each trial as a percentage of the gait cycle between subsequent heel strikes on the tested leg. To finalize the gait cycle data, ACL length, relative ACL strain, knee flexion, and ground reaction force (GRF) data were linearly interpolated to the nearest percent increment for each trial and then averaged across all trials.

3. Results

In general, the length of the ACL decreased as flexion angle increased during both the stance and swing phases of gait (Figure 3). For example, the ACL elongated to a maximum length of 34.5±1.4mm (Mean±95% CI) when the knee was slightly extended by 1±2°. This corresponded to a peak relative strain of 13±2% and occurred at approximately 60% of the stance phase. In addition, there was another local peak in ACL length of 33.8±3.2mm toward the end of swing phase immediately prior to heel strike. This peak in ACL length corresponded to a relative strain of 10±7% and occurred when the knee was extended by 4±3°. Regression analysis demonstrated a significant inverse linear relationship between knee flexion and ACL length ($R^2 = 0.61$, $p<0.001$) (Figure 4).

4. Discussion

A number of recent studies have indicated that ACL reconstruction techniques do not restore normal knee motion (Abebe et al., 2011b; Gao and Zheng, 2010; Papannagari et al., 2006; Tashman et al., 2007). Thus, in vivo data characterizing the biomechanical function of the ACL during activities of daily living is important to improving the clinical management of ACL injury (Fleming and Beynnon, 2004; Herfat et al., 2012; Wu et al., 2010). Specifically, in vivo data describing the deformation of the ACL graft could be used to evaluate whether a reconstruction restores the function of the native ACL (Abebe et al., 2011a; Tashman et al., 2007). In this in vivo study, we used a combination of MR imaging, biplanar fluoroscopy, and marker-based motion capture (Taylor et al., 2011) to evaluate the relative strains experienced in the ACL throughout a single gait cycle. Our results indicate that ACL length and knee flexion are inversely related (Figure 4). Accordingly, maximal relative strain levels correlated to instances when flexion angles were near their lowest.

Similar ACL loading trends with respect to knee flexion changes have been shown in other in vivo studies of quasi-static lunges (Jordan et al., 2007; Li et al., 2004), jumping (Taylor et al., 2011), biking (Fleming et al., 1998), and squatting (Beynnon et al., 1997b). Further analysis of our results shows excellent agreement with a biplanar fluoroscopic study of in vivo ACL kinematics during the stance phase of treadmill walking at 0.67m/s (Wu et al., 2010). In particular, our ACL relative strain patterns are nearly identical, with loads initially decreasing with weight acceptance following heel strike and steadily increasing through mid-stance. ACL elongations subsequently plateaued to their maximum values and then decreased sharply as flexion increased and the knee prepared for swing phase. Furthermore, our regression values were in excellent agreement with their reported data (Wu et al., 2010). However, no direct comparisons between their study and this one could be made during swing phase.

Although we observed an inverse relationship of flexion and ACL length during gait, our data also suggests that external loads, such as the GRF, influence ACL elongation patterns during walking. For example, plotting the average ACL length data versus flexion angle reveals a pattern indicative of variations in ACL strain during weightbearing (stance) and non-weight bearing (swing) loading conditions at similar knee flexion angles (Figure 4). Nonetheless, an inverse relationship was observed over the entire gait cycle, as demonstrated by the regression analysis. Moreover, these trends align well with a previous investigation of in vivo local ACL strain in response to closed- and open-kinetic chain exercises by Beynnon et al.; specifically, they reported that maximum strains generated during a squatting and an active flexion-extension activity were similar (Beynnon et al., 1997b). Despite decreases in strain with increasing flexion during both activities, ACL loading during closed-kinetic chain movements was increased relative to open-kinetic chain activity in the midrange of flexion (Beynnon et al., 1997b). These findings parallel our ACL length data and the bifurcation weightbearing creates within the curve (Figure 4). Additional studies offer further explanation by indicating that weight-bearing in low flexion leads to increased anterior tibial translations once compressive joint loads are applied (Beynnon et al., 1997a; Fleming et al., 2001a; Li et al., 1998; Torzilli et al., 1994). With the ACL as a primary restraint to anterior tibial translations, these compressive loads experienced in stance phase would be expected to result in elevated ACL strains (Beynnon et al., 1997a; Fleming et al., 2001a).

Another important factor to consider is the influence of muscle activity. With respect to the ACL, it has been suggested that quadriceps activity directly correlates with increased loading due to the anterior shear forces transmitted through the patellar tendon onto the tibia at lower knee flexion angles (DeFrate et al., 2007; Li et al., 1999; Mesfar and Shirazi-Adl, 2005; Renstrom et al., 1986; Yu and Garrett, 2007). Based on previous EMG studies, the quadriceps begin firing just prior to heel strike in anticipation of ground contact and continue to ramp up their activity in the early portions of stance during weight acceptance (Huber et al., 2011; Knarr et al., 2012; Shelburne et al., 2004). In comparison with our results, these trends relate to instances when the ACL is strained, thus supporting an association between quadriceps contraction and ACL loading. Interestingly, within this section of the walking cycle, the localized peak ACL strain is experienced prior to heel strike when the knee is extended. These results parallel our previous findings in jump landing (Taylor et al., 2011), where we observed peak ACL strain just prior to ground impact when the knee was most extended. Furthermore, even though maximal ACL strains were experienced during the stance phase of walking, this pattern may indicate that ACL dysfunction could have important ramifications to normal swing phase joint mechanics that should not be underestimated when examining long-term meniscal and cartilage degeneration. In particular, the peak relative strain during swing phase reached 10%, a difference of only 3% compared to the overall peak relative strain that occurred during

stance. This might correspond to a considerable restraining force that, when absent, could result in unfavorable relative joint positioning just prior to ground contact. Given the forces generated by weightbearing, even small deviations could be detrimental to normal cartilage homeostasis.

Examining stance phase muscle activity provides further insight into the observed ACL strains. In a fashion similar to closed-kinetic chain exercises, stance phase requires more quadriceps muscle activity than swing phase (Kvist and Gillquist, 2001). This is most likely due to the limb stabilization demands of single-legged weight-bearing. As previously discussed, quadriceps contraction in low flexion increases ACL loading (Li et al., 1999), which is likely due in part to the changing orientation of the patellar tendon with flexion (DeFrate et al., 2007; Nunley et al., 2003). Furthermore, because peak ACL strain occurs near heel rise, the addition of an activating gastrocnemius could translate the tibia even farther forward than the contraction of the quadriceps muscle alone. Fleming et al.(Fleming et al., 2001b) previously identified gastrocnemius contractions as an antagonist to the ACL, inducing increases in ACL strain if the knee flexion angle was below 15°. Given the supporting EMG data and knee flexion angles experienced at this point in the gait cycle, it is reasonable to associate the two with maximal ACL loading during walking.

Finally, our data may help to explain altered motion patterns arising after ACL injury (Ahmed and McLean, 2002; Andriacchi and Dyrby; Gao and Zheng, 2010; Hurd and Snyder-Mackler, 2007). For example, previous studies have reported increased knee flexion around the incidence of terminal extension during the stance phase of gait in ACL deficient subjects (Gao and Zheng, 2010; Hurd and Snyder-Mackler, 2007). This may be a neuromuscular adaptation to minimize knee extension, where the quadriceps loading can induce anterior translation of the tibia (Berchuck et al., 1990). Moreover, this is a region of the gait cycle where ACL deficiency resulted in increased anterior tibial translation in a recent study using biplanar fluoroscopy and MR imaging (Chen et al., 2012). In addition to stance phase abnormalities, other investigators found changes in anterior translation and internal rotation of the tibia during terminal swing in ACL deficient patients when the knee was extended prior to heel strike (Andriacchi and Dyrby, 2005). These two portions of the gait cycle correspond with regions of elevated ACL strain in our study.

Several limitations of this study should be considered. With an oblique orientation in both the sagittal and coronal planes, the ACL may have important restraining roles in other degrees of freedom (DeFrate et al., 2006; Fleming et al., 2001a). In this study, we only reported kinematic data on knee flexion during overground gait. However, additional relationships to other kinematic parameters may exist and should be evaluated across a wide range of in vivo activities. Such data might provide critical information regarding what motions and activities are most demanding to the ACL. Additionally, relative strain was approximated by normalizing ACL elongations to a reference length measured during the initial static standing trial collected prior to motion capture. This reference length was selected due to the difficulties associated with determining true resting length in vivo (Fleming and Beynnon, 2004). Despite uncertainty in the true strain values, the trends observed in relative strain during stance demonstrated excellent agreement with a previous fluoroscopic study (Wu et al., 2010).

In conclusion, our novel approach provides a new means for measuring in vivo kinematic patterns and ACL strains during unrestricted dynamic movements. For this study, we determined the normal in vivo ACL strain patterns that occur during unhindered, overground, self-paced walking. Our main findings included a peak relative strain of 13% transpiring during stance when knee flexion was near its minimum, as well as a predominantly inverse relationship between knee flexion and ACL loading (R^2=0.61,

p<0.001). An additional localized peak strain of 10% occurred when the knee was extended just prior to heel strike, indicating the importance of the ACL to normal knee function under non-weightbearing conditions as well. This information is pertinent to the continued improvement of current ACL reconstruction techniques that attempt to restore normal physiologic function.

Acknowledgments

This work was supported by the National Institutes of Health (Grant No. R03AR055659) and a grant from the National Football League Charities. We also gratefully acknowledge the financial support of the Duke University Medical Center's Departments of Orthopaedic Surgery and Radiology. The authors thank Libby Pennington and Megan Terry for technical support.

Bibliography

Abebe ES, Kim JP, Utturkar GM, Taylor DC, Spritzer CE, Moorman CT 3rd, Garrett WE, DeFrate LE. The effect of femoral tunnel placement on ACL graft orientation and length during in vivo knee flexion. J Biomech. 2011a; 44:1914–1920. [PubMed: 21570688]

Abebe ES, Moorman CT 3rd, Dziedzic TS, Spritzer CE, Cothran RL, Taylor DC, Garrett WE Jr. DeFrate LE. Femoral tunnel placement during anterior cruciate ligament reconstruction: an in vivo imaging analysis comparing transtibial and 2-incision tibial tunnel-independent techniques. Am J Sports Med. 2009; 37:1904–1911. [PubMed: 19687514]

Abebe ES, Utturkar GM, Taylor DC, Spritzer CE, Kim JP, Moorman CT 3rd, Garrett WE, DeFrate LE. The effects of femoral graft placement on in vivo knee kinematics after anterior cruciate ligament reconstruction. J Biomech. 2011b; 44:924–929. [PubMed: 21227425]

Ahmed AM, McLean C. In vitro measurement of the restraining role of the anterior cruciate ligament during walking and stair ascent. Journal of biomechanical engineering. 2002; 124:768–779. [PubMed: 12596646]

Andriacchi TP, Alexander EJ, Toney MK, Dyrby C, Sum J. A point cluster method for in vivo motion analysis: applied to a study of knee kinematics. Journal of biomechanical engineering. 1998; 120:743–749. [PubMed: 10412458]

Andriacchi TP, Dyrby CO. Interactions between kinematics and loading during walking for the normal and ACL deficient knee. J Biomech. 2005; 38:293–298. [PubMed: 15598456]

Andriacchi TP, Mundermann A, Smith RL, Alexander EJ, Dyrby CO, Koo S. A framework for the in vivo pathomechanics of osteoarthritis at the knee. Ann Biomed Eng. 2004; 32:447–457. [PubMed: 15095819]

Berchuck M, Andriacchi TP, Bach BR, Reider B. Gait adaptations by patients who have a deficient anterior cruciate ligament. J Bone Joint Surg Am. 1990; 72:871–877. [PubMed: 2365720]

Beynnon BD, Johnson RJ, Fleming BC, Peura GD, Renstrom PA, Nichols CE, Pope MH. The effect of functional knee bracing on the anterior cruciate ligament in the weightbearing and nonweightbearing knee. Am J Sports Med. 1997a; 25:353–359. [PubMed: 9167816]

Beynnon BD, Johnson RJ, Fleming BC, Stankewich CJ, Renstrom PA, Nichols CE. The strain behavior of the anterior cruciate ligament during squatting and active flexion-extension. A comparison of an open and a closed kinetic chain exercise. Am J Sports Med. 1997b; 25:823–829. [PubMed: 9397272]

Cerulli G, Benoit DL, Lamontagne M, Caraffa A, Liti A. In vivo anterior cruciate ligament strain behaviour during a rapid deceleration movement: case report. Knee Surg Sports Traumatol Arthrosc. 2003; 11:307–311. [PubMed: 14523613]

Chen CH, Li JS, Hosseini A, Gadikota HR, Gill TJ, Li G. Anteroposterior stability of the knee during the stance phase of gait after anterior cruciate ligament deficiency. Gait & posture. 2012; 35:467–471. [PubMed: 22169387]

DeFrate LE, Nha KW, Papannagari R, Moses JM, Gill TJ, Li G. The biomechanical function of the patellar tendon during in-vivo weight-bearing flexion. J Biomech. 2007; 40:1716–1722. [PubMed: 17070815]

DeFrate LE, Papannagari R, Gill TJ, Moses JM, Pathare NP, Li G. The 6 degrees of freedom kinematics of the knee after anterior cruciate ligament deficiency: an in vivo imaging analysis. Am J Sports Med. 2006; 34:1240–1246. [PubMed: 16636348]

Fleming BC, Beynnon BD. In vivo measurement of ligament/tendon strains and forces: a review. Ann Biomed Eng. 2004; 32:318–328. [PubMed: 15095807]

Fleming BC, Beynnon BD, Renstrom PA, Peura GD, Nichols CE, Johnson RJ. The strain behavior of the anterior cruciate ligament during bicycling. An in vivo study. Am J Sports Med. 1998; 26:109–118. [PubMed: 9474411]

Fleming BC, Renstrom PA, Beynnon BD, Engstrom B, Peura GD, Badger GJ, Johnson RJ. The effect of weightbearing and external loading on anterior cruciate ligament strain. J Biomech. 2001a; 34:163–170. [PubMed: 11165279]

Fleming BC, Renstrom PA, Ohlen G, Johnson RJ, Peura GD, Beynnon BD, Badger GJ. The gastrocnemius muscle is an antagonist of the anterior cruciate ligament. J Orthop Res. 2001b; 19:1178–1184. [PubMed: 11781021]

Gao B, Zheng NN. Alterations in three-dimensional joint kinematics of anterior cruciate ligament-deficient and -reconstructed knees during walking. Clin Biomech (Bristol, Avon). 2010; 25:222–229.

Gardinier ES, Manal K, Buchanan TS, Snyder-Mackler L. Gait and Neuromuscular Asymmetries after Acute ACL Rupture. Med Sci Sports Exerc. 2012

Griffin TM, Guilak F. The role of mechanical loading in the onset and progression of osteoarthritis. Exercise and sport sciences reviews. 2005; 33:195–200. [PubMed: 16239837]

Herfat ST, Boguszewski DV, Shearn JT. Applying simulated in vivo motions to measure human knee and ACL kinetics. Ann Biomed Eng. 2012; 40:1545–1553. [PubMed: 22227973]

Hosseini A, Van de Velde S, Gill TJ, Li G. Tibiofemoral cartilage contact biomechanics in patients after reconstruction of a ruptured anterior cruciate ligament. J Orthop Res. 2012

Huber C, Nuesch C, Gopfert B, Cattin PC, von Tscharner V. Muscular timing and inter-muscular coordination in healthy females while walking. Journal of neuroscience methods. 2011; 201:27–34. [PubMed: 21784101]

Hurd WJ, Snyder-Mackler L. Knee instability after acute ACL rupture affects movement patterns during the mid-stance phase of gait. J Orthop Res. 2007; 25:1369–1377. [PubMed: 17557321]

Jordan SS, DeFrate LE, Nha KW, Papannagari R, Gill TJ, Li G. The in vivo kinematics of the anteromedial and posterolateral bundles of the anterior cruciate ligament during weightbearing knee flexion. Am J Sports Med. 2007; 35:547–554. [PubMed: 17261571]

Knarr BA, Zeni JA Jr. Higginson JS. Comparison of electromyography and joint moment as indicators of co-contraction. J Electromyogr Kinesiol. 2012

Kvist J, Gillquist J. Sagittal plane knee translation and electromyographic activity during closed and open kinetic chain exercises in anterior cruciate ligament-deficient patients and control subjects. Am J Sports Med. 2001; 29:72–82. [PubMed: 11206260]

Lafortune MA, Cavanagh PR, Sommer HJ 3rd, Kalenak A. Three-dimensional kinematics of the human knee during walking. J Biomech. 1992; 25:347–357. [PubMed: 1583014]

Li G, DeFrate LE, Sun H, Gill TJ. In vivo elongation of the anterior cruciate ligament and posterior cruciate ligament during knee flexion. Am J Sports Med. 2004; 32:1415–1420. [PubMed: 15310565]

Li G, Rudy TW, Allen C, Sakane M, Woo SL. Effect of combined axial compressive and anterior tibial loads on in situ forces in the anterior cruciate ligament: a porcine study. J Orthop Res. 1998; 16:122–127. [PubMed: 9565084]

Li G, Rudy TW, Sakane M, Kanamori A, Ma CB, Woo SL. The importance of quadriceps and hamstring muscle loading on knee kinematics and in-situ forces in the ACL. J Biomech. 1999; 32:395–400. [PubMed: 10213029]

Lohmander LS, Englund PM, Dahl LL, Roos EM. The long-term consequence of anterior cruciate ligament and meniscus injuries: osteoarthritis. Am J Sports Med. 2007; 35:1756–1769. [PubMed: 17761605]

Mesfar W, Shirazi-Adl A. Biomechanics of the knee joint in flexion under various quadriceps forces. Knee. 2005; 12:424–434. [PubMed: 15939592]

Nunley RM, Wright D, Renner JB, Yu B, Garrett WE. Gender Comparison of Patellar Tendon Tibial Shaft Angle with Weight Bearing. Research in Sports Medicine. 2003; 11:173–185.

Papannagari R, Gill TJ, Defrate LE, Moses JM, Petruska AJ, Li G. In vivo kinematics of the knee after anterior cruciate ligament reconstruction: a clinical and functional evaluation. Am J Sports Med. 2006; 34:2006–2012. [PubMed: 16885514]

Renstrom P, Arms SW, Stanwyck TS, Johnson RJ, Pope MH. Strain within the anterior cruciate ligament during hamstring and quadriceps activity. Am J Sports Med. 1986; 14:83–87. [PubMed: 3752352]

Salmon LJ, Russell VJ, Refshauge K, Kader D, Connolly C, Linklater J, Pinczewski LA. Long-term outcome of endoscopic anterior cruciate ligament reconstruction with patellar tendon autograft: minimum 13-year review. Am J Sports Med. 2006; 34:721–732. [PubMed: 16399931]

Shelbourne KD, Freeman H, Gray T. Osteoarthritis after anterior cruciate ligament reconstruction: the importance of regaining and maintaining full range of motion. Sports health. 2012; 4:79–85. [PubMed: 23016073]

Shelburne KB, Pandy MG, Anderson FC, Torry MR. Pattern of anterior cruciate ligament force in normal walking. J Biomech. 2004; 37:797–805. [PubMed: 15111067]

Shin CS, Chaudhari AM, Andriacchi TP. Valgus plus internal rotation moments increase anterior cruciate ligament strain more than either alone. Med Sci Sports Exerc. 2011; 43:1484–1491. [PubMed: 21266934]

Tashman S, Kolowich P, Collon D, Anderson K, Anderst W. Dynamic function of the ACL-reconstructed knee during running. Clin Orthop Relat Res. 2007; 454:66–73. [PubMed: 17091011]

Taylor KA, Terry ME, Utturkar GM, Spritzer CE, Queen RM, Irribarra LA, Garrett WE, DeFrate LE. Measurement of in vivo anterior cruciate ligament strain during dynamic jump landing. J Biomech. 2011; 44:365–371. [PubMed: 21092960]

Torzilli PA, Deng X, Warren RF. The effect of joint-compressive load and quadriceps muscle force on knee motion in the intact and anterior cruciate ligament-sectioned knee. Am J Sports Med. 1994; 22:105–112. [PubMed: 8129092]

Wu JL, Hosseini A, Kozanek M, Gadikota HR, Gill T.J.t. Li G. Kinematics of the anterior cruciate ligament during gait. Am J Sports Med. 2010; 38:1475–1482. [PubMed: 20442323]

Yu B, Garrett WE. Mechanisms of non-contact ACL injuries. Br J Sports Med. 2007; 41(Suppl 1):i47–51. [PubMed: 17646249]

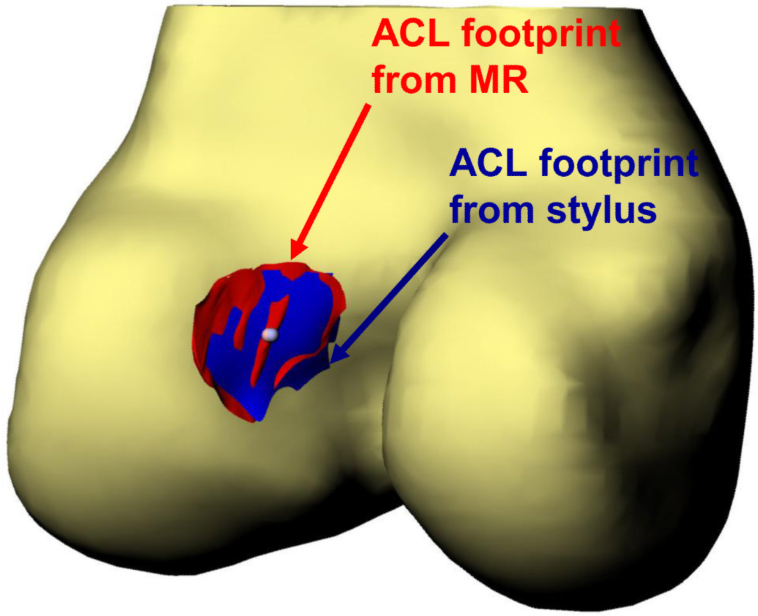

Figure 1.
Cadaveric bone models created using either the MR images or digital stylus were aligned via the iterative closest point technique. Comparing the ACL footprints associated with each model revealed that their centroids (represented by spheres) were within 0.3±0.2mm (Mean ±SD) of each other.

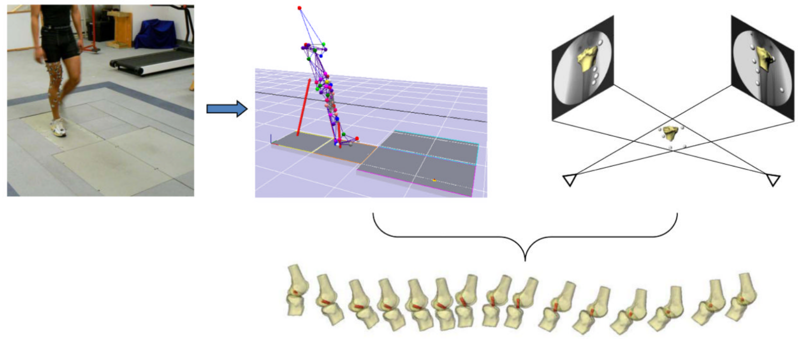

Figure 2.

Forty-four markers were placed on each subject, with twenty-eight on their dominant thigh and shank, before they walked at a self-selected pace (left). Motion capture data from the gait cycles (center) were combined with a marker registration acquired from biplanar fluoroscopy (right) using numerical optimization to recreate the positions of the 3D knee and ACL model throughout gait (bottom).

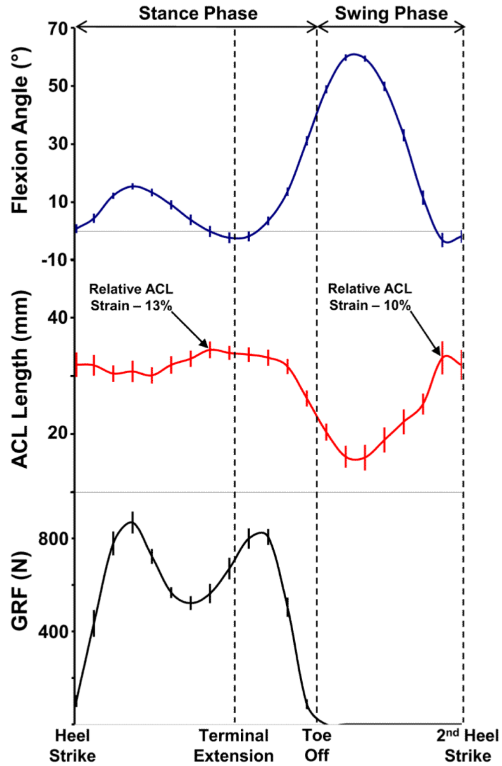

Figure 3.
Motion capture data were collected from eight subjects each performing four individual trials, such that the averaged curves shown represent thirty-two total trials (Mean±95% CI). ACL strain peaked to 13±2% during stance phase when knee flexion angles were near minimum and the heel began to rise. A second localized peak of 10±7% occurred during swing phase when the knee was most extended just prior to second heel strike.

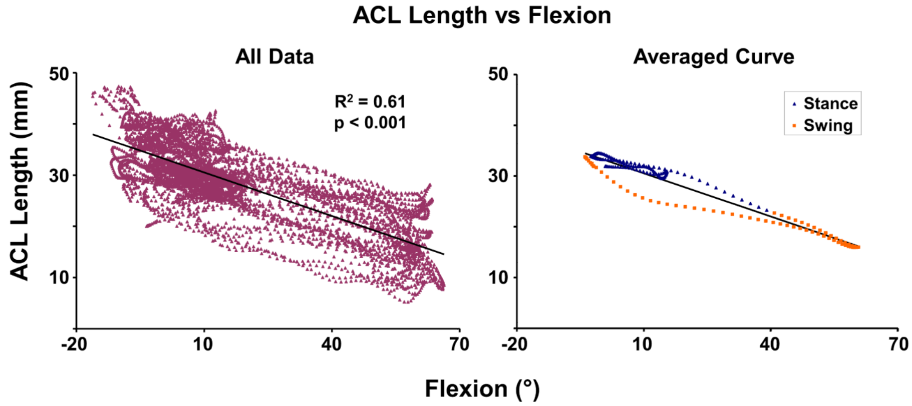

Figure 4.
Regression analysis of ACL length data versus knee flexion angle revealed a strong inverse relationship (left, R^2=0.61, p<0.001). Examining the averaged data curve (right), a closed loop pattern emerged that demonstrates ACL function being further dependent on if the limb is under stance or swing phase at similar flexion angles.