DEVELOPING SUBJECT SPECIFIC METHODS FOR KNEE JOINT INJURY DETECTION USING FINITE ELEMENT ANALYSIS

A Dissertation Presented

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Alexander Dickerson Orsi

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Anterior cruciate ligament (ACL) tear occurs upwards of 400,000 times annually in the U.S. Reconstructive surgery and rehabilitation combine to create an annual $1 Billion expense. Associated cartilage damage leads to degenerative osteoarthritis. Injury mechanism theories include risk factors such as specific motion combinations, unbalanced musculature, intercondylar notch impingement, and gender. Despite ongoing research into injury prevention, injury rates have not improved. Better understanding the injury mechanisms is important to improve medical procedures, and reduce rehabilitation costs.

Subject specific injury analysis can determine motion combinations leading to soft tissue injury for individual patients. The importance of individualized injury detection stems from the unique tissue geometries seen in each patient. Subject specific modeling provides a way to account for these irregularities. This thesis describes techniques developed to investigate subject specific knee joint injury using finite element (FE) analysis.

3D FE knee joint models were developed to determine the combinations of motion leading to ligament and soft tissue injury. ACL tear and articular cartilage injury were monitored over a spectrum of knee joint motions. The relationship between femoral axial rotation and frontal plane valgus/varus rotation was determined for both injuries. External femoral rotation increased susceptibility for ligament failure compared to internal rotation. Frontal plane knee angles at ACL injury were lower in varus than in valgus.

Surgical variations during ACLR are thought to affect success rates by increasing intercondylar notch impingement. Understanding how surgical variations affect impingement will provide insight into the high re-injury rates. A study was performed to determine how variations in graft size and insertion site location affect impingement. Several simulations were conducted using three ACL graft sizes and polar arrays of tibial and femoral insertion sites. Larger graft sizes, antero-lateral tibial insertion site shift and anterior-proximal femoral insertion site shift increased impingement.
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Chapter 1

Introduction, Literature Review and Research Aims
1.1 Introduction

The knee joint is the most injured joint in the human body. Injuries to the soft tissues of the knee joint, such as ligament and cartilage, can be very debilitating. These injuries can be influenced by several factors including knee joint abnormalities, gender, various sports activities, or surgical reconstruction procedures. This thesis explores the effects of various imposed knee joint kinematics on anterior cruciate ligament (ACL) injury and concomitant articular cartilage damage. Furthermore, this thesis will investigate how surgical variability during ACL reconstruction may increase the likelihood of ACL re–injury.

1.2 Knee Joint Anatomy

The knee is composed of two joints; the patellofemoral joint and the tibiofemoral joint, and four bones; the femur, the tibia, the patella and the fibula, seen in Figure 1.1a. The patellofemoral joint includes the patella and the femur. The patella is a sesamoid bone located between the distal insertion of the quadriceps muscle group and the superior insertion of the patellar ligament. The patella articulates with the trochlear groove of the femur and its function is to increase leverage for the quadriceps muscle group during knee extension (Goldblatt and Richmond, 2003).

The tibiofemoral joint includes the proximal end of the tibia and the distal end of the femur. This joint is subjected to motion on three axes; flexion–extension, varus–valgus, and internal–external rotation, seen in Figure 1.1b. The distal end of the femur includes the larger medial condyle and the smaller lateral condyle. The
proximal end of the tibia is comprised of two tibial plateaus (medial and lateral). At the tibiofemoral joint, the condyles of the femur articulate with their respective tibial plateau counterparts (Goldblatt and Richmond, 2003).

The fibula is the smaller of the lower leg bones, located lateral to the tibia. This bone supports a small amount of load compared to the tibia (Takebe et al., 1984). However it serves as a muscle attachment and lateral collateral ligament (LCL) insertion location.

![Figure 1.1: a)Anterior view of left knee. Femur, tibia, patella and fibula highlighted. b) Exploded view of left knee showing motions of the knee joint. Frontal plane motion is known as Varus/Valgus motion. Sagittal plane motion is Flexion/Extension. Transverse plane motion is Internal/External rotation.](image)

1.2.1 Ligaments

Ligaments are fibrous collagenous bundles which connect bones together and function to stabilize and constrain joint motion. The construction of ligament varies widely based on individual characteristics, as well as the function and the location of the
ligament. In general, ligament consists of 60–80% water. The remaining solid structures consist of 70–80% collagen (90% type I, 10% type III), and smaller amounts of elastin. Proteoglycans exist in the extracellular matrix of ligament, with a function of regulating the movement of molecules through the matrix (Simon, 1994).

There are four main ligaments within the knee joint; the anterior cruciate ligament (ACL), the posterior cruciate ligament (PCL), the lateral collateral ligament (LCL) and the medial collateral ligament (MCL), seen in Figure 1.2. Each ligament functions as a kinematic stabilizer for different knee joint motions.

The ACL is a major motion constraining ligament of the knee. The ACL originates on the lateral wall of the femoral intercondylar notch and inserts anterior and medial to the tibial eminence (Amis and Dawkins, 1991; Arnoczky, 1983; Girgis et al., 1975; Takahashi et al., 2006). The main function of the ACL is to resist anterior tibial displacement. The secondary function of the ACL is to resist excessive axial rotation between the femur and tibia. The ACL is constructed of two main bundles, the anteromedial bundle (amACL) and the posterolateral bundle (plACL) (Butler et al., 1980; Fukubayashi et al., 1982; Girgis et al., 1975; Grood et al., 1981; Markolf et al., 1976). These bundles work together, with the plACL tight in extension and slackening during flexion, and the amACL less tight in extension, and tightening during flexion.

The PCL originates on the medial condyle of the femur, and inserts posteriorly and medially between the tibial plateaus. The PCL restricts posterior translation of the tibia. Similarly to the ACL, it has a secondary function of resisting axial rotation between the femur and tibia.

The MCL is located on the medial side of the knee. The MCL originates on the
Figure 1.2: Anterior view, right knee. Diagram identifying the four main ligaments of the knee joint (Media, 2009).
femur at the medial epicondyle, and inserts on the medial tibial surface. The primary function of the MCL is to resist valgus motion. A secondary function of the MCL is to resist axial rotation and medial–lateral tibiofemoral translation.

The LCL is located on the lateral side of the knee. The LCL originates on the femur at the lateral epicondyle, and inserts on the lateral side of the fibular head. The primary function of the LCL is to resist varus motion. A secondary function of the LCL is to resist axial rotation as well as anterior–posterior tibiofemoral translation.

1.2.2 Articular Cartilage

Articular cartilage is a smooth soft tissue located at the joint surfaces of bones (Martin et al., 1998). Articular cartilage acts as a bearing surface for bones by providing a near frictionless interaction at joint surfaces. The cartilage provides lubrication and prevents joint wearing. Articular cartilage is composed of approximately 70% water by mass. The remaining matrix is composed of approximately 40–70% collagen, and 15–40% proteoglycan. Less than 5% of the cartilage volume is made up of chondrocytes, the cell type involved in cartilage extracellular matrix production and maintenance.

The primary type of collagen existent in articular cartilage is type II collagen. The alignment of collagen fibers changes throughout the thickness of the articular cartilage. At the surface, and just below in the tangential zone, the fibers are parallel to the articular surface. In the deeper regions of the cartilage, the fibers transition to an orientation normal to the surface. This is seen in Figure 1.3.

The other main polymer within the articular cartilage is proteoglycan.
Figure 1.3: Diagram explaining the structure of the articular cartilage (Martin et al., 1998).

can is composed of a hyaluronic acid backbone with glycosaminoglycan (GAG) chains attached. The aggregate molecules of proteoglycan, *aggrecan*, provide the ability for articular cartilage to resist compressive loading. This is due to the proteoglycans inherent hydrophilicity due to their negative charge. This hydrophilicity prevents water from flowing out of the cartilage matrix during compression. Because water is retained during compression, the collagen molecules experience tension, allowing them to resist the compressive load (Martin et al., 1998).

Chondrocytes are the primary cell type existent in the articular cartilage. These cells regulate the maintenance of the extracellular matrix. Chondrocytes produce collagen and proteoglycan molecules to remodel the cartilage due to damage from joint loading. Due to the avascular nature of cartilage, the ability for self repair is limited. Permanent articular cartilage damage occurs when the range of healthy loading conditions is exceeded, leading to osteoarthritis (OA) later in life (Martin et al., 1998).
1.2.3 Meniscus

The menisci of the knee joint are wedge shaped fibrocartilaginous structures located on both the medial and lateral tibial plateaus, seen in Figure 1.4. They are attached to the tibia via the tibial intercondylar fossae. The lateral meniscus has a more uniform cross section and a more circular shape than the medial meniscus, which has a wider cross section in the posterior horn and is semi-circular in shape (AufderHeide and Athanasiou, 2004).

![Superior View: Right Tibial Plateau](image)

Figure 1.4: Superior view, right tibial plateau. Lateral and medial menisci shown (Media, 2015).

The menisci are made up of approximately 70% water. The remaining mass is approximately 98% type I collagen, with trace amounts of type II collagen, and 2–3% GAG (Fithian et al., 1990). The meniscus contains a superficial layer of a mesh of collagen fibers, a thin layer of radially aligned fibers, and a deep region which contains circumferentially aligned fibers of collagen type I. This complex organization is shown
in Figure 1.5 (AufderHeide and Athanasiou, 2004).

Figure 1.5: Cross section view of wedge shaped meniscus. Detailed fiber orientations for various layers (AufderHeide and Athanasiou, 2004).

The menisci serve to disperse the compressive load between the femur and tibia. Due to their wedge shaped cross section and their circumferential alignment, the menisci are strained in the radial direction during tibiofemoral compression. This radial strain loads the circumferentially aligned fibers into tension and increases the resistance to compressive loads at the knee joint.

1.3 Injury Mechanisms and Surgical Interventions

1.3.1 ACL Injury

ACL injury occurs when the ligament is partially or completely ruptured due to excessive loading. ACL injuries are classified by two mechanisms: “contact” and “non–contact”. A contact ACL injury results from direct contact with another person or object. Non–contact ACL injuries result from the subjects own motion, and are not
influenced by external perturbations such as another person. ACL injuries commonly occur in non–contact scenarios (McNair et al., 1990). Several studies show non–contact ACL injury occurring with athletes landing near full knee extension, in valgus, and in femoral external rotation (ER) (Boden et al., 2000a,b; Ferretti et al., 1992; Griffin et al., 2006; Ireland, 1999; Malone et al., 1993; McNair et al., 1990). Video evidence has shown the main contributors to non–contact ACL injuries are motions in the frontal and transverse planes (Boden et al., 2000a,b; Krosshaug et al., 2007; Olsen et al., 2004). These studies also described a deceleration closed chain mechanism associated with ACL injury, with the knee reported to be between $20^\circ$ – $30^\circ$ of knee flexion (Besier et al., 2001; Boden et al., 2000a,b; Ireland, 1999; McNair et al., 1990).

### 1.3.2 Bone Bruising, Cartilage Damage and Osteoarthritis

Concomitant bone bruising has been reported in over 80% of ACL ruptures (Dunn et al., 2010; Illingworth et al., 2014; Johnson et al., 1998; Murphy et al., 1992; Rosen et al., 1991; Speer et al., 1992, 1995; Spindler et al., 1993). These injuries are found primarily on the posterior lateral tibial plateau, as well as the lateral femoral condyle (Johnson et al., 1998; McCauley et al., 1994). Due to the location of these injuries, valgus loading is thought to be a major influencing factor because the lateral condyles of the femur and tibia compress on one another during valgus. These combined ACL–articular cartilage injuries are known to lead to OA later in life (Faber et al., 1999; Johnson et al., 1998; Vellet et al., 1991).

OA, a degenerative disease of the articular cartilage, is the most common joint
disease affecting nearly 15% of the United States. OA occurs due to an imbalance between the catabolic and anabolic activities of chondrocytes. Alteration in chondrocyte metabolism leads to the degradation of the cartilage extracellular matrix, weakening the cartilage thus causing OA (Griffin and Guilak, 2005). This disease is linked to excessive loading at the knee joint. During athletic maneuvers, it is reported that compressive forces at the knee joint can be up to 24 times body weight (Arokoski et al., 2000). Magnitude and loading cycles as well as cartilage stress and strain may all affect the progression of OA (Yang, 2009). Several studies report cartilage damage linked to certain stress, strain and/or loading thresholds (Atkinson et al., 1998; Chen et al., 2003; Heiner and Martin, 2004; Langelier and Buschmann, 2003; Mansour, 2003; Morel and Quinn, 2004). Other studies report OA occurring at both high and low load bearing regions within the knee joint articular cartilage (Akizuki et al., 1986). This variability is most likely due to experimental limitations such as ex–vivo testing, and sample to sample variations. Care must be taken selecting a failure criterion for the articular cartilage due to the wide range of reported values.

1.3.3 ACL Reconstruction and Notchplasty

Up to 90% of ACL injured patients elect to have anterior cruciate ligament reconstruction (ACLR). ACLR can be performed using a bone–patellar tendon–bone (BPTB) graft seen in Figure 1.6a, or using an anatomical double bundle reconstruction performed with a hamstring graft seen in Figure 1.6b (Longo et al., 2008; Meredick et al., 2008; Sajovic et al., 2006). Various types of grafts can be used during reconstruc-
tion. An autograft is taken from the patient, while allografts are taken from a donor. Autograft is considered the safest and fastest healing tissue that can be used. However if an autograft is selected, there are two surgical sites, one for harvesting and one for surgical reconstruction, which may extend hospital stays for patients due to extra healing time. Allograft takes longer to incorporate into the patient, however there is only one surgical site. A difference in ACL reinjury rate is reported when comparing autograft and allograft techniques (Bartlett et al., 2001; Ellis et al., 2012; Vishal et al., 2010). Ellis et al. (2012) reported that 35% of patients who underwent allograft BPTB replacement required revision surgery within one year, compared to 3% for patients who had autograft replacement. Vishal et al. (2010) reported that with a mean follow up of 49 months, 0.7% of patients who had an autograft BPTB replacement required revision surgery compared to 9.7% for patients who had an allograft BPTB replacement.

Several review papers have reported no significant benefit exists from using a double bundle hamstring or quad tendon graft, and recommend the use of a single bundle BPTB graft (Longo et al., 2008; Meredick et al., 2008; Sajovic et al., 2006).
Reinjury rate has been reported as high as six times greater in ACLR patients compared to healthy patients (Paterno et al., 2014). A study by Wright et al. (2007) reported 6% of patients who underwent ACLR had a reinjury within two years. Another study by Salmon et al. (2005) reported 12% of patients with an ACL replacement had a reinjury within five years. Leys et al. (2012) performed a 15 year follow up of the same population from the Salmon et al. (2005) study in which a 34% reinjury rate was reported. Several factors may play a role in the high revision rates associated with ACLR. Inaccurate placement of the graft tunnel locations has been reported (Musahl et al., 2003, 1999). A range of ACL graft sizes has also been reported for use during ACL reconstruction (Magnussen et al., 2012; Tuman et al., 2007; Wallace et al., 1997; Wilson et al., 1999). Understanding how these surgical variations affect the surgical revision rate can help improve future surgical outcomes.

Impingement can occur between the ACL and the intercondylar notch wall during
Figure 1.7: Notchplasty during ACLR. a) Osteotome is inserted endoscopically. b) Osteotome is turned parallel to the intercondylar notch wall. c) Notchplasty is performed with the osteotome. d) A motorized bur reshapes the notch wall (Mann et al., 1999).

Knee joint motion (Boden et al., 2000a,b; Dienst et al., 2007; Fung et al., 2007; Fung and Zhang, 2003; Park et al., 2010). This impingement is thought to lead to ACL injury (Boden et al., 2000a,b; Dienst et al., 2007). Femoral notchplasty is a procedure in which the intercondylar notch is reshaped and/or widened during ACLR to prevent graft impingement with the notch. The process of notchplasty is displayed in Figure 1.7. Notchplasty is common, however a standard protocol has yet to be defined (Berg, 1991; Hame et al., 2003; Markolf et al., 2002).
1.4 Related Work in Biomechanical Finite Element Analysis

Finite element analysis (FEA) for biomechanics is a growing field. While cadaver testing is the gold standard for studying soft tissue mechanics, computational models are improving, and are used more widely due to the limitations of cadaver testing. Knee joint biomechanics has been of high interest as the tissues within the knee joint are some of the most commonly injured (Boden et al., 2000b; Fleming et al., 2010; Nau and Teuschl, 2015; Smith et al., 2012). Finite element (FE) models provide non-invasive predictive modeling capabilities for soft tissue. These models are able to simulate how knee joint soft tissue reacts to specific loading conditions. Computational models allow for subject specific investigations, which is important because individual subjects have unique bone and soft tissue geometries (Bisson and Gurske-DePerio, 2010; Hashemi et al., 2010; Simon et al., 2010).

1.4.1 Literature Review

Over the past several decades, the body of literature has been growing in the field 3D FE knee joint modeling. Several studies have been performed outlining the techniques used to develop these 3D models (Bendjaballah et al., 1997; Boyd et al., 2012; Gardiner and Weiss, 2003; Homyk et al., 2012; Kiapour et al., 2014; Limbert et al., 2004; Mesfar and Shirazi-Adl, 2006; Papaioannou et al., 2010, 2008; Park et al., 2010; Peña et al., 2006; Song et al., 2004; Wang et al., 2014; Wangerin, 2013). The purpose of these investigations has been to evaluate the stresses and strains of various soft
tissues within the knee. Various loading conditions have been investigated, and a wide variety of material models and material properties have been used to describe the different soft tissues of the knee.

Bendjaballah et al. (1997) performed a study using a 3D FE knee joint model to investigate how collateral ligament deficiency affects cruciate ligament forces and cartilage and meniscal stresses during valgus and varus knee joint motions. This study was one of the first studies to incorporate all of the knee joint soft tissue structures, as well as contact between the structures. 3D structures for the articular cartilage and menisci were incorporated, while the ligaments were modelled as multi-bundle spring element structures. Several model configurations were constructed by selectively removing knee joint structures. One configuration kept the knee joint intact. One configuration removed the MCL during a valgus torque. Another configuration had the LCL removed during varus torque. The aim was to investigate how the removal of specific tissues affect knee joint kinematics during valgus and varus torque application. The load in the ACL was shown to increase by up to 160% due to the removal of the LCL. The results suggested that the collateral ligaments are the primary restraints against valgus and varus moments at the knee joint (Bendjaballah et al., 1997).

Donahue et al. (2002) investigated a new construction technique for 3D FE knee joint models. Their goal was to use a digitized laser scanning method to accurately characterize the geometries of the meniscus. Using this model, the researchers were interested in investigating the validity of modelling bones as rigid bodies. More importantly, the focus of the research was to understand how properly constraining a
FE knee joint model affects the contact stress distribution during compressive loading. Digitized CT images were used to create solid models of the bones. The laser scanned surfaces were converted to solid structures for the soft tissue. Cartilage was modelled isotropic elastic, and the meniscus was modelled as transversely isotropic elastic. Bone was modelled as both rigid, as well as orthotropic elastic. Several configurations of body weight loading were simulated with different kinematic constraints applied to the model. It was determined that modelling the bone as rigid did not significantly change the results, and drastically reduced computational time. It was also determined that by varying the constraints on the knee joint, significant changes in contact pressures were seen. For example, when valgus/varus motion was constrained a 19% decrease in maximum pressure was seen in the medial compartment during body weight loading. The key findings from this study were that modelling bones as rigid is a valid assumption when studying the soft tissue of the knee joint using a FE model and that soft tissue contact pressures are highly sensitive to the kinematic constraints applied to FE knee joint models (Donahue et al., 2002).

Gardiner and Weiss (2003) performed a subject specific study in which they constructed several FE models from cadaver knees to study the stress–strain behavior of the medial collateral ligament (MCL). Eight individual 3D FE models of the MCL, femur and tibia were developed using volumetric CT images. Unique material properties for each MCL specimen were obtained through uniaxial testing, and were applied to each individual FE model. Each knee underwent valgus loading at different knee flexion angles during which kinematic and strain data was collected. Experimental validation was performed by comparing experimentally obtained MCL strain data to
the strain data predicted in the FE simulations during valgus loading. The results of this study showed that the complex non-uniform in-vivo strain fields seen in the MCL under valgus loading can be accurately predicted using FE techniques (Gardiner and Weiss, 2003).

Limbert et al. (2004) investigated the effect of prestress on the ACL during passive flexion. They used a FE model of a single bundle ACL with two configurations, one with and one without prestrain. Passive knee flexion was applied to the knee joint through the femur, with a fixed tibia. Uniform prestretch was applied to the ACL which the authors state as a limitation. This was done by applying an identical amount of stretch to each element. This is not physiologically accurate, as ligament fibers are heterogeneous, and the stretch is likely varied based on the location within the ligament fiber bundles. The results of their study closely resembled results from cadaver testing in terms of the resultant force in the ligament during passive knee flexion. Their results also support the use of prestretch in an ACL FE model, as this better simulated the mechanical response of the ligament during knee joint motion (Limbert et al., 2004). The authors also state that their results support the theory that the ACL does not have a stress free state throughout the flexion cycle.

Song et al. (2004) performed a study using an FE model of a double bundle ACL structure. The goal of this study was to investigate the forces and stress distributions in the ACL bundles during anterior tibial loading. An anterior force of 134 N was applied to an intact cadaveric knee specimen, and the resulting kinematic data was recorded. The cadaver specimen was then dissected, leaving only the bony structures and the ACL. The recorded kinematic data from the intact cadaver specimen was
applied to the dissected specimen, and the resultant forces in the ACL were recorded. A subject specific FE model was constructed from radiographic data of the dissected knee, and the same kinematic data was applied. The ACL force data compared well between the FE model and the dissected cadaver knee specimen. This study provided a methodology validating the use of a simplified FE model of the ACL (Song et al., 2004). The authors state that the methods provide relevant data which can be used to improve ACL replacement technologies. The authors mention that by expanding these projects to larger populations, a better understanding of ACL interactions will improve surgery and rehabilitation protocols.

Mesfar and Shirazi-Adl (2006) investigated how changes to the material properties and the amount of pretension for the cruciate ligaments affect the biomechanics of the knee joint. A 3D FE model of the human knee joint was constructed including the femur, patella and tibial articular cartilages, menisci, as well as the cruciate, collateral and patellar tendon and ligaments. Several simulations were performed of 90° of knee flexion using different ACL and PCL material properties. Varying amounts of prestrain were applied to the ACL (+6% and −6%) and PCL (+6% and +10%). Patellar tendon material properties were also used for the ACL and PCL as well. The results highlighted that incorrectly applied ligament prestrain can significantly affect the natural motion of the knee joint. The study also recommends that if stiffer materials are to be used for an ACL replacement graft, such as the patellar tendon, then less prestrain should be applied to reduce adverse changes to knee joint biomechanics (Mesfar and Shirazi-Adl, 2006).
Peña et al. (2006) performed an investigation on how combined loading simulations affect meniscal and ligament stress. The purpose of this study was to understand how the menisci and ligaments stabilize the knee joint during complex loading scenarios. Two simulations were performed to compare results with other investigations. Simulation one included a combined tibiofemoral compression of 1150N, and a 134N anterior–posterior load applied to the femur. Simulation two included the same conditions as simulation one, combined with a 10N·m valgus torque. The FE model was validated based on several experimental and numerical studies. The results supported the theory that the MCL is the main restraint to valgus rotation. The results also showed that valgus loading decreased load transmission to the medial meniscus by 43%, similar to other numerical studies. This study provided a complete knee model which was validated by numerical and experimental studies. The results showed how the individual knee joint soft tissue structure play a role in restraining different loading conditions (Peña et al., 2006).

Kiapour et al. (2014) developed a 3D FE model of a knee subjected to different loading conditions. The model incorporated 3D deformable solid structures for all soft tissues. Their model was validated against several cadaveric kinematic conditions, with good correlation seen for ligament stresses and strains, as well as cartilage pressures. Rate dependent material properties were included, and a range of loading rates were validated against as well. They demonstrated their FE models accurately predict soft tissue stresses and joint kinematics. The authors state that their model will be used to determine risk factors for ACL injury in the future (Kiapour et al., 2014).
Park et al. (2010) performed a study to investigate how tibiofemoral kinematics affect ACL–intercondylar notch impingement using a 3D FE model of the ACL. The purpose was to identify whether impingement could be a factor leading to ACL injury. A cadaver experiment was performed using a knee joint specimen to record ACL–intercondylar notch impingement during knee joint motion simulation. The cadaver was moved from an initial position of 45° of flexion, 0° valgus, and 0° external tibial rotation to an end position of 45° of knee flexion, 10° of valgus and 30° of tibial internal rotation. ACL–intercondylar notch impingement was recorded using flexible pressure sensor. A 3D FE model of the knee joint including the femoral and tibial surfaces and a 3D ACL were constructed from a digitizing probe. The kinematics from the cadaver experiment were applied to the FE model as boundary conditions, and the contact area and impingement force results were compared. The FE model accurately predicted the impingement force and contact area from the cadaver experiment, validating the model as a useful tool for determining ACL intercondylar notch impingement. The authors claim that future work using similar models will help uncover ACL failure mechanism related to impingement (Park et al., 2010).

Homyk et al. (2012) performed a study to determine the failure locus of the ACL using a 3D FE model of the human knee joint. The purpose of this study was to determine the various motions associated with ACL injury. A FE model of the knee joint was constructed using solid structures for the bones, cartilage, and menisci. Nonlinear spring elements were used to describe the ligament structures. A parametric analysis of knee joint motion was performed using varying degrees of internal/external rotation and varus/valgus motion. Knee flexion was held at 25° as this is known to
be associated with ACL injury. The results showed that less varus angle was needed to reach ACL failure compared to valgus knee angle. The results also showed that external femoral rotation increased the likelihood of ACL rupture. At the time of ACL failure, cartilage stresses were higher on the lateral tibial cartilage during valgus motion loading compared to the medial tibial cartilage during varus loading. The study was the first attempt at determining the relative dangers for ACL and cartilage injury between different knee joint motions (Homyk et al., 2012). The present thesis is a continuation of this work, with several notable improvements which will be outlined in the future chapters.

1.4.2 Material Modelling

Several material models have been proposed for the bone and soft tissue of the knee joint using the finite element method. The material properties used throughout this thesis were taken from several previous publications (Blankevoort et al., 1991; Donahue et al., 2003, 2002; Fung and Zhang, 2003; Kiapour et al., 2014; Peña et al., 2006; Puso and Weiss, 1998; Wangerin, 2013; Yang et al., 2010). Different models exist to model each tissue type. Material model selection is a process which should consider several factors such as loading scenario of interest, accuracy of results, and computational efficiency. The material model selection process for the knee joint tissues used in this thesis is outlined below.
Bone

While bone is a deformable material it can be modelled as rigid to decrease computational time. Previous work determined that modeling bone as rigid versus a combination of isotropic and orthotropic elastic did not affect cartilage contact results. The analysis showed that less than 2% difference across all cartilage contact variables occurred when modelling bone as rigid compared to modelling bone as deformable (Donahue et al., 2002). For this reason, bone was modeled as rigid throughout the investigations in this thesis.

Cartilage

Cartilage is known to be a viscoelastic and incompressible material. However, depending on the loading conditions of interest, it may not be necessary to model the cartilage with time dependent properties. Because the loading conditions used in this thesis are short duration, it is acceptable to model the cartilage as isotropic elastic, as the relaxation time constant of \textit{in–vivo} cartilage is roughly $1,500 \text{sec}$ (Armstrong et al., 1984; Donzelli et al., 1999; Eberhardt et al., 1990; Kiapour et al., 2014). A purely incompressible material would exhibit a poisson ratio of 0.50. Due to the limitations of FE software at avoiding volumetric locking, incorporating a high poisson ratio of $(0.45–0.49)$ is sufficient for modelling the incompressibility of cartilage (Donahue et al., 2003; Kiapour et al., 2014; Peña et al., 2006; Yang et al., 2010).
**Meniscus**

Due to the shape and curvature of the meniscus, the preferred fiber direction is aligned circumferentially. To model this, a transversely isotropic elastic material model can be used by defining a cylindrical coordinate system. The preferential fiber direction is then applied along the circumferential direction of each meniscus. Figure 1.8 shows an axial view of the tibial plateau with the circumferential fiber direction of the menisci. The menisci are attached to the tibial plateau at the meniscal horns using linear spring element sets, similar to previous studies (Donahue et al., 2003; Homyk et al., 2012; Orsi et al., 2015; Yang et al., 2010), seen in Figure 1.8. At each horn attachment 10 linear springs were used to attach the meniscal horn to the tibial plateau. The transverse ligament is modeled as a single spring element, which attaches the anterior horns of the menisci to each other (Donahue et al., 2003; Homyk et al., 2012; Orsi et al., 2015; Yang et al., 2010).

**Ligament**

Ligament is a highly nonlinear material which can not support compressive loading. This can be modelled in several ways, using continuum solid elements or nonlinear spring elements. One of the first ligament models was developed by Blankevoort et al. (1991) who defined a nonlinear spring model for ligaments. Several hyperelastic constitutive models have been developed to characterize the nonlinear material properties of ligaments (Limbert et al., 2004; Peña et al., 2006; Weiss et al., 1996). Throughout this thesis, the ACL was modelled as a double bundle solid structure
Figure 1.8: Superior axial view of tibia. Preferential fiber direction for meniscus shown in the circumferential direction. Meniscal horns and transverse ligament defined using linear spring elements.
using the constitutive model developed by Weiss et al. (1996). Fiber direction was
defined as the vector from the femoral origin to the tibial insertion for each bundle.
The material coefficients were extracted from Peña et al. (2006), who curve fit cadav-
eric test data to the Weiss et al. (1996) model. The other ligaments (PCL, MCL, and
LCL) were modelled as multi bundled nonlinear spring elements using the material
properties from Blankevoort et al. (1991).

1.5 Unexplored Research

Individual subjects exhibit unique geometries for their soft tissue structures which
may affect their risk of injury. Until now, no studies have investigated subject specific
injury detection using FE modelling. Using individualized FE knee joint models,
methods can be developed to investigate subject specific injury mechanisms. By
performing parametric analyses of different knee joint kinematic combinations, these
FE models can be used to virtually diagnose ACL injuries and concomitant articular
cartilage damage (Homyk et al., 2012; Orsi et al., 2015; Yang et al., 2010).

Ligament is known to be a rate dependent material (Puso and Weiss, 1998). No
studies were found analyzing different knee joint loading rates to understand the effect
rate dependency has on ligament injury. It is important to understand if viscoelastic
material properties are necessary to model these injuries as they occur during dynamic
sports motions. Doing so will help highlight detrimental motion combinations leading
to ACL injury. These types of investigations are valuable for the orthopedic and sports
medicine communities as they improve the knowledge of knee injury mechanisms.
A high percent of anterior cruciate ligament reconstruction surgeries (ACLR) require revision surgeries. ACL–intercondylar notch impingement is thought to be a leading factor to ACL reinjury. No studies have investigated the affects of surgical variations on ACL–intercondylar notch impingement. It is known that variability exists in graft selection, and both tibial and femoral graft insertion sites location during ACLR. Understanding how these variations affect ACL–intercondylar notch impingement will help improve surgical outcomes.

Notchplasty is an unregulated procedure, commonly performed based on the preference of the surgeon. Recommendations are needed to develop a proper protocol for this procedure. Until now, FE modelling has not been used to investigate the affects of notchplasty on ACL–intercondylar notch impingement. By understanding how notchplasty affects ACL–intercondylar notch impingement recommendations can be made for this procedure to improve ACLR and help reduce revision surgeries.

1.6 Research Aims

The aim of this research is to answer the following three important questions. First, which motions are most detrimental to the ACL and tibial articular cartilage? A method will be developed to determine the failure locus for the soft tissue of the knee joint using a subject specific model. This will identify high risk motions for ACL and articular cartilage injury. Second, what is the affect of a rate dependent material model on the ACL failure locus? Ligament is known to be viscoelastic, therefore it is necessary to understand how this affects ligament failure. A transversely isotropic
visco–hyperelastic material model will be incorporated for the ACL and a range of physiological loading rates will be simulated to determine if viscoelasticity is necessary for modelling ACL injury. Third, how do variations during ACLR such as graft size and both tibial and femoral insertion site location affect ACL–intercondylar notch impingement? Large variations in graft size have been reported from $6mm - 11mm$ in diameter. It has also been reported that experienced surgeons are capable of errors up to $3mm$ when locating the tibial and femoral insertion sites. Understanding how these factors affect ACL–intercondylar notch impingement will provide insight into the high revision surgery rates, and may support the use of surgical notchplasty procedures.

By understanding the motions leading to ACL and cartilage injury, and understanding how sensitive ACLR is to surgical variations, clinicians will better understand the mechanisms of these injuries and will be better prepared for surgical repairs. These questions will be addressed with the following three Specific Aims.

**Aim 1.** Develop a failure locus to understand the relative dangers between different knee motions for ACL and articular cartilage injury.

Within this aim, the following items will be addressed:

- Develop a failure locus for a 3D ACL
- Investigate concomitant cartilage damage during ACL injury
- Evaluate a simplified FE model for developing a failure locus for the ACL

**Aim 2.** Determine how a rate dependent material model affects the ACL failure locus.
Within this aim, the following items will be addressed:

- Incorporate viscoelastic material properties for the ACL
- Evaluate the effects of the viscoelastic material model on the ACL failure locus

**Aim 3. Understand how surgical variations during ACL reconstruction affect ACL–femoral intercondylar notch impingement.**

Within this aim, the following items will be addressed:

- Investigate how graft selection during ACLR affects ACL–intercondylar notch impingement
- Investigate how insertion site location during ACLR affects ACL–intercondylar notch impingement
- Simulate and provide recommendations for notchplasty during ACL reconstruction

### 1.7 Importance and Application

ACL injuries occur up to 400,000 times annually in the United States which puts a massive burden on the healthcare system as they are linked to articular cartilage and meniscal injuries (Boden et al., 2000a,b; Feagin Jr and Lambert, 1985; Fleming et al., 2010; Griffin et al., 2006; Illingworth et al., 2014; Johnson et al., 1998; Murphy et al., 1992; Nau and Teuschl, 2015; Rosen et al., 1991; Smith et al., 2012; Spindler et al., 1993). Nearly 50% of ACL injured knees develop early onset OA (Boden
et al., 2000b; Fleming et al., 2010; Hall et al., 2012; Lohmander et al., 2004; Nau and Teuschl, 2015; Scanlan et al., 2010; Smith et al., 2012). According to a recent study, OA has accounted for nearly $200 Billion in combined medical costs between insurers and out of pocket expenses annually in the Unites States alone (Kotlarz et al., 2009). With a healthy young population, the prevalence of these injuries and the associated damaging affects will continue to rise. A better understanding of how ACL injury and articular cartilage damage are related will improve treatment and prevention programs. With such a heavy burden on the health care system, this research has the potential to save patients and health care professionals significant time and money.

Approximately 90% of ACL injured patients elect to have ACLR, however this procedure may increase the risk of reinjury by six times (Linko et al., 2005; Paterno et al., 2014). With a high revision surgery rate, it is important to understand why so many of these reconstructions are unsuccessful. Several variables exist during the ACLR procedure, including varying graft size, graft tunnel location. Surgical notchplasty has been used during ACLR to prevent impingement, however a proper protocol remains to be documented. Understanding how surgical variability affects impingement is necessary to improve surgical outcomes and reduce revision surgeries.

FEA provides an efficient way to investigate the biomechanics of knee joint injuries and surgical interventions. Previous studies have validated FE models against experimental data, and have used these models to investigate soft tissue stress and strain. Studies have also validated FE models to predict ACL–intercondylar notch impingement. This thesis will use FE modelling to investigate several different knee joint injury mechanisms.
To date, no known studies exist investigating which knee joint motions affect ACL injury and concomitant articular cartilage damage. Along with this, no known studies have investigated the effects of surgical variations on ACL impingement. This thesis aims to develop and validate subject specific FE models to study injury mechanisms and surgical interventions for the soft tissues of the knee joint. Understanding how various risk factors affect these injuries is important for improving injury prevention. These studies address the need of understanding ACL injury and ACLR on a subject specific basis. The results will provide useful data describing the injury mechanisms, and the effectiveness of surgical interventions for the ACL. Using this data, clinical orthopaedic professionals can improve injury prevention and rehabilitation through targeted movement avoidance and muscle group training and improve surgical techniques through personalized knee joint geometry assessment. With increases in computational power, these tools will be more valuable clinically as future studies can perform more simulations efficiently, improving subject specific injury detection. Continued research is needed to better understand the mechanisms surrounding these injuries. Doing so will improve prevention and rehabilitation techniques, helping people live more active and healthy lifestyles.
REFERENCES


Chapter 2

The Effects of Knee Joint Kinematics on Anterior Cruciate Ligament Injury and Articular Cartilage Damage
2.1 Abstract

This study determined which knee joint motions lead to anterior cruciate ligament (ACL) rupture with the knee at 25° of flexion. The knee was subjected to internal and external rotations, as well as varus and valgus motion. A failure locus representing the relationship between these motions and ACL rupture was established using finite element simulations. This study also considered possible concomitant injuries to the tibial articular cartilage prior to ACL injury. For the range of motions investigated, the posterolateral bundle of the ACL demonstrated higher rupture susceptibility than the anteromedial bundle. The average varus angular displacement required for ACL failure was 46.6% lower compared to the average valgus angular displacement. Femoral external rotation decreased the average frontal plane knee angle (varus and valgus combined) required for ACL failure by 27.5% compared to internal rotation. Tibial articular cartilage damage initiated prior to ACL failure in all valgus simulations. The results from this investigation agreed well with other experimental and analytical investigations. This study provides a greater understanding of the various knee joint motion combinations leading to ACL injury and articular cartilage damage. The results of this investigation could be used by athletes and trainers to avoid certain motions leading to knee joint soft tissue injuries.

2.2 Introduction

The anterior cruciate ligament (ACL) is a major stabilizing ligament of the knee. The ACL is known to prevent anterior tibial displacement relative to the femur, and
provide torsional stability during internal and external rotations (Butler et al., 1980; Fukubayashi et al., 1982; Girgis et al., 1975; Grood et al., 1981; Markolf et al., 1976). The ACL originates anterior and medial to the tibial eminence, rising posteriorly and laterally, inserting on the lateral wall of the femoral intercondylar notch (Amis and Dawkins, 1991; Arnoczky, 1983; Girgis et al., 1975; Takahashi et al., 2006). Studies report the ACL as comprising two main bundles, the anteromedial bundle (amACL) and the posterolateral bundle (plACL) seen in Figure 2.1a (Blankevoort et al., 1991; Girgis et al., 1975; Peña et al., 2006; Sinkov et al., 2004; Steckel et al., 2007; Takahashi et al., 2006). These bundles work together with the plACL tight in extension and slackening during flexion and the amACL less tight in extension, and tightening during flexion, seen in Figures 2.1b and c. The amACL limits anterior tibial translation during higher degrees of knee flexion. The plACL limits anterior tibial translation, hyperextension, and tibial rotation relative to the femur. The amACL is prone to injury in knee flexion, while the plACL is susceptible to injury in low flexion (0°–30°) and external femoral rotation relative to the tibia. A combination of knee joint motion could cause injury to both bundles if a large enough force is applied to the knee joint (Sinkov et al., 2004).

ACL tears are painful injuries, and are reported to occur from 250,000 to 400,000 times annually in the United States (Boden et al., 2000a,b; Fleming et al., 2010; Griffin et al., 2006; Nau and Teuschl, 2015; Smith et al., 2012). Roughly 90% of ACL injured patients elect to have reconstructive surgery. This expensive procedure, combined with rehabilitation put significant burden on the health care system on the order of $1 billion annually in the U.S. alone (Boden et al., 2000a,b; Feagin Jr
Figure 2.1: a) Anterior view of ACL showing the two bundles of the ACL. b) ACL bundle alignment at knee extension. c) ACL bundle alignment during knee flexion.

and Lambert, 1985; Griffin et al., 2006). It is known that articular cartilage and meniscal injuries are commonly associated alongside ACL injury. These combined ACL/articular cartilage injuries are also known as major risk factors for degenerative osteoarthritis later in life (Faber et al., 1999; Johnson et al., 1998; Vellet et al., 1991). It is clinically important to determine which movement combinations are likely to cause injury to knee joint tissues, as an improved understanding of these injury mechanisms will improve prevention techniques, rehabilitation and surgical procedures (Renstrom et al., 2008). ACL injuries are classified by two mechanisms: “contact” and “non–contact”. ACL injuries occur more frequently in non–contact scenarios, reportedly up to 70% of the time (McNair et al., 1990). Anterior tibial shear relative to the femur is another reported ACL injury mechanism. This mechanism was not investigated in this study. This study focused on the two most prominently reported injury mechanisms, which are varus/valgus motion and internal/external tibiofemoral rotation.

Several studies report non–contact ACL injury occurring with athletes landing near full knee extension, in valgus, and in femoral external rotation (Boden et al.,
These studies also described a deceleration closed chain mechanism associated with ACL injury. The knee was reported to be within $20^\circ - 30^\circ$ of knee flexion at injury (Besier et al., 2001; Boden et al., 2000a,b; Ireland, 1999; McNair et al., 1990). Video evidence has reported the main contributors to non–contact ACL injuries are motions in the frontal and transverse planes (Boden et al., 2000a,b; Krosshaug et al., 2007; Olsen et al., 2004).

While cadaver testing is the gold standard for studying the mechanics of soft tissue, computational models are improving, and are being used more widely due to the limitations of cadaver testing. Investigating ligament and soft tissue failure for various knee joint motions using cadaveric knees would be cost and time prohibitive. A single cadaver sample provides only one failure result as the original state cannot be restored after the ACL is torn. Because of this, a number of cadavers would be required to establish an injury locus. Furthermore, developing a failure locus using multiple cadaver samples would not be advantageous as each sample would have individual geometries, creating sample-to-sample variations in the results. In contrast, finite element (FE) models can simulate multiple failures using different loading scenarios for an individual knee joint model. Computational models also provide a method to investigate subject specificity as it is known that every subject has unique bone and soft tissue geometries (Bisson and Gurske-DePerio, 2010; Hashemi et al., 2010; Simon et al., 2010). Using this method, a subject specific set of failure data points, known as a failure locus, can be generated. The present study used a 3D FE model to perform a parametric analysis of knee joint motions to generate a failure locus. The
failure locus included bundle specific failure data for the ACL along with articular cartilage injury data. The failure locus is based on Varus/Valgus (V/V) angle and Internal/External (I/E) rotation when the knee is at 25° of flexion. Knee flexion at 25° was chosen as this is within the range of flexion angles commonly observed during ACL injury (Besier et al., 2001; Boden et al., 2000a,b; Ireland, 1999; McNair et al., 1990). Axial stress within the ACL, and shear stress within the articular cartilage were monitored during FE simulations, allowing for virtual injury diagnoses.

Previous studies have used 3D FE knee joint models to investigate the stress and strain fields in knee joint tissues under various loading conditions (Boyd et al., 2012; Gardiner and Weiss, 2003; Kiapour et al., 2014; Limbert et al., 2004; Mesfar and Shirazi-Adl, 2006; Papaioannou et al., 2010, 2008; Peña et al., 2006; Wang et al., 2014; Wangerin, 2013). Soft tissue injury was not investigated in any of these studies. Peña et al. (2006) investigated the effect of combined loading on the stress field in the ligaments and menisci. They showed that subject specific finite element knee joint models can accurately predict the stresses and strains seen in these tissues under complex loading (Peña et al., 2006). Mesfar and Shirazi-Adl (2006) investigated the effects of changes in pretension and material properties in the cruciate ligaments on the biomechanics of the knee joint. The results of their study highlighted that incorrectly applied ligament prestrain can significantly affect the natural motion of the knee joint (Mesfar and Shirazi-Adl, 2006). Limbert et al. (2004) modeled the ACL as a single bundle with and without prestrain and investigated the differences in ligament stress during passive flexion. The results of their study closely resembled results from cadaver testing in terms of the resultant force in the ligament during passive
knee flexion (Limbert et al., 2004). Limbert et al. (2004) assumed uniform prestretch throughout the ACL, which the authors stated as a limitation. The method they used applied an identical amount of stretch to each element. This is not physiologically accurate, as ligament fibers are heterogeneous, and the stretch is likely varied based on the location within the ligament fiber bundles. Gardiner and Weiss (2003) performed a subject specific study on eight sample cadaver knees to study the stress–strain behavior of the medial collateral ligament (MCL) during knee joint loading. Each knee underwent valgus loading at different knee flexion angles during which kinematic and strain data was collected. Material properties were obtained through uniaxial testing of the eight MCL specimens. FE models of the bony geometries and the MCL were constructed from volumetric CT image data for each subject. The unique MCL material properties extracted from the uniaxial testing of each specimen were applied to each FE model. Each model was validated by comparing the experimental results to the FE predictions of MCL strain during valgus loading. Gardiner and Weiss (2003) demonstrated that the complex non–uniform in–vivo strain fields seen in the MCL under valgus loading can be accurately predicted using FE techniques. Kiapour et al. (2014) developed a 3D FE model of a knee subjected to different loading conditions. The purpose of their study was to demonstrate the accuracy of their FE models at predicting soft tissue stresses and joint kinematics. Kiapour et al. (2014) state that their model will be used to determine risk factors for ACL injury in the future. These studies have all produced useful results by modeling the knee joint using finite element analysis to monitor soft tissue stress and strain during specific loading schemes. There were no studies found which use finite element knee models to
investigate the effect of various knee joint motions on ACL injuries and concomitant tissue damage.

The purpose of the present study was to determine which knee joint motions are most detrimental to the ACL and articular cartilage at 25° of knee flexion. By displaying the relative dangers between the designated knee motions, the failure locus identifies knee motions that should be avoided. This study produces useful information for improving orthopaedic care by better understanding how these injuries occur. The goal of this study is to help health care professionals improve injury prevention programs.

2.3 Methods

In order to develop a reliable model and to validate our results based on available experimental data and available numerical investigations, the following assumptions were made:

1. The ACL was modelled as transversely isotropic hyperelastic in this investigation. Preliminary simulations modelled the ACL as transversely isotropic visco-hyperelastic. These simulations, performed at physiologically accurate loading rates, showed no significant variation in the frontal plane knee angle at ACL failure existed between the different loading rates. Because of these results, a transversely isotropic hyperelastic model was chosen for the ACL in this study. This model accurately represents the response of cadaveric ACL, and is more computationally efficient compared to the rate dependent material.
2. It is known that a various axes of rotation have been proposed for the knee flexion axis (Hollister et al., 1993). Research has validated the transepicondylar axis as an optimal axis for representing proper knee flexion (Churchill et al., 1998). Also, the transepicondylar axis was used in several previous investigations, and the results were in good agreement with experimental data (Homyk et al., 2012; Yang et al., 2010b). For these reasons, in this investigation the knee flexion axis of rotation was chosen to be the transepicondylar axis.

3. Displacement driven analyses were used as opposed to force driven analyses for several reasons. Accounting for muscle force contribution in a force driven analysis is very inaccurate. Muscle forces are not constant, and to record these muscle forces experimentally for the motions of interest would lead to further inaccuracies. Displacement controlled finite element analyses provide a way to bypass these limitations. This is useful as the displacement based injury results can be compared to video evidence which is also displacement based. If an accurate model can be established which accounts for the contributions of the musculature and other soft tissues during stress analyses of the knee joint, the stress field would correspond to the same kinematics used in the present investigation (compatibility condition). A number of investigators have used displacement driven analyses to study the knee joint soft tissue stresses (Gardiner and Weiss, 2003; Park et al., 2010; Song et al., 2004). The boundary conditions for these displacement controlled simulations are generally extracted from cadaver experiments. Knee joint kinematics can be obtained from cadav-
eric testing of various loading conditions, and can be used in FE simulations. However, as the present study simulates several different failure modes for the ACL, obtaining experimental cadaver data for this would be extremely difficult, as each cadaver knee is capable of only one ACL failure.

A subject-specific 3D finite element model of a left knee was created from sagittal view magnetic resonance images (MRI) of a healthy 26-year-old male with a frontal plane alignment angle of 7.67° valgus, seen in Fig. 2.2 using the method provided by Homyk et al. (2012), and Yang et al. (2010b), similar to Donahue et al. (2003). Images were obtained using short bore, high-field 1.5 Tesla MRI and a fat suppressed fast spin echo sequence with a $TE = 10ms$, $160 \times 160mm$ field of view, and slice thickness of $2mm$ with $256 \times 256$ matrix. A similar model was successfully used in our previous investigation (Homyk et al., 2012; Yang et al., 2009). The subject was imaged early in the morning and had been unloaded for 30 minutes prior to imaging to minimize a full day of weight bearing. This was done to preserve the native geometry of the cartilage and menisci, as the geometries of these tissues have been reported to change due to body weight loading (Gründer et al., 2000; Herberhold et al., 1999; Nishii et al., 2008; Vedi et al., 1999). This may have prevented erroneous measurement of the ACL length at full knee extension, which is required in our subsequent analyses. However, we believe that the error in ACL length measured from MRI after a full day of load bearing would be small. The MRIs of the subject

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1 Informed consent was obtained prior to MR imaging.
2 Future subject specific models can be established including higher resolution MRI with a slice thickness of 0.5mm. Regardless of MRI resolution, this study establishes a procedure for investigating subject specific ACL failure.
Figure 2.2: a. Anterior view: 3D FE model of left knee joint. Bone is shown as transparent, meshed with four node bilinear rigid quadratic elements. Articular cartilage is shown in dark blue and meniscus is shown in red, both were meshed using linear tetrahedron elements. The ACL bundles are detailed in b., the amACL shown in light blue and the plACL shown in purple, both meshed with eight node brick elements. c. Boundary condition reference for the failure locus simulations performed.
were taken in the supine, non–load–bearing position. They were converted into 3D solid structures using Rhinoceros and SolidWorks, these solid structures were then imported into ABAQUS and converted to an FE mesh.

A free meshing technique was used for the cartilage and meniscus using four–node linear tetrahedral elements. The ACL was meshed using hexahedral elements. The model included a total of 40,793 nodes with 160,522 elements. The cartilage mesh size was between 0.8\text{mm} and 1.0\text{mm}, while the meniscus mesh size was 0.5\text{mm}, and the ACL was meshed at an average size of 1.0\text{mm}. A mesh sensitivity analysis was performed and an optimum mesh size was selected based on less than a 5% difference from the subsequently higher mesh density. For example, under body weight loading there was only a 2.76% difference in peak cartilage stress between the chosen mesh size (0.8\text{mm} spacing) and the next higher mesh size (0.6\text{mm} spacing). Linear elements, in conjunction with material incompressibility, may lead to volumetric locking resulting in higher stresses (Askes et al., 1999). This problem can be overcome by using higher order elements, using refined mesh, or reduced integration. Also, the reduced integration scheme may result in an erroneous solution, or no solution at all (Bell et al., 1993). For these reasons, both refined linear and quadratic elements were used. Both approaches produced the same results, and fine tetrahedron elements were adopted for the cartilage and meniscus in all simulations. This substantially reduced computational time.

Bone was modeled as rigid, as it is much stiffer than the soft tissue it interacts with (Donahue et al., 2003; Fung and Zhang, 2003; Homyk et al., 2012; Yang et al., 2010b). The articular cartilage was modeled as isotropic linear elastic and the meniscus was
Table 2.1: Cartilage and meniscus material properties (Donahue et al., 2003; Homyk et al., 2012; Kiapour et al., 2014; Yang et al., 2010b)

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Constitutive Model</th>
<th>Properties</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cartilage</td>
<td>Isotropic Elastic</td>
<td>$E = 15.0, MPa, \nu = 0.45$</td>
</tr>
<tr>
<td>Meniscus</td>
<td>Transversely Isotropic Elastic</td>
<td>$E_\theta = 140, MPa, E_r = E_z = 20, MPa$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$\nu_{rz} = 0.2, \nu_{r\theta} = \nu_{z\theta} = 0.3$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$G_{r\theta} = G_{z\theta} = 57.7, MPa, G_{rz} = 8.33, MPa$</td>
</tr>
</tbody>
</table>

modeled as transversely isotropic linear elastic, with the material properties shown in Table 2.1. Linear elastic material properties were appropriate in this study as similar material properties have been used to model cartilage and meniscus in several previous studies (Donahue et al., 2003, 2002; Kiapour et al., 2014; Wangerin, 2013; Yang et al., 2010b). The menisci were attached to the tibial plateau at the meniscal horns using linear spring element sets, similar to the methods used by Donahue et al. (2003), and Yang et al. (2010b). At each horn attachment 10 linear springs were used to attach the meniscal horn to the tibial plateau. A transverse ligament was modeled as a single spring element, which attached the anterior horns of the menisci to each other (Donahue et al., 2003; Homyk et al., 2012; Yang et al., 2010b).

There were six contact interactions between the femoral articular cartilage, the tibial articular cartilage and the medial and lateral menisci. All contact was modeled using a frictionless finite–sliding formulation where separation and sliding of finite amplitude and arbitrary rotation of the surfaces are allowed. Contact interaction normal to the contacting surfaces was constrained using the standard penalty enforcement method.
The posterior cruciate ligament (PCL), MCL, and lateral collateral ligament (LCL) were modeled as multi bundled nonlinear spring elements. 3D structures were considered for these ligaments, specifically to see if interaction existed between the ACL and PCL. The results indicated no interaction between the ACL and PCL existed for the range of motion used in this study (Orsi, 2015). This validated modeling the PCL, MCL, and LCL as spring elements, while providing an accurate estimation of the stresses in the ACL, and substantially reducing computational intensity. Ligament insertion sites were determined from the MRI, similar to the group’s previous work (Homyk et al., 2012; Yang et al., 2010b). The nonlinear spring force–displacement relationship used is defined as a piecewise continuous function, Eq. 2.1

\[
f = \begin{cases} 
\frac{1}{4} k \epsilon^2 / \epsilon_l, & 0 \leq \epsilon \leq 2 \epsilon_l \\
k(\epsilon - \epsilon_l), & \epsilon > 2 \epsilon_l \\
0, & \epsilon < 0,
\end{cases}
\]  

(2.1)

where \( f \) is the tensile force, \( k \) is a stiffness parameter and \( 2 \epsilon_l \) is the lower bound strain limit for the linear ligament behavior. \( \epsilon \) is the strain in the ligaments defined as \( \epsilon = (L - L_0)/L_0 \), where \( L \) is the ligament length and \( L_0 \) is the unstretched zero–load length of the ligament. At full knee extension the initial reference strain, \( \epsilon_r \), is listed in Table 2.2 for each bundle. \( L_0 \) is found using \( \epsilon_r \) along with the initial reference length of the ligament, \( L_r \), using \( L_0 = L_r/(\epsilon_r + 1) \), where \( L_r \) is determined from the MRI as the distance between the tibial and femoral insertion sites. This study modeled the PCL as a double bundle (anterior and posterior bundle). The LCL and
Table 2.2: Material properties for nonlinear spring ligaments (PCL, LCL, MCL) adopted from (Blankevoort et al., 1991).

<table>
<thead>
<tr>
<th>Ligament</th>
<th>Bundle</th>
<th>Stiffness parameter, $k$[N]</th>
<th>$\epsilon_r$</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCL</td>
<td>Anterior</td>
<td>9000</td>
<td>-0.24</td>
</tr>
<tr>
<td></td>
<td>Posterior</td>
<td>9000</td>
<td>-0.03</td>
</tr>
<tr>
<td>LCL</td>
<td>Anterior</td>
<td>2000</td>
<td>-0.25</td>
</tr>
<tr>
<td></td>
<td>Superior</td>
<td>2000</td>
<td>-0.05</td>
</tr>
<tr>
<td></td>
<td>Posterior</td>
<td>2000</td>
<td>0.08</td>
</tr>
<tr>
<td>MCL</td>
<td>Anterior</td>
<td>2750</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>Inferior</td>
<td>2750</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>Posterior</td>
<td>2750</td>
<td>0.03</td>
</tr>
</tbody>
</table>

MCL were modeled with three bundles. The properties of each ligament were adapted from the work of Blankevoort et al. (1991), shown in Table 2.2.

2.3.1 ACL Modeling

Studies report the ACL as comprising two main bundles, the amACL and the plACL (Blankevoort et al., 1991; Girgis et al., 1975; Peña et al., 2006; Sinkov et al., 2004; Steckel et al., 2007; Takahashi et al., 2006). This composition was chosen for modeling the ACL in this study. In full knee extension, both bundles are parallel and under prestrain. The prestrain values are provided by Blankevoort et al. (1991) and are listed in Table 2.3, showing the plACL has a higher prestrain at full extension than the amACL. During larger degrees of knee flexion the amACL increases in tension and the plACL decreases in tension (Blankevoort et al., 1991). The 3D structure of the double bundle ACL is shown in Fig.2.2b. The bundle cross sections and insertion sites were determined from the MRI, and were validated based on anatomical studies.
Table 2.3: Initial prestrain data for ACL bundles

<table>
<thead>
<tr>
<th>ACL Bundle</th>
<th>% Prestrain</th>
<th>(L_r) (mm)</th>
<th>(L_0) (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>plACL</td>
<td>10.0</td>
<td>24.54</td>
<td>22.08</td>
</tr>
<tr>
<td>amACL</td>
<td>6.0</td>
<td>27.57</td>
<td>25.91</td>
</tr>
</tbody>
</table>

(Girgis et al., 1975; Takahashi et al., 2006). The bundles were created by lofting from both insertion sites to mid–substance uniform cross section regions using SolidWorks 2010 (Dassault Systemes, Concord, MA). This produced ligament structures at the reference length, \(L_r\), seen in Fig.2.3a. \(L_r\) was defined as the distance between the centroids of the insertion sites. The ligament bundles were then resized from \(L_r\) to \(L_0\), based on the data provided by Blankevoort et al. (1991) in Table 2.3. This was done by transecting the bundles in their uniform regions according to their prestrain values. The transected bundles were then reattached to achieve zero–load length structures, shown in Fig.2.3. The superior (red) portions remained fixed to the femur, while the inferior (blue) portions were translated superiorly to attach to the superior (red) portions. This created zero–load length structures able to develop prestrain during extension from \(L_0\) to \(L_r\) during FE simulation. The 3D structures were then meshed using ABAQUS (Simulia, Providence, RI).

Using a user subroutine, a fiber orientation dependent transversely isotropic hyperelastic material, defined by a strain energy density, \(\psi\) was used to obtain the constitutive equations of the ACL (Weiss et al., 1996). The strain energy density was defined as,
Figure 2.3: a. Outline of ACL resizing process. The bundles were initially created at the reference length ($L_r$). They were then transected in the midsubstance according to their prestrain values. The bundles were then reconnected to create an unstretched structure at the zero load length ($L_0$). b. ACL prestrain simulation showing the ligament stretching from $L_0$ to $L_r$, creating the bundle specific strain which is present at full knee extension. $LE$ is defined as maximum principal strain.
\[
\psi = C_1(\tilde{I}_1 - 3) + C_2(\tilde{I}_2 - 3) + F_2(\tilde{\lambda}) + \frac{K}{2}(\ln(J))^2.
\] (2.2)

\(J\) is the Jacobian of the deformation gradient \(F\) defined as \(\frac{\partial x}{\partial X}\) where \(x\) and \(X\) are coordinates of each point in the deformed and undeformed configurations. \(C_1\) and \(C_2\) are constants representing the Mooney–Rivlin material model and \(K\) is the bulk modulus of the material. \(\tilde{I}_1\) and \(\tilde{I}_2\) are the first and second invariant of the modified Cauchy–Green strain tensor \(\overline{C} = J^{-\frac{2}{3}}F^TF\). The derivative of the fiber strain energy function \(F_2\) defined in Eq.2.3 is given as,

\[
\tilde{\lambda} \frac{\partial F_2}{\partial \tilde{\lambda}} = 0, \quad \tilde{\lambda} \leq 1
\]

\[
\tilde{\lambda} \frac{\partial F_2}{\partial \tilde{\lambda}} = C_3[e^{C_4(\tilde{\lambda}) - 1}], \quad 1 < \tilde{\lambda} < \lambda^* \quad (2.3)
\]

\[
\tilde{\lambda} \frac{\partial F_2}{\partial \tilde{\lambda}} = C_5\tilde{\lambda} + C_6, \quad \tilde{\lambda} \geq \lambda^*.
\]

The first relationship reflects the inability of ligament structures to support compressive loads. The second relationship is the nonlinear ‘toe region’ corresponding to the uncrimping of the collagen fibers. The third corresponds to the linear stress–stretch response of the straightened fibers. \(\lambda^*\) is the amount of stretch at which the material transitions from unstraightened to straightened fibers. \(\tilde{\lambda}\) is defined as the deviatoric stretch along the fiber direction (Weiss et al., 1996). The material constants were extracted from the work of Peña et al. (2006). These constants are shown in Table 2.4. Fiber direction was defined by the lines between the tibial and femoral
Table 2.4: Material coefficients for the ACL

<table>
<thead>
<tr>
<th>$C_1$ (MPa)</th>
<th>$C_2$</th>
<th>$C_3$ (MPa)</th>
<th>$C_4$</th>
<th>$C_5$ (MPa)</th>
<th>$\lambda^*$</th>
<th>$K$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.95</td>
<td>0.0</td>
<td>0.0139</td>
<td>116.22</td>
<td>535.039</td>
<td>1.046</td>
<td>1950</td>
</tr>
</tbody>
</table>

insertion sites for both ligament bundles.

As stated earlier, to simulate \textit{in–vivo} prestrain of the ACL at full knee extension, the ACL bundles were stretched from their zero–load lengths to their reference lengths. Fig.2.3 shows the double bundle ACL structure undergoing the simulated \textit{in–vivo} prestrain inherent in the ACL at full knee extension.

2.3.2 Spring Ligament Validation

While ligaments are 3D structures, modeling them as spring structures reduces complexity whereas there is no ligament–ligament interaction or ligament–bone interactions. As the purpose of this research was to investigate which combinations of knee joint movements (displacements) create mid substance rupture within the ACL bundles, it was necessary to investigate if there was interaction between the ligaments of interest within the range of motions considered in this investigation. To determine if this was a factor, a preliminary study investigated PCL–ACL interaction for the knee motions considered, and determined whether the spring model was appropriate for modeling the PCL in order to reduce computation cost. The 3D PCL was constructed from MRI images, similar to the ACL construction method described in the previous section. Four extreme case knee motions were simulated to monitor PCL–
ACL interactions. Each simulation started with the ACL prestrained followed by 25° knee flexion. The four simulations investigated were; i) 15° internal femoral rotation (IR) with varus angle applied until ACL failure, ii) 15° IR with valgus angle applied until ACL failure, iii) 15° external femoral rotation (ER) with varus angle applied until ACL failure, and iv) 15° ER with valgus angle applied until ACL failure. The ACL failure results from these simulations were compared to those obtained from the PCL spring ligament model. No significant differences were found regarding the stress distribution in the ACL or the failure angles between these two models. Also, Fig.2.4 shows for the range of motion studied, no ligament–ligament contact or ligament–bone contact was observed, justifying the use of nonlinear springs in modeling the PCL for the range of motions considered in this investigation. The PCL is slack at knee extension, which was not directly incorporated into the 3D PCL model. Because of this, the PCL was tighter than throughout knee flexion during these simulations. This increased the likelihood of ACL–PCL interactions due to an increase in PCL strain, further validating the results which showed no ACL–PCL interaction.

2.3.3 Failure Criterion

Previous investigations have reported on the ultimate tensile strength of the ACL using cadaveric knees. Woo et al. (1991) determined the ultimate failure load for ACL from nine separate young donors to be 2160 ± 157 N. Kennedy et al. (1976) determined the ultimate failure load to be 625 ± 22 N. Noyes et al. (1976) determined the ultimate failure load for the ACL to be 1730 ± 660 N. The failure load determined
Figure 2.4: Posterior view, left knee. ACL–PCL contact validation simulations. No contact seen in any of the extreme case simulations validating the use of spring elements for modelling the PCL
by Woo et al. (1991) was adapted for this study as Kennedy et al. (1976) had a high median age for the donors (62 yrs), and Noyes et al. (1976) used a smaller sample size of six samples from three young donors. The lower bound from Woo et al. (1991) was selected corresponding to a failure force of 2003N. To adapt this for a 3D structure, the axial force was converted to an axial true stress using $\sigma_t = (F/A_0)(1+\varepsilon)$. $F$ is the failure force, $A_0$ is the cross sectional area of the ACL bundles, $\varepsilon$ is the engineering strain at failure determined from Blankevoort et al. (1991), and $\sigma_t$ is the corresponding true failure stress. Both bundles had identical mid-substance cross sectional areas, $(A_0 = 23.04 mm^2)$. The axial failure force was divided between the bundles and the true failure stress at tear initiation was determined to be 50.2MPa. Butler et al. (1986) found the average failure stress for the ACL to be 36.4MPa. This value was found using the engineering stress at ACL failure from a small sample size of three subjects. Using true stress would greatly change this result which could explain the difference between the failure stress used in this investigation and the failure stress reported by Butler et al. (1986).

The articular cartilage failure criterion for this study was adapted from the work of Atkinson et al. (1998). They determined that articular cartilage fissuring is caused primarily from shear stress initiated at the surface of the articular cartilage. The authors suggested a critical shear stress of 4.15MPa is required to initiate articular cartilage fissuring. In this study, when the maximum shear stress in the articular cartilage reached 4.15MPa, cartilage was assumed to have been damaged. The critical shear stress obtained by Atkinson et al. (1998) should be considered as the lower bound shear stress required to initiate cartilage damage. It is known that fluid pres-
sure in the cartilage may result in higher critical shear stress for damage initiation. As there is no available critical shear stress for cartilage damage obtained through in–vivo experiments, we used 4.15 MPa as the critical shear stress damage criterion. This limitation may apply to all available mechanical properties obtained through in–vitro experiments.

As the simulations are under displacement control, the kinematics needed for ACL failure will not be affected by the initiation of cartilage fissuring. The extent of articular cartilage damage reported at ACL injury may be inaccurate due to changes in the material properties of damaged cartilage. Currently there is no accurate data regarding the constitutive equations for damaged cartilage. The extent of cartilage damage reported at ACL injury should be considered an underestimation as a damage model was not used. As stated earlier the knee joint motions required for ACL injury were not affected by cartilage injury in these simulations as the simulations were under displacement control. This further demonstrates that displacement control should be considered as a proper method for investigating many injuries in future research.

2.3.4 Sequential Loading Validation

Two simulations were performed to validate that loading sequence had no significant effect on the results. In the first validation simulation knee flexion was applied initially, followed by simultaneous IR and valgus loading until ACL failure. IR angles were recorded at ACL failure from this simulation for use in the second validation simulation. In the second validation simulation, knee flexion and IR were applied si-
multaneously, followed by valgus loading until failure. There was negligible difference in the valgus angle at ACL failure between the first and second validation simulations, indicating that ACL failure is independent of loading sequence in these simulations.

**2.3.5 Failure Locus Simulations**

Fourteen simulations were conducted with knee flexion held at 25°, as this angle is reported to be within the range of maximum injury susceptibility (Besier et al., 2001; Boden et al., 2000a,b; Ireland, 1999). Each simulation was a 5 step quasi-static sequential loading analysis with the following order; i) ligament prestrain, ii) 3.6×body weight, iii) knee flexion, iv) axial femoral rotation, and v) valgus or varus angular displacement. As this model does not consider muscle forces, the body weight was adjusted to 3.6×body weight to account for joint compression due to muscle contraction. This value was used to simulate the maximum compressive joint force during gait and was applied through the midpoint of the trans–epicondylar axis, consistent with previous investigations (Yang et al., 2010b). Furthermore, we believe body weight does not contribute significantly to ACL failure in this investigation. Excessive body weight may slightly reduce the ACL pretension, however the main contributors to ACL stresses are I/E rotation and V/V motion. 25° knee flexion was applied through the trans–epicondylar axis of the femur as this axis has been determined as the optimum knee flexion axis (Churchill et al., 1998). In this investigation, the relative motion between the femur and tibia is important. One can move the femur with respect to the tibia, or the tibia with respect to the femur. The femur was moved
with respect to the tibia by fixing the tibia to the ground as this simulated closed-chain foot-to-ground contact. The axis of rotation for I/E femoral rotation was the line normal to the tibial plateau intersecting the midpoint of the femoral trans-epicondylar axis. Fig.2.2c shows the boundary conditions for these simulations. Each simulation had a unique axial femoral rotation angle ranging from 15° IR to 15° ER in 5° increments (7 increments of axial rotation with varus + 7 increments of axial rotation with valgus = 14 total simulations).

ACL failure locus points were processed by tracking the normal stress within the mid-substance of both ACL bundles. Each simulation produced data points indicating the frontal plane knee angle at tear initiation for both bundles. Valgus and varus reaction moments for each ACL failure point were also extracted. The simulations also provided tibial articular cartilage damage data. This included the frontal plane knee angle at cartilage damage initiation as well as the extent of cartilage damage at the point of ACL failure. This produced the relationship between knee orientation and tissue injury, seen in Fig.2.5, which shows a complete failure locus for the ACL and cartilage. The locus is divided into four quadrants, valgus/IR, valgus/ER, varus/IR and varus/ER.

2.3.6 Simplified Model

A simplified knee model was also developed to compare ACL failure results with the full model which was developed. The objective was to observe whether a simplified model could predict ACL failure in close agreement with the complex model. This
could provide advantages for simulating ACL failure alone, as it will drastically reduce computational time taken to determine the ACL failure results. To simplify the model, the meniscus and cartilage were removed, leaving the bony geometries, and the 3D ACL double bundle structure as the only structures in the model. This eliminated all contact scenarios in the model, including cartilage–cartilage and cartilage–meniscus contact. To compare results between the simplified model and full model, four simulations were conducted with the simplified model. Knee flexion was held constant at 25°, and body weight was accounted for from the displacement results of the full model. The four simulations performed using the simplified model are as follows:

1. Simulation 1: 15° IR with valgus loading until ACL failure.
2. Simulation 2: 15° ER with valgus loading until ACL failure.
4. Simulation 4: 15° ER with varus loading until ACL failure.

The boundary conditions were all displacement driven which were extracted from the complete model. By using the displacement data from the full model, this allowed the simple model to account for the articulation of the cartilage–cartilage and cartilage–meniscus contact. The amACL and plACL failure results were recorded for each simulation and were compared against the full model.
Figure 2.5: ACL tear initiation and articular cartilage damage locus relative to the subjects initial frontal plane alignment of 7.67° valgus. The figure provides the relationship between knee orientation and knee tissue failure. The locus shows lateral tibial articular cartilage failure for valgus motions (Lat Cart Valgus), medial tibial articular cartilage failure for varus motions (Med Cart Varus), and both amACL and plACL failure for varus and valgus motions.²
2.4 Results

2.4.1 ACL Bundles: plACL vs. amACL

For all simulations, the plACL required less V/V angle to reach failure compared to the amACL. In the valgus/IR quadrant, the plACL required an average of 11.1° of valgus rotation to tear, while the amACL required 14.5°, meaning 26.7% less valgus angle was needed to reach plACL failure. In the valgus/ER quadrant the plACL required on average 20.4% less valgus angle than the amACL to reach failure. In the varus/IR quadrant the plACL required on average 36.8% less varus angle than the amACL to reach failure. In the varus/ER quadrant the plACL required on average 34.0% less varus angle than the amACL to reach failure. The average combined (varus and valgus) failure angle for the plACL was 7.9°, while the average combined failure angle for the amACL was 10.6°. Overall, the plACL was 28.5% more susceptible to tearing than the amACL. Complete ACL tear occurs when both bundles rupture. The data demonstrates that complete ACL tear is more likely to occur in ER than in IR. Furthermore, the results indicate that the ACL is more prone to complete rupture in varus knee motion than in valgus motion, especially in varus/ER.

2.4.2 Frontal Plane Motion: Varus vs. Valgus

Comparing varus motion simulations to valgus motion simulations indicated the varus motion simulations required less angle to initiate ACL tear for all degrees of I/E rotation.

\(^{315^\circ} \text{IR Valgus simulation reached } \sim 80\% \text{ amACL failure, noted by the hollow blue triangle in Fig.2.5} \)
Comparing varus/IR to valgus/IR for the amACL, the average frontal plane knee angle at tear initiation for varus/IR was 9.7° varus, while in valgus/IR it was 14.5° valgus, a 40.1% increase in frontal plane knee angle at amACL failure from varus/IR to valgus/IR. The same comparison performed on the plACL resulted in a 50.0% increase in frontal plane knee angle at plACL failure from varus/IR to valgus/IR. Comparing varus/ER to valgus/ER for the amACL, the average frontal plane knee angle for varus/ER was 7.0° varus, while in valgus/ER it was 11.0° valgus, indicating a 44.8% increase in the frontal plane knee angle at amACL failure from varus/ER to valgus/ER. The same comparison for plACL resulted in a 57.7% increase in frontal plane knee angle at plACL failure from varus/ER to valgus/ER. Overall, varus simulations required 46.6% less frontal plane knee angle for tear initiation compared to valgus simulations.

2.4.3 Transverse Plane Motion: ER vs. IR

The ER simulations required less V/V angle for ACL failure compared to IR simulations. Looking at valgus simulations, the average amACL valgus failure angle in the valgus/IR quadrant was 14.5° compared to 11.0° in the valgus/ER quadrant indicating a 27.8% decrease in valgus failure angle from valgus/IR to valgus/ER. The same analysis on the plACL in valgus resulted in a 21.5% decrease from valgus/IR to valgus/ER. Looking at varus simulations, the average amACL varus tear initiation angle in varus/IR was 9.7° compared to 7.0° in varus/ER indicating a 32.5% decrease in varus failure angle from varus/IR to varus/ER. The same analysis on the plACL in
varus resulted in a 29.7% decrease from varus/IR to varus/ER. Overall ER decreased the V/V failure angle by 27.5% relative to IR.

2.4.4 Reaction Moment at ACL Failure

Fig.2.6 shows the valgus and varus reaction moment values for each simulation. The average reaction moment required for ACL failure was in valgus simulations was 71.0N·m. For the varus simulations, the average reaction moment required for ACL failure was 35.8N·m. Comparing the average valgus moment to the average varus moment at ACL failure, the percent difference between varus and valgus was 65.81%. In ER simulations, the percent difference in reaction moment between varus and valgus was 57.96%, and in IR simulations the percent difference in reaction moment was 73.04%.

2.4.5 Articular Cartilage Injury

In all valgus cases, articular cartilage injury initiation occurred prior to ACL failure, Fig.2.5. In varus cases it occurred in between the amACL and plACL bundle failures in six out of the seven simulations. For valgus cases, the average injury initiation angle for articular cartilage was 3.2° valgus. For varus cases the average articular cartilage injury angle was 7.6° varus. The varus/IR simulations demonstrated greater varus angles at articular cartilage injury compared to the varus/ER simulations, following a similar trend as the ACL bundle failures. This was not seen in valgus, as an increase in both ER and IR increased the valgus injury initiation angle for the articular cartilage.
Figure 2.6: Valgus and varus reaction moment results at ACL failure. Data points represent frontal plane moment required for ACL tear initiation.
In valgus simulations, the most detrimental orientation for articular cartilage injury initiation was 5° IR. In varus, the articular cartilage is most susceptible to injury initiation at higher degrees of ER.

The extent of articular cartilage damage at ACL failure was much higher in valgus cases than in varus cases. This is because articular cartilage injury initiated prior to ACL failure in valgus while in varus it occurred coincidentally with ACL tear initiation. Thus, it was only necessary to investigate the extent of articular cartilage damage for the valgus cases. The results, seen in Fig.2.7a, indicate that with greater internal rotation there will be greater articular cartilage damage at ACL failure for valgus cases. This is in part due to ACL failure occurring at greater degrees of valgus when in internal rotation. If an ACL injury were to occur in IR, the cartilage damage would be greater compared to that seen from an ACL injury occurring in ER. Fig.2.7b demonstrates the growth of the lateral articular cartilage injury from initiation until ACL failure for the 10° IR valgus case. As indicated before, damage extent should be considered as an estimate as the exact constitutive equations of damaged tissue and critical stress required for damage growth are not available at this time. Regardless, the results indicate that the extent of cartilage injury is greater when ACL failure occurs in valgus motion compared to varus motion.

2.4.6 Simplified Model

The results from the simplified knee model showed minimal difference in V/V ACL failure angles compared to the full knee model. The four extreme case scenarios
Figure 2.7: a. Articular cartilage damage map reporting the % volume of damaged tissue relative to femoral axial rotation and valgus angle b. Isosurface plot of the 10° IR valgus simulation showing the growth in articular cartilage damage from injury initiation at $\tau_{max} = 4.15 MPa$ to the time of ACL failure. Here, Tresca stress is defined as $2 \times \tau_{max}$. 
Figure 2.8: Comparison of ACL bundle failure between simple and full knee model for the four extreme case simulations.

were simulated with the simplified model, and failure angles for both bundles were compared with those from the full knee model, shown in Figure 2.8. The largest percent difference between the two models was seen in the plACL in the varus/ER case at 23.3% corresponding to a discrepancy of only 0.9° degrees between the two models. The largest discrepancy in V/V failure angles between the two models was 2.3°, corresponding to a 14.2% difference which was seen for the amACL in the 15° IR valgus case. Due to excessive distortion in elements the amACL only reached ~80% of failure in this simulation. This value is denoted in Figure 2.8 with the hollow blue bar. If the full knee model were to reach complete amACL failure in this simulation, the failure angle would be greater, bringing the value closer to that of the simple model.
2.5 Discussion

The accuracy of the FE model used in this investigation was validated in our previous investigations (Homyk et al., 2012). The results from Homyk et al. (2012) compared well with the published experimental data reported by Rupp et al. (1999). Rupp et al. (1999) performed cadaver knee experiments, measuring patellar tendon ACL graft forces under various loading conditions. ACL graft tension was recorded due to valgus moments of 5N·m and 15N·m and varus moments of 5N·m and 15N·m at 30° and 60° of knee flexion, respectively. Using the model from this study, simulations were performed using the same loading conditions to compare results with Rupp et al. (1999). The ACL forces from the model used in this investigation compared well with the ACL forces reported by Rupp et al. (1999).

In this investigation, the results obtained across all simulations indicate that the plACL was on average 28.5% more susceptible to rupture than the amACL. This may be partly due to the prestrain values at full knee extension. The plACL had 10% prestrain compared to the 6% prestrain in the amACL at full extension. The higher prestrain decreased the amount of displacement needed for tear initiation. While the plACL was more likely to tear prior to the amACL, the largest separation between the V/V failure angles for the two bundles was only 3.9° and the average separation between bundle failures across all simulations was only 2.6°.

From the subjects’ initial frontal plane alignment of 7.67° valgus, less varus angle was needed for ligament failure relative to valgus angle for all values of I/E rotation, similar to the previous study (Homyk et al., 2012). Evidence exists that supports
valgus motion as the predominant injury mechanism for ACL failure (Arnold et al., 1979; Boden et al., 2000a,b; Ferretti et al., 1992; Ireland, 1999; Malone et al., 1993). The present study indicates that varus motion may be more influential than what has been previously been understood for ACL injury. The present study does not refute the valgus mechanism theory for ACL failure; however, it is suggested that future cadaver and FE studies may be needed to confirm this finding. Based on these results, we believe that ACL injury may occur during the varus portion of the contra–coup as the knee moves from valgus to varus.

ER simulations demonstrated a 27.4% increase in ACL injury susceptibility relative to the IR simulations in terms of frontal plane angle at ACL failure. This can be partially explained by the geometries of the insertion sites. With the femoral insertion located posteriorly and laterally from the tibial insertion, an applied IR will displace the femoral insertion site anteriorly and medially relative to the fixed tibial insertion. This decreases the distance between the insertion sites, decreasing ligament stress. An applied ER will do the opposite, increasing ligament stress. This increase in stress due to ER reduces the V/V failure angle for ACL injury relative to IR. Along with this, there is increased probability of a full ligament tear compared to IR simulations. Because ER increases the chance for an ACL tear by decreasing the V/V angle needed for ACL injury, and ER increases the chance of a complete tear, this suggests ER may be a more injury prone motion than IR.

In terms of reaction moments at ACL failure, varus simulations required less moment than valgus simulations. Similar to the failure angle results, ER rotation simulations required less moment to reach ACL failure than IR simulations. The
reported lower moment required to create ACL failure in varus is interesting as valgus motion more widely supported as the detrimental motion.

The model predicted which motions will cause ACL failure and concomitant injuries. For the ACL, the valgus/ER quadrant demonstrated greater susceptibility to ACL failure than the valgus/IR quadrant. The varus/ER quadrant also demonstrated greater susceptibility to ACL failure than the varus/IR quadrant. Concomitant articular cartilage damage initiated prior to ACL failure in valgus cases, and coincidentally with the ACL in all varus simulations. Both valgus/ER and varus/ER could be argued as the most detrimental motions investigated for several reasons. First, ER had lower V/V failure angles for ACL failure. Second, in ER the articular cartilage damage initiated prior to, or in sequence with, ACL failure which increased the likelihood for concomitant injuries. Last, ER demonstrated greater susceptibility for complete ACL rupture. In terms of failure angle, the valgus/ER quadrant was arguably more detrimental compared to varus/ER as the articular cartilage damage at ACL failure is greatest. However, in terms of frontal plane moment at the time of failure, the varus/ER quadrant is the most detrimental for the ACL as it requires much less torque for ACL failure.

The extent of articular cartilage damage at ACL failure was minimal in varus cases as cartilage damage initiation was coincident with ACL rupture. In valgus cases significant cartilage damage could occur prior to ACL failure. In addition, an ACL tear in valgus/IR may create the most articular cartilage damage. These results correlate well with many studies which report ER and valgus motion as leading mechanisms for ACL injury (Arnold et al., 1979; Boden et al., 2000a,b; Ferretti et al., 1992; Ireland,
1999; Krosshaug et al., 2007; Malone et al., 1993; Olsen et al., 2004). The results also
demonstrate that in addition to ACL failure occurring from this motion, cartilage
injury will occur prior to ACL failure. The results are validated with available data
in the literature and support the use of displacement controlled FE analyses of knee
joint models to investigate subject specific tissue failure mechanisms. Other stud-
ies have used displacement controlled FE models to investigate knee joint ligament
kinetics. Song et al. (2004) used a displacement controlled FE knee joint model to
compare ACL forces with a cadaver experiment. They recorded the kinematics of a
cadaver knee under a 134N anterior force. The cadaver model was then dissected,
leaving only the two bundles of the ACL. The dissected knee was subjected to the
kinematics recorded from the intact cadaver model, and the forces in the ACL bundles
were recorded. An FE model of the two ACL bundles, femur, and tibia was subjected
to the same kinematics, and the forces were compared with the dissected model. The
FE model accurately predicted the forces in the ACL bundles (Song et al., 2004).

The simplified model disregarded tissues that were not of interest which drasti-
cally decreased computational time. Under displacement control, the stresses within
individual tissue structures are independent of the presence of other tissue structures.
The simplified model showed accurate results when compared with the full knee model
in terms of ligament failure. This provides an argument towards the use of simplified
models when investigating patient specific ACL failure mechanisms. This method
dramatically decreases computation time, and allows for a fast turnaround in terms of
diagnosing patients for ACL injury predisposition. Clinically, this procedure is ad-
vantageous as it is an accurate way to quickly investigate detrimental motion schemes
for specific patients.

The present study used sequential loading analyses, while simultaneous loading simulations may better simulate in–vivo ACL disruptions. Using sequential loading was validated as the results reported little to no difference in the V/V angle at ACL tear initiation between sequential and simultaneous loading simulations. Using sequential analysis enables independent control of I/E rotation and V/V angle, allowing the results to reveal the dominant motion causing the injury. Such an understanding is not possible using simultaneous loading. By using displacement controlled simulations, the results will determine the tibiofemoral orientations causing damage or injury to knee joint tissue. For this procedure it is not required to know muscle forces. This is beneficial as modeling the subject specific muscular forces is complex and inaccurate. Material properties for the various tissues included in our model were taken from several recent publications which have been used and validated in several simulation based studies (Blankevoort et al., 1991; Donahue et al., 2003, 2002; Fung and Zhang, 2003; Kiapour et al., 2014; Peña et al., 2006; Puso and Weiss, 1998; Wangerin, 2013; Yang et al., 2010b). It is known that ACL injury and cartilage damage will change the material properties of these soft tissues. Accurately developing a constitutive model for damaged soft tissue is extremely complex. This requires numerous careful experimental investigations to properly establish the constitutive equations for the damaged tissue. As an accurate material model for damaged tissue has yet to be determined, the material properties did not change through our investigation. The cartilage damage initiation results should be considered reliable; however, the cartilage damage growth up until ACL injury may be underestimated. It should be noted
that because the simulations are displacement controlled, the extent of articular cartilage damage will have little effect on the ACL failure results. As stated earlier, the cartilage failure criterion determined from Atkinson et al. (1998) should be viewed with discretion as this criterion was determined through *ex-vivo* experimentation.

Sagittal plane motion (knee flexion) was fixed at 25° in all simulations as this angle was seen as most commonly associated with ACL injury. Future investigations may be conducted to see the relationship knee flexion has on the failure locus.

In Chapter 3, the ACL is modeled using a visco hyperelastic material to understand the effect of loading rate on ACL rupture. The results from this study demonstrated no difference existed in the frontal plane knee angle at ACL failure across a range of physiologically accurate loading rates. The differences between the frontal plane knee angles at ACL failure between the visco hyperelastic model and the hyperelastic model were minimal, validating the use of the time independent hyperelastic material model in the present study.

This investigation highlights detrimental motions leading to ACL injury and concomitant cartilage damage. The knee motion based tissue failure results provide valuable data for clinicians and sports medicine professionals. By better understanding how these injuries occur, orthopaedic surgeons can more accurately diagnose patients. The novel methods described in this investigation can provide subjects with individualized ACL failure loci and can help to identify individuals at risk for ACL failure, articular cartilage damage, and potentially future osteoarthritis. Athletic training professionals can also adapt training programs to address targeted movement avoidance of injury prone motions. Physical therapy programs will benefit by incorporating
strengthening for specific muscles which stabilize the knee joint against detrimental motions.
REFERENCES


Chapter 3

The Effect of Loading Rate on the
Anterior Cruciate Ligament Tear
Initiation Locus: A 3D Finite
Element Analysis
3.1 Abstract

Up to 400,000 annual anterior cruciate ligament (ACL) injuries occur in the U.S. creating over a $1 billion annual expense. Clearly understanding the mechanisms causing ACL injury is crucial for improving injury prevention. This study aimed to determine which movement combinations are most detrimental to the ACL, and to determine the effect of loading rate on ACL failure. A 3-dimensional knee joint model including bones and ligament was developed by digitizing magnetic resonance images. Bone was modeled as rigid; ligament was modeled as transversely isotropic visco–hyperelastic incorporating bundle specific prestrain. An ACL tear initiation locus was created from displacement driven finite element simulations. The relationships between knee joint orientation and ACL rupture were plotted providing susceptibility spectrums for ACL injury under three loading rates. The posterolateral bundle demonstrated higher rupture susceptibility than the anteromedial bundle. The frontal plane knee angles at ACL failure were 26.19% less in varus compared to valgus. Femoral external rotation decreased valgus/varus ACL failure angle by 40.67% compared to internal rotation. Loading rate had negligible effect on ACL failure, indicating that knee orientation dominates the injury mechanism. The results improve ACL injury knowledge by highlighting detrimental tibiofemoral positions. Using the data from this investigation, subject specific training programs can be tailored to help prevent ligament injuries.


3.2 Introduction

The anterior cruciate ligament (ACL) is a major motion constraining ligament of the knee. The ACL originates anterior and medial to the tibial eminence, rising posteriorly and laterally, inserting on the lateral wall of the femoral intercondylar notch (Amis and Dawkins, 1991; Arnoczky, 1983; Girgis et al., 1975; Takahashi et al., 2006). Current studies show the ACL as comprised of two main bundles, the anteromedial bundle (amACL) and posterolateral bundle (plACL), which was considered for this study (Blankevoort et al., 1991; Girgis et al., 1975; Peña et al., 2006; Sinkov et al., 2004; Steckel et al., 2007; Takahashi et al., 2006). In full knee extension, both bundles are parallel and under prestrain. The plACL has a higher prestrain at full extension than the amACL. During larger degrees of knee flexion the amACL increases in tension and the plACL decreases in tension (Blankevoort et al., 1991). ACL tears are common, painful injuries occurring upwards of 400,000 times annually in the United States (Boden et al., 2000a,b; Feagin Jr and Lambert, 1985; Fleming et al., 2010; Griffin et al., 2006; Illingworth et al., 2014; Johnson et al., 1998; Murphy et al., 1992; Nau and Teuschl, 2015; Rosen et al., 1991; Smith et al., 2012; Spindler et al., 1993). Reconstructive surgery and rehabilitation can cost $10,000 to $15,000 per incident creating an annual billion dollar expense in the United States alone (Boden et al., 2000a,b; Feagin Jr and Lambert, 1985; Griffin et al., 2006). It is clinically important to determine which movement combinations initiate tearing in the ACL bundles, as well as the influence of the rate of these movements on ACL injuries (Renstrom et al., 2008). Improved understanding of the injury mechanisms will assist the development
of prevention, rehabilitation and surgical procedures.

ACL injuries are classified by two common mechanisms: contact and non–contact. ACL injuries commonly occur in non–contact scenarios (McNair et al., 1990). Several studies show ACL injury occurring with athletes landing near full knee extension, in valgus, and in femoral external rotation (Boden et al., 2000a,b; Ferretti et al., 1992; Griffin et al., 2006; Ireland, 1999; Malone et al., 1993; McNair et al., 1990). These studies also described a deceleration closed chain mechanism associated with ACL injury. Many reports indicate that the knee was within 20° – 30° of knee flexion at the time of ACL injury (Besier et al., 2001; Boden et al., 2000a,b; Ireland, 1999; McNair et al., 1990). For this reason, in this investigation the flexion angle for the knee joint was chosen to be 25°, followed by Internal/External (I/E) rotation, and Varus/Valgus (V/V) movements.

Investigating ligament failure using cadaveric knees is cost and time prohibitive. Furthermore, a single cadaveric knee produces only one failure data point for one particular knee position. In order to generate a ligament failure locus, substantial knee samples would need to be tested. However, these data may not be related to each other because each subject has their own specific geometries. In contrast computational models can simulate multiple failures using different loading scenarios on a single knee joint. Computational models also provide a way to investigate subject specificity as each subject has unique ligament geometries. Using this method a subject specific set of failure data points can be generated, producing a failure locus.

In this investigation, a parametric study of knee joint movement combinations leading to ACL injury was performed at three different loading rates using a subject
specific 3D Finite Element (FE) model. The purpose of this study was to determine which movement combinations are most detrimental to the ACL, and to determine the effect of loading rate on ACL failure. A bundle specific failure locus was created based on Varus/Valgus (V/V) angle and Internal/External (I/E) rotation. Each simulation held knee flexion at 25° as this is within the range of commonly observed ACL injury (Besier et al., 2001; Boden et al., 2000a,b; Ireland, 1999; McNair et al., 1990). Axial stress within the midsubstance of each bundle was monitored during FE simulations, allowing for virtual tear initiation diagnoses. The failure locus provides a spectrum of injury propensity based on knee joint movements. The importance of this study, and similar future studies, is that it provides a method for determining high risk motions for specific patients. This is clinically beneficial as it provides valuable data for rehabilitation and strength training professionals. If the domain of healthy motion is understood for individual athletes, they can train within this to help prevent these harmful injuries from occurring.

### 3.3 Methods

Several important details were taken into consideration to develop this model, and are described as follows.

1. Sagittal plane motion (knee flexion) was fixed at 25° in all simulations. Future investigations may be conducted at different degrees of knee flexion to see the effect knee flexion has on the failure locus.

2. The knee flexion axis of rotation was the transepicondylar axis. It is known that
a variable axis of rotation has been proposed for the knee flexion axis (Hollister et al., 1993). However, research has validated the transepicondylar axis as an optimal axis for representing proper knee flexion (Churchill et al., 1998). Also, the transepicondylar axis was chosen to keep consistency with several previous studies performed by the group (Homyk et al., 2012; Orsi et al., 2015; Yang et al., 2010).

3. Displacement driven analyses were validated for use in this investigation. Under load control, forces within each tissue structure are affected by the presence of other tissue structures. Under displacement control, such as the simulations in this investigation, the stresses within the cruciate ligaments are independent of the presence of other tissues, as long as there is no interaction between them. The stresses in the ligaments are just a function of their end displacements. Displacement driven analyses were validated as no contact between the cruciate ligaments was seen for the range of displacements studied in this investigation. Also if force driven analyses were used, accounting for muscle force contributions would be necessary. However it is known that both measuring muscle force experimentally and estimating muscle force contributions are both inaccurate. Displacement controlled finite element analyses provide a way to bypass these limitations.

A subject–specific 3D finite element model of a male left knee was created from sagittal view magnetic resonance images (MRI), seen in Figure 3.1a, using the method provided by Homyk et al. (2012) and Yang et al. (2010), similar to Donahue et al.
Table 3.1: Initial prestrain data for ACL bundles

<table>
<thead>
<tr>
<th>ACL Bundle</th>
<th>% Prestrain</th>
<th>$L_r$(mm)</th>
<th>$L_0$(mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>plACL</td>
<td>10.0</td>
<td>24.54</td>
<td>22.08</td>
</tr>
<tr>
<td>amACL</td>
<td>6.0</td>
<td>27.57</td>
<td>25.91</td>
</tr>
</tbody>
</table>

(2003) 3D ligament bundle structures were lofted from the femoral and tibial insertion sites to mid–substance uniform cross section regions using SolidWorks (Dassault Systemes, Concord, MA) seen in Figure 3.1b. The bundle cross sections and insertion sites were determined from the MRI, and were validated using anatomical studies (Girgis et al., 1975; Takahashi et al., 2006).

To account for the inherent prestrain in the ligament bundles each bundle was resized from its reference length ($L_r$), to its zero load length ($L_0$) similar to the work of Peña et al. (2006), based on the data provided by Blankevoort et al. (1991), Table 3.1. Bundle reference lengths were determined by the distance between the centroids of the insertion sites. The reference length ligament bundles were transected in their uniform regions according to their prestrain values. The bundles were then remated together to achieve zero load length structures, this process is shown in Figure 3.2a. The superior (red) bundle sections remained fixed to the femur, while the inferior (blue) sections were translated superiorly to combine with the superior sections. This created structures which would develop prestrain in FE simulations during extension from $L_0$ to $L_r$.

The 3D model was meshed using ABAQUS CAE (Simulia, Providence, RI). The mesh was imported into the finite element package FEBio (Univ. of Utah) (Maas
Figure 3.1: a. Anterior view of knee joint: 3D finite element model of left knee reconstructed from MRI images. Bone is shown as transparent, meshed with four node bilinear rigid quadratic elements. The ACL bundles are detailed in b., the amACL shown in light blue and the plACL shown in purple, both meshed with linear tetrahedral elements. c. Boundary condition reference for the failure locus simulations performed.
Figure 3.2: a. Outline of ACL resizing process. The bundles were initially created at the reference length ($L_r$). They were then transected in the midsubstance according to their prestrain values. The bundles were then reconnected to create an unstretched structure at the zero load length ($L_0$). b. ACL prestrain simulation showing the ligament stretching from $L_0$ to $L_r$, creating the bundle specific strain which is present at full knee extension (Blankevoort et al., 1991).
et al., 2012). Bone was modeled as rigid, as it is much stiffer than the soft tissue it interacts with (Donahue et al., 2003; Fung and Zhang, 2003; Homyk et al., 2012; Yang et al., 2010). Ligament was modeled as transversely isotropic visco hyperelastic using the constitutive equations developed by Puso and Weiss (1998). For the visco hyperelastic material, the second Piola Kirchhoff stress, \( S(t) \), was defined as

\[
S(t) = \int_{-\infty}^{t} G(t - s) \frac{dS^e}{ds} ds
\]

where \( G(t) \) is the relaxation function defined as

\[
G(t) = \gamma_0 + \sum_{i=1}^{N} \gamma_i \exp \left( \frac{-t}{\tau_i} \right)
\]

and \( S^e \) is the long term elastic stress obtained by defining an elastic energy density

\[
\psi = C_1(\tilde{I}_1 - 3) + C_2(\tilde{I}_2 - 3) + F_2(\tilde{\lambda}) + \frac{K}{2} (\ln(J))^2
\]

Here \( J \) is the Jacobian of the deformation gradient \( F \) defined as \( \frac{\delta x}{\delta X} \) where \( x \) and \( X \) are coordinates of each point in the deformed and undeformed configurations. \( C_1 \) and \( C_2 \) are constants representing the Mooney–Rivlin material model and \( K \) is the bulk modulus of the material. \( \tilde{I}_1 \) and \( \tilde{I}_2 \) are the first and second invariant of the modified Cauchy–Green strain tensor \( \overline{C} = J \frac{\delta^2}{\delta x \delta x} F^T F \). The derivative of the fiber strain energy function \( F_2 \) was defined below in Equation 3.4.
Table 3.2: Viscous Material coefficients for the ACL

<table>
<thead>
<tr>
<th></th>
<th>(\tau_1)</th>
<th>(\tau_2)</th>
<th>(\tau_3)</th>
<th>(\tau_4)</th>
<th>(\tau_5)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.01</td>
<td>0.10</td>
<td>1.0</td>
<td>10.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>(\gamma_1)</th>
<th>(\gamma_2)</th>
<th>(\gamma_3)</th>
<th>(\gamma_4)</th>
<th>(\gamma_5)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.288</td>
<td>0.288</td>
<td>0.288</td>
<td>0.288</td>
<td>0.288</td>
</tr>
</tbody>
</table>

\[ \tilde{\lambda} \frac{\partial F_2}{\partial \tilde{\lambda}} = 0, \quad \tilde{\lambda} \leq 1 \]

\[ \tilde{\lambda} \frac{\partial F_2}{\partial \tilde{\lambda}} = C_3 [e^{C_4(\tilde{\lambda} - 1)} - 1], \quad 1 < \tilde{\lambda} < \lambda^* \]  \hspace{1cm} (3.4)

\[ \tilde{\lambda} \frac{\partial F_2}{\partial \tilde{\lambda}} = C_5 \tilde{\lambda} + C_6, \quad \tilde{\lambda} \geq \lambda^* \]

The first relationship reflects the assumption that ligament structures cannot support compressive loads. The second relationship is the non-linear “toe region” corresponding to the uncrimping of the collagen fibers. The third relation is linear, reflecting the mechanical properties of straightened fibers. \(\lambda^*\) is the stretch point at which the material changes from unstraightened to straightened fibers. \(\tilde{\lambda}\) is defined as the deviatoric stretch along the fiber direction. For the viscous portion of the material model constants were extracted from Puso and Weiss (1998). The constants for the elastic portion of the material model were extracted from the work of Gardiner and Weiss (2003). These constants are shown in Table 3.2 and Table 3.3. Fiber direction was the vector defined by the line between the centroids of the tibial and femoral insertion sites for both ligament bundles.
Table 3.3: Elastic material coefficients for the ACL

<table>
<thead>
<tr>
<th>$C_1$ (MPa)</th>
<th>$C_2$</th>
<th>$C_3$ (MPa)</th>
<th>$C_4$</th>
<th>$C_5$ (MPa)</th>
<th>$\lambda^*$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.44</td>
<td>0.0</td>
<td>0.57</td>
<td>48.0</td>
<td>467.1</td>
<td>1.062</td>
</tr>
</tbody>
</table>

Previous studies have investigated the ultimate tensile strength of the ACL using cadaveric knees. Woo et al. (1991) determined the ultimate failure load for ACL from nine separate young donors to be $2160 \pm 157$ N. Kennedy et al. (1976) determined the ultimate failure load to be $625 \pm 22$ N. Noyes et al. (1976) determined the ultimate failure load for the ACL to be $1730 \pm 660$ N. The failure load determined by Woo et al. (1991) was adapted for this study for the following reasons: Kennedy et al. (1976) had a high median age for the donors (62 yrs), and Noyes et al. (1976) used a smaller sample size of six samples from three young donors. The failure criterion provided by Woo et al. (1991) was also selected to keep consistency with previous work Homyk et al. (2012). The lower bound from Woo et al. (1991) was selected corresponding to a failure force of 2003 N. To adapt this to a 3D structure, the axial force was converted to an axial true stress using $\sigma_t = (F/A_0)(1 + \varepsilon)$. $F$ is the failure force, $A_0$ is the cross sectional area of the ACL bundles, $\varepsilon$ is the engineering strain at failure determined from Blankevoort et al. (1991), and $\sigma_t$ is the corresponding true failure stress. Both bundles had identical mid–substance cross sectional areas, $(A_0 = 23.04mm^2)$. These were validated by anatomical studies (Danylchuk, 1975; Girgis et al., 1975). The axial failure force of 2003 N for the whole ACL was divided accordingly between the two bundles ($F = 1001.5$ N). The true failure stress at tear initiation was determined to be 50.2 MPa. Butler et al. (1986) found the average failure stress for the ACL to be
36.4 MPa. This value was found using engineering stress and from a small sample size of three subjects. Using true stress would greatly change the results from Butler et al. (1986).

The purpose of this research was to investigate which combinations of knee joint movements and loading rates create mid substance rupture within the ACL bundles using displacement driven FE analyses. As stress and strain within the ligament bundles are functions of the ligament end position, the model can be simplified by just considering ACL, tibia and femur in displacement control simulations. This approach is only justified as long as there is no interaction between ligaments and bony surfaces. If the posterior cruciate ligament (PCL) were to contact the ACL during knee joint movement, it would affect the ACL failure scenarios. To determine if this was a factor, a preliminary investigation was conducted considering both the PCL and ACL in the model. The PCL was constructed using a similar procedure to that of the ACL. Four worst case scenario simulations were investigated, which allowed possible contact between the PCL and ACL. Each simulation started by imposing ACL and PCL prestrain, followed by 25° knee flexion. The four simulations investigated were; i) 25° internal femoral rotation (IR) with varus angle applied until ACL failure, ii) 25° IR with valgus angle applied until ACL failure, iii) 25° external femoral rotation (ER) with varus angle applied until ACL failure, and iv) 25° ER with valgus angle applied until ACL failure. The results indicated no PCL-ACL contact. Furthermore, no contact between ACL bundles and bony surfaces was observed. These results justify the use of displacement controlled analyses in the simplified model to investigate ACL failure. The simplified model substantially reduced computation time and
should be considered in any future investigations when the knee joint is subjected to displacements.

Twenty two simulations were conducted at three different V/V loading rates (sixty six simulations total). The rates chosen for varus and valgus angular displacement of 25° were 250°/sec, 500°/sec, and 2500°/sec. These rates were chosen as they provide a physiologically accurate range of rates which coincide with previous findings from Chappell et al. (2002). Each simulation held knee flexion at 25° as this angle is within the range of maximum injury susceptibility reported from previous studies (Besier et al., 2001; Boden et al., 2000a,b; Ireland, 1999). Each simulation was a three step quasi–static sequential loading analysis with the following order; i) ligament prestrain, ii) knee flexion and axial femoral rotation, and iii) valgus or varus angular displacement. The anterior tibial shear mechanism was not included in this study as video research shows the main contributors to non–contact ACL injuries are V/V motion and I/E femoral rotation (Boden et al., 2000a,b).

To simulate in–vivo prestrain of the ACL at full knee extension, each bundle was stretched from its zero load length to its reference length (Blankevoort et al., 1991; Peña et al., 2006). Figure 3.2b shows the double bundle ACL structure undergoing the prescribed prestrain displacement, simulating the in vivo prestrain inherent in the ACL at full knee extension. Due to the viscoelastic time dependent properties of the ligament material, the steady state stress field of the ligament was attained by a slow ramping rate with sufficient relaxation time. The steady state of prestrain for the visco hyperelastic ligament was validated as it converged to a similar stress state as the hyperelastic material model from Gardiner and Weiss (2003). Knee flexion
was applied at a slow angular velocity around the trans–epicondylar axis of the femur until 25°. The tibia was fixed throughout all simulations, simulating closed chain foot to ground contact. From preliminary simulations, the condylar contact points were established for use as the centers of rotation for V/V angle application (Homyk et al., 2012; Yang et al., 2010). The axis of rotation for I/E femoral rotation was the line normal to the tibial plateau intersecting the midpoint of the femoral trans–epicondylar axis. Figure 3.1c shows the boundary conditions for these simulations.

In order to understand the effects of sequential loading on ACL failure, two validation sets were performed. Each set had its own loading sequence, and was conducted at three loading rates. In the first validation set, knee flexion was applied initially followed by simultaneous IR and valgus loading until failure. IR angles were recorded at failure from this set for use in the second validation set. In the second validation set, knee flexion and IR were applied simultaneously with the IR angle corresponding to that obtained from set one, followed by valgus loading until failure. Figure 3.3 shows the valgus failure angle results for the amACL for both validation sets. In the first validation set, the average valgus failure angle across the three loading rates was 8.60°, with the largest percent difference from this average being 3.75%. In the second validation set the average valgus failure angle across the three rates was 8.79°, with the largest percent difference from this average being 1.03%. This shows negligible change in the valgus angle for ACL tear initiation across the three loading rates for both validation sets. The percent difference in average valgus failure angle between the two validation sets was 5.88%, corresponding to a difference of 0.19° valgus. The validation results indicate that valgus and varus angular displacement are the crucial
Figure 3.3: Validation simulation results showing negligible effect of loading sequence on valgus failure angle aspect of multi-axial movements leading to the ACL tear compared to femoral axial rotations. The results also indicate for the displacement rates investigated, the ACL tear is dominated by the valgus or varus angle rather than its applied loading rate.

3.4 Results

ACL failure locus points were processed by tracking the normal stress within the mid-substance of both ligament bundles. Tear initiation was achieved when the axial true stress in the tracked regions reached 50.2 MPa, based on the tensile test data from Woo et al. (1991). Each of the 66 simulations produced a data point indicating
Figure 3.4: Valgus failure locus points for both bundles under all three ramping rates
Figure 3.5: Varus failure locus points for both bundles under all three ramping rates
Figure 3.6: ACL tear initiation locus, normalized to initial frontal plane alignment of 7.67° valgus.
the femoral I/E rotation and V/V angle required for tear initiation in both bundles. Points were plotted, producing the relationship between knee orientation and ligament bundle tear. Figure 3.4 shows valgus angle vs. femoral I/E rotation for tear initiation for both bundles at all three loading rates. Figure 3.5 shows a similar relationship for varus angle. Figure 3.4 and Figure 3.5 show that the loading rate has a negligible effect on V/V failure angles for either bundle as the largest variation in these V/V failure angles across all loading rates and axial rotations was only 0.42°. Considering these results, average varus and valgus failure angles from the three rates at each axial rotation were plotted in Figure 3.6 showing a complete failure locus. The locus is divided into four quadrants, valgus/IR, valgus/ER, varus/IR and varus/ER.

The results indicated that for 17 out of 22 orientations, the plACL requires less V/V angle to achieve failure compared to the amACL. In the valgus/IR quadrant, the plACL requires an average of 9.74° of valgus rotation to tear, while the amACL requires 10.43°, showing 6.81% less valgus angle for plACL failure. The valgus/ER quadrant is the only quadrant in which the amACL requires less frontal plane angular displacement (valgus in this quadrant) than the plACL. This difference is minimal as there is only a 1.53% difference in failure angle between the bundles. In the varus/IR quadrant the plACL requires an average 15.59% less varus angle than the amACL for failure. In the varus/ER quadrant the plACL requires an average 26.92% less varus angle than the amACL for failure. While a majority of orientations show the plACL more susceptible to tearing compared to the amACL, the average separation between plACL and amACL rupture was only 0.24° in valgus orientations, and 1.13° in varus orientations.
Comparing varus angle to valgus angle simulations with identical I/E rotations, in 20 out of the 22 orientations, the varus angle simulations required less angle to initiate tearing. Looking at the IR simulations, the average tear initiation angle for the amACL in the varus quadrant was 7.40°, while in valgus it was 10.43°, a 34.01% increase. The plACL showed a 42.50% increase from varus to valgus. In ER the amACL showed a 4.45% increase in average tear initiation angle from varus to valgus with average values of 5.20° and 5.43° respectively. The plACL showed a 32.77% increase from varus to valgus. Overall, varus simulations required 30.13% less rotation for tear initiation compared to valgus simulations.

The ER simulations required less V/V angle for failure compared to IR simulations. The average amACL valgus failure angle in IR was 10.43° compared to 5.43° in ER showing a 62.99% decrease in failure angle from IR to ER. The same analysis on the plACL shows a 55.38% decrease. The average amACL varus tear initiation angle in IR is 7.40° compared to 5.20° in ER showing a 34.95% decrease in failure angle from IR to ER. The same analysis on the plACL shows a 45.94% decrease. Overall ER decreases the V/V failure angle by 51.05% relative to IR.

3.5 Discussion

A transversely isotropic visco hyperelastic material model was used to understand the effect of displacement loading rate on ACL failure as these injuries reportedly occur during short duration sports motions such as jump landing. For the range of loading rates used, there was minimal effect on the varus or valgus angle at ACL
failure initiation. Different displacement rates may result in changes to the varus and valgus failure angles. However an applied varus or valgus loading rate outside of the range investigated (slower than 250°/sec, or faster than 2500°/sec) would not be physiologically accurate for mimicking jump landing (Chappell et al., 2002). This study performed displacement controlled simulations which allowed for model simplification by disregarding tissues that were not of interest. This increased computational efficiency and should be considered as a valid method for future investigations.

The results indicate that the plACL is more susceptible to rupture than the amACL. This could be due to the prestrain values at full knee extension. The plACL has 10% prestrain compared to the 6% prestrain in the amACL at full extension. The higher prestrain may decrease the amount of displacement needed for tear initiation.

From the subjects initial frontal plane alignment of 7.67° valgus, less varus angle was needed for ligament failure relative to valgus angle, similar to the previous study from Homyk et al. (2012). This is interesting as the valgus mechanism is widely accepted as the predominant ACL failure mechanism (Arnold et al., 1979; Boden et al., 2000a,b; Ferretti et al., 1992; Ireland, 1999; Malone et al., 1993). The results provide orientation data which does not refute the valgus mechanism theory for ACL failure. However, it is interesting that this study highlights varus movement as detrimental. It breeds the question: has the varus mechanism been overlooked? It also provides an avenue for further research. Based on these results, it is believed that ACL injury is likely to occur during the varus portion of the contra–coup as the knee moves from valgus to varus.

ER simulations demonstrated a 40.67% increase in ACL injury susceptibility rel-
ative to the IR simulations. This can be explained by the geometries of the ACL insertion sites. With the femoral insertion located posteriorly and laterally from the tibial insertion, an applied IR will displace the femoral insertion site anteriorly and medially relative to the fixed tibial insertion. This decreases the distance between the insertion sites, decreasing ligament stress. An applied ER will do the opposite, increasing ligament stress. This increase in stress due to ER reduces the V/V failure angle for ACL injury relative to IR. This result correlates well with many studies which show ER as a leading mechanism for ACL failure (Arnold et al., 1979; Boden et al., 2000a,b; Ferretti et al., 1992; Griffin et al., 2006; Ireland, 1999; Malone et al., 1993).

Limitations in this study include the use of only one subject. This study also used sequential loading analyses, where simultaneous loading simulation would better simulate in vivo ACL disruptions. However, it was shown that there was little difference in the V/V ACL tear initiation angle between sequential and simultaneous loadings. In addition, this procedure allows for independent control of I/E rotation and V/V angle during FE simulations. The results show the dominant motions associated with ACL tear. Such an understanding may not be possible in a simultaneous loading simulation. Other injuries are associated with ligament rupture such as meniscal tearing, bone bruising and cartilage damage. Because the model is displacement controlled, neglecting these concomitant injuries will not affect the results.

This study provides a method for determining the range of subject specific ligament injury risk based on knee joint motion. The methods described are useful clinically as they can help rehabilitation and strength training professionals to im-
prove injury prevention strategies using subject specific data. Knowing the range of healthy motion for individual athletes provides boundaries for them to train within, helping to prevent ligament injury. The data provided from this study can also help with injury diagnosis as it provides the link between knee joint motion and ligament injury. Subject specific injury investigation is a valuable tool for the orthopedic and sports medicine communities. Future studies using similar techniques should be considered to further understand the causes of, and help prevent, these common and debilitating injuries.
REFERENCES


properties in fascicle-bone units from human patellar tendon and knee ligaments.


Chapter 4

The Effects of Graft Size and Insertion Site Location during Anterior Cruciate Ligament Reconstruction on Intercondylar Notch Impingement
4.1 Abstract

Intercondylar notch impingement is detrimental to the anterior cruciate ligament (ACL). Notchplasty is a preventative remodeling procedure performed on the intercondylar notch during ACL reconstruction (ACLR). This study investigates how ACL graft geometry and both tibial and femoral insertion site location affect ACL–intercondylar notch interactions post ACLR. A range of ACL graft sizes are reported during ACLR, from $6\text{mm} - 11\text{mm}$ in diameter. Variability of up to $3\text{mm}$ is also reported in the ACL insertion site locations during ACLR. Several 3D finite element knee joint models were constructed using three ACL graft sizes and polar arrays of tibial and femoral insertion locations. Each model was subjected to flexion, tibial external rotation, and valgus motion. Impingement force and contact area between the ACL and intercondylar notch compared well with experimental cadaver data. A $3\text{mm}$ anterior–lateral tibial insertion site shift of the average and maximum size ACL increased impingement force by 155.4% and 242.9% respectively. A $3\text{mm}$ anterior–proximal femoral insertion site shift of the average and maximum size ACL increased impingement by 292.6%, and 346.2% respectively. Simulated notchplasty of $5\text{mm}$ eliminated all impingement for the simulation with greatest impingement. For the kinematics applied, small differences in graft size and insertion site location lead to large increases in impingement force and contact area. The study aims to improve ACLR success rates by understanding how minor variations in graft size and insertion site location affect intercondylar notch impingement. Because minor variations occur during ACLR, the results may support the argument for performing notchplasty
during ACLR.

### 4.2 Introduction

The anterior cruciate ligament (ACL) acts as a major motion stabilizer for the knee joint by preventing anterior tibial displacement and providing torsional stability (Fukubayashi et al., 1982; Girgis et al., 1975; Grood et al., 1981). The ACL originates anterior and medial to the tibial eminence, rising posteriorly and laterally, and inserts on the lateral wall of the femoral intercondylar notch (Amis and Dawkins, 1991; Arnoczky, 1983; Girgis et al., 1975; Takahashi et al., 2006). The ACL is the most commonly injured ligament within the knee (Boden et al., 2000b; Griffin et al., 2000; Park et al., 2010). These painful injuries occur upwards of 400,000 times annually in the United States (Boden et al., 2000b, 2009; Griffin et al., 2006). ACL reconstructive surgery (ACLR) and rehabilitation are costly, and create an annual $1 billion expense in the United States alone (Boden et al., 2000a, 2009; Griffin et al., 2006). This cost is expected to rise with the increase in ACLR performed annually (Schreiber et al., 2010).

Up to 90% of ACL injured patients elect to have ACLR. ACLR can be performed using a bone–patellar tendon–bone (BPTB) graft, or using an anatomical double bundle reconstruction performed with a hamstring graft (Longo et al., 2008; Meredick et al., 2008; Sajovic et al., 2006). Several review papers have reported no significant benefit exists from using a double bundle hamstring graft, and recommend the use of a single bundle BPTB graft (Longo et al., 2008; Meredick et al., 2008; Sajovic et al.,...
ACLR may lead to an increased risk of reinjury (Linko et al., 2005; Paterno et al., 2014). Paterno et al. (2014) provide an overview of the studies investigating the reinjury rate of the ACL after ACLR. Wright et al. (2007) reported that 6% of patients who underwent ACLR had a reinjury within 2 years. Salmon et al. (2005) reported that 12% of patients with an ACLR had a reinjury within a five year follow up window. In this same patient population, in a 15 year follow up window, Leys et al. (2012) reported up to a 34% reinjury rate.

It has been reported that the ACL reinjury rate varies depending on the reconstruction technique (Bartlett et al., 2001; Ellis et al., 2012; Vishal et al., 2010). Several studies have compared autograft vs allograft techniques for ACL reconstruction. Ellis et al. (2012) reported that 35% of patients who underwent allograft BPTB replacement required revision surgery within one year, compared to 3% for patients who had autograft replacement. Vishal et al. (2010) reported that with a mean follow up of 49 months, 0.7% of patients who had an autograft BPTB replacement required revision surgery compared to 9.7% for patients who had an allograft BPTB replacement.

Differences have been reported regarding the reinjury rate of ACLR patients (Paterno et al., 2014; Salmon et al., 2005; Wright et al., 2007). However the reinjury rate has been reported as high as six times greater than healthy patients (Paterno et al., 2014). Because of these high surgical revision rates, it is important to understand the potential factors contributing to reinjury. Inaccurate placement of the ACL insertion sites during ACLR has been reported in 10% to 40% of ACL tunnel placements (Musahl et al., 2003, 1999). Up to 3mm of variation in graft placement from
the anatomical insertion site has been reported with experienced surgeons (Burkart et al., 2001). A range of ACL graft sizes have also been reported for use during ACL reconstruction (Magnussen et al., 2012; Tuman et al., 2007; Wallace et al., 1997; Wilson et al., 1999). Wilson et al. (1999) reported an average BPTB graft diameter of 9.9 mm and an average cross section of 44.6 mm², with a 2.2 mm standard deviation for the diameter, and a 23.1 mm² standard deviation for the cross sectional area. Magnussen et al. (2012) reported graft sizes between 7 mm and 9 mm in diameter. Tuman et al. (2007) reported that in a group of 106 patients, the average graft size was 7.7 mm in diameter, with 2% of patients having grafts 6 mm in size, 1% of patients having grafts 10 mm in size and another 1% of patients having grafts 11 mm in diameter.

Several investigations have reported impingement between the ACL and the intercondylar notch during knee joint motion (Fung et al., 2007; Fung and Zhang, 2003; Park et al., 2010). Park et al. (2010) performed a study which evaluated a 3D finite element (FE) model of ACL impingement within the femoral intercondylar notch. Park et al. (2010) validated their model with experimental data collected from an instrumented cadaver experiment. Knee flexion, external tibial rotation, and valgus motion were applied to the cadaver specimen, and the contact area and impingement force data were collected. The same kinematic data was applied to the FE model and the contact area and impingement force were monitored. The results predicted in the FE model were in close agreement with the cadaver experiment, validating the FE model as a useful tool for predicting ACL impingement.

Intercondylar notch impingement is thought to be a leading cause of ACL injury
Figure 4.1: Notchplasty diagram. a) During ACLR an osteotome is inserted endoscopically. b) The osteotome is turned parallel to the intercondylar notch wall and notchplasty is performed. c) Notchplasty is seen as the bone is removed on the intercondylar notch wall. d) A motorized bur reshapes the notch wall (Mann et al., 1999).

(Boden et al., 2000a,b; Dienst et al., 2007). Femoral notchplasty is a surgical procedure in which the intercondylar femoral notch is widened during ACLR in order to prevent impingement of the replacement graft with the femoral intercondylar notch. Figure 4.1 provides an overview of this procedure (Mann et al., 1999). Notchplasty is a common surgical technique associated with ACLR; however a standard protocol for this procedure has not been well defined. There is no recommended amount of bone removal during notchplasty, and different studies suggest a varying degree of notchplasty be performed during ACLR (Berg, 1991; Hame et al., 2003; Markolf et al., 2002).

The purpose of this chapter is to understand how differences in ACL graft size and variation in the location of the tibial and femoral insertion sites affect the interactions
between the ACL and the intercondylar notch after ACLR. This is important as it has been reported that ACL–intercondylar notch impingement may lead to ACL injury (Boden et al., 2000a,b; Davis et al., 1999; Dienst et al., 2007; Fung et al., 2007; Fung and Zhang, 2003; Park et al., 2010). The results of this study have the potential to improve the success rate of ACLR. Furthermore, as this study provides a method to quantify the amount of notchplasty that should be performed during ACLR, the results may support the use of surgical notchplasty to reduce the risk of ACL reinjury.

4.3 Methods

A subject–specific 3D FE model of a male left knee was created from sagittal view magnetic resonance images (MRI) using the method provided by Homyk et al. (2012), Yang et al. (2009, 2010), Orsi et al. (2015) and Donahue et al. (2003). The 3D knee joint model is seen in Figure 4.2a. Details of the MR data acquisition can be found in our previous work (Homyk et al., 2012; Orsi et al., 2015; Yang et al., 2009, 2010). The MRIs were converted into 3D solid structures using Rhinoceros and SolidWorks. The solid structures were then imported into ABAQUS where they were converted to an FE mesh for use in FE kinematic simulations.

A free meshing technique was used for the cartilage and meniscus using four–node linear tetrahedral elements. The ACL was meshed using hexahedral elements. Linear elements, in conjunction with material incompressibility, may lead to volumetric locking occurring in high stress regions. This problem can be overcome by using higher order elements, refining the mesh, or using reduced integration. However, the reduced
Table 4.1: Cartilage and meniscus material properties (Donahue et al., 2003; Homyk et al., 2012; Kiapour et al., 2014; Yang et al., 2010)

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Constitutive Model</th>
<th>Properties</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cartilage</td>
<td>Isotropic Elastic</td>
<td>$E = 15.0, MPa, \nu = 0.45$</td>
</tr>
<tr>
<td>Meniscus</td>
<td>Transversely Isotropic Elastic</td>
<td>$E_{\theta} = 140, MPa, E_r = E_z = 20, MPa$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$\nu_{rz} = 0.2, \nu_{r\theta} = \nu_{z\theta} = 0.3$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$G_{r\theta} = G_{z\theta} = 57.7, MPa, G_{rz} = 8.33, MPa$</td>
</tr>
</tbody>
</table>

Integration scheme may result in an erroneous solution or no solution at all. For these reasons, both refined linear and quadratic elements were used. Both approaches produced the same results, and fine tetrahedron elements were adopted for the cartilage and meniscus in all simulations. This substantially reduced computational time.

Bone was modeled as rigid as it is much stiffer than the soft tissue it interacts with (Donahue et al., 2003; Homyk et al., 2012; Orsi et al., 2015; Yang et al., 2009, 2010). The articular cartilage was modeled as isotropic linear elastic and the meniscus was modeled as transversely isotropic linear elastic with the material properties shown in Table 4.1. The menisci were attached to the tibial plateau with linear spring element sets. A transverse ligament was modeled as a single spring element which connected the anterior horns of the medial and lateral meniscus.

Contact interactions between the femoral articular cartilage, tibial articular cartilage and medial and lateral menisci were included as well as contact between the ACL and the femoral intercondylar notch wall surface. All contact was incorporated using a frictionless finite-sliding formulation where separation and sliding of finite amplitude and arbitrary rotation of the surfaces are allowed. Contact interaction
Figure 4.2: a) Anterior–lateral view of 3D FE left knee joint model. Bone shown in white (femur transparent), articular cartilage shown in dark blue, meniscus shown in red, ACL shown in light blue. b) Boundary condition diagram. c) Knee joint model after boundary condition application with cartilage and meniscus removed. Figure shows contact pressure developing between ACL and intercondylar notch. d) Zoom view of ACL showing contact pressure due to impingement with intercondylar notch.
normal to the contacting surfaces was constrained using the standard penalty enforcement method. The contact surfaces were defined by the surface of the ACL and the intercondylar notch surface of the femur.

The posterior cruciate ligament (PCL), MCL, and lateral collateral ligament (LCL) were modeled as multi bundled nonlinear spring elements. Ligament insertion sites were determined from the MRI, similar to the groups previous work (Homyk et al., 2012; Orsi et al., 2015; Yang et al., 2009, 2010). The nonlinear spring force–displacement relationship used is defined as a piecewise continuous function,

\[
f = \begin{cases} 
\frac{1}{4}k\varepsilon^2, & 0 \leq \varepsilon \leq 2\varepsilon_l \\
k(\varepsilon - \varepsilon_l), & \varepsilon > 2\varepsilon_l \\
0, & \varepsilon < 0,
\end{cases}
\]  

(4.1)

where \( f \) is the tensile force, \( k \) is a stiffness parameter and \( 2\varepsilon_l \) is the lower bound strain limit for the linear ligament behavior. \( \varepsilon \) is the strain in the ligaments defined as \( \varepsilon = (L - L_0)/L_0 \), where \( L \) is the ligament length and \( L_0 \) is the unstretched zero–load length of the ligament. At full knee extension the initial reference strain, \( \varepsilon_r \), is listed in Table 4.2 for each bundle. \( L_0 \) is found using \( \varepsilon_r \) along with the initial reference length of the ligament, \( L_r \), using \( L_0 = L_r/(\varepsilon_r + 1) \), where \( L_r \) is determined from the MRI as the distance between the tibial and femoral insertion sites. This study modeled the PCL as a double bundle (anterior and posterior bundle). The LCL and MCL were modeled with three bundles. The properties of each ligament were adapted from the work of Blankevoort et al. (1991), shown in Table 4.2.
Table 4.2: Material properties for nonlinear spring ligaments (PCL, LCL, MCL) adopted from Blankevoort et al. (1991).

<table>
<thead>
<tr>
<th>Ligament</th>
<th>Bundle</th>
<th>Stiffness parameter, $k$ [N]</th>
<th>$\epsilon_r$</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCL</td>
<td>Anterior</td>
<td>9000</td>
<td>-0.24</td>
</tr>
<tr>
<td></td>
<td>Posterior</td>
<td>9000</td>
<td>-0.03</td>
</tr>
<tr>
<td>LCL</td>
<td>Anterior</td>
<td>2000</td>
<td>-0.25</td>
</tr>
<tr>
<td></td>
<td>Superior</td>
<td>2000</td>
<td>-0.05</td>
</tr>
<tr>
<td></td>
<td>Posterior</td>
<td>2000</td>
<td>0.08</td>
</tr>
<tr>
<td>MCL</td>
<td>Anterior</td>
<td>2750</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>Inferior</td>
<td>2750</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>Posterior</td>
<td>2750</td>
<td>0.03</td>
</tr>
</tbody>
</table>

A fiber orientation dependent transversely isotropic hyperelastic material, defined by a strain energy density, $\psi$ was used to obtain the constitutive equations of the ACL (Weiss et al., 1996). The strain energy density was defined as,

$$
\psi = \psi_{\text{strain}} = C_1(\tilde{I}_1 - 3) + C_2(\tilde{I}_2 - 3) + F_2(\tilde{\lambda}) + \frac{K}{2}(\ln(J))^2.
$$

(4.2)

Where $J$ is the Jacobian of the deformation gradient $F$ defined as $\frac{\partial \mathbf{x}}{\partial \mathbf{X}}$ where $\mathbf{x}$ and $\mathbf{X}$ are coordinates of each point in the deformed and undeformed configurations. $C_1$ and $C_2$ are constants representing the Mooney–Rivlin material model and $K$ is the bulk modulus of the material. $\tilde{I}_1$ and $\tilde{I}_2$ are the first and second invariant of the modified Cauchy–Green strain tensor $\mathbf{C} = J^{\frac{2}{3}}\mathbf{F}^T\mathbf{F}$. The derivative of the fiber strain energy function $F_2$ was defined as
Table 4.3: Material coefficients for the ACL

<table>
<thead>
<tr>
<th>$C_1$ (MPa)</th>
<th>$C_2$</th>
<th>$C_3$ (MPa)</th>
<th>$C_4$</th>
<th>$C_5$ (MPa)</th>
<th>$\lambda^*$</th>
<th>$K$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.95</td>
<td>0.0</td>
<td>0.0139</td>
<td>116.22</td>
<td>535.039</td>
<td>1.046</td>
<td>1950</td>
</tr>
</tbody>
</table>

\[
\bar{\lambda} \frac{\partial F_2}{\partial \lambda} = 0, \quad \bar{\lambda} \leq 1
\]

\[
\bar{\lambda} \frac{\partial F_2}{\partial \lambda} = C_3 [e^{C_4(\bar{\lambda}^{-1})} - 1], \quad 1 < \bar{\lambda} < \lambda^*
\]  
(4.3)

\[
\bar{\lambda} \frac{\partial F_2}{\partial \lambda} = C_5 \bar{\lambda} + C_6, \quad \bar{\lambda} \geq \lambda^*.
\]

The first relationship reflects the inability of ligament structures to support compressive loads. The second relationship is the nonlinear ‘toe region’ corresponding to the un–crimping of the collagen fibers. $C_3$ scales the exponential stresses, and $C_4$ represents the rate at which the collagen fibers uncrimp. The third relationship corresponds to the linear stress–stretch response of the straightened fibers. $C_5$ is the modulus of the straightened fibers and $C_6$ is calculated so that the stress is continuous at $\lambda^*$. $\lambda^*$ is the amount of stretch at which the material transitions from un–straightened to straightened fibers. $\tilde{\lambda}$ is defined as the deviatoric stretch along the fiber direction. The material constants were extracted from the work of Peña et al. (2006). These constants are presented in Table 4.3. Fiber direction was defined as the vector connecting the centroids of the tibial and femoral insertion sites.

Three ACL size models were constructed based on the average, maximum and minimum ACL graft cross sectional geometries reported in the literature (Magnussen
et al., 2012; Tuman et al., 2007; Wallace et al., 1997; Wilson et al., 1999). The ACL graft models were created by lofting solid structures from the insertion sites on the femur and tibia to a uniform cross section in the midsubstance of the ACL. The average ACL graft size was reported between 7\text{mm} and 9\text{mm} in diameter, the maximum size was reported upwards of 11\text{mm} in diameter, and the minimum size was reported as 6\text{mm} in diameter (Magnussen et al., 2012; Tuman et al., 2007; Wallace et al., 1997; Wilson et al., 1999). The cross sectional area of the average size ACL model was 46.1\text{mm}^2, with an anterior–posterior thickness of 9.5\text{mm} and a medial–lateral thickness of 6.2\text{mm}. The cross sectional area of the maximum size ACL model was 56.1\text{mm}^2, with an anterior–posterior thickness of 10.5\text{mm} and a medial–lateral thickness of 6.8\text{mm}. The cross sectional area of the minimum size ACL model was 37.4\text{mm}^2, with an anterior–posterior thickness of 8.5\text{mm} and a medial–lateral thickness of 5.6\text{mm}.

Time dependent material properties were not used to model the ACL in this investigation. The transversely isotropic hyperelastic Mooney–Rivlin model has been used extensively to model human ligament (Orsi et al., 2015; Park et al., 2010; Peña et al., 2006; Weiss et al., 1996). Previous work has validated the use of this material model for predicting the impingement force and contact area of the ACL with the femoral intercondylar notch (Park et al., 2010). Furthermore, the boundary conditions from previous work are also used in the present study to further explore ACL–intercondylar notch impingement (Park et al., 2010).

To validate the FE model, the kinematic data from the Park et al. (2010) study was applied to the average size ACL model configuration with the original insertion
sites. The knee was moved from its initial position with a knee flexion angle of 46.3°, abduction angle of 0°, and 0° of external tibial rotation (ER), to its final position with a knee flexion angle of 44.8°, 10.0° of abduction, and 29.1° of ER. The knee joint axes of motion are provided in Figure 4.2b. The final position is shown in Figure 4.2c, with an isolated view provided in Figure 4.2d. Figure 4.3a shows the kinematic boundary conditions applied to the model. The applied kinematic data simulates landing or cutting maneuvers which are known to be associated with high ACL impingement and ACL injury (Fung and Zhang, 2003; Olsen et al., 2004; Park et al., 2010). The contact area and impingement force data were compared between the average size FE model and the experimental data from Park et al. (2010).

The cadaver sample from Park et al. (2010) exhibited impingement, and had a notch width index (NWI) of 0.22 (Fung and Zhang, 2003). NWI is defined as the width of the intercondylar notch divided by the distance between the lateral and medial epicondyles (Souryal et al., 1988). The model developed in the present investigation exhibited a NWI of approximately 0.25. Figure 4.3b provides the impingement force and contact area data from the average size ACL model. The results of the model closely approximate the experimental data from Park et al. (2010). The peak impingement force from the Park et al. (2010) experiment was 36.9N, and the present model predicted a peak impingement force of 35.1N. The contact area at peak impingement from Park et al. (2010) was 19.7mm², while the model presented in this paper predicted a contact area of 20.7mm².

To investigate how ACL graft size and both tibial and femoral insertion site location affect impingement within the intercondylar notch, several configurations of the
FE model were developed. Three ACL size models were constructed to simulate the maximum, average, and minimum graft sizes reported in the literature. The center line of tibial and femoral insertion sites for each ACL size model were relocated in the $r$ and $\theta$ directions. Two radii were chosen ($r = 1\, \text{mm}$ and $3\, \text{mm}$) in a polar array of $45^\circ$ increments from $0^\circ$ to $360^\circ$ for each ACL graft size model. Figure 4.4 shows the tibial and femoral insertion site configurations used in this investigation. The kinematic data from Park et al. (2010) was applied to each model configuration. Impingement force and contact area data were obtained from each simulation.

For the imposed knee kinematics used in this investigation, the model also incorporated a 3D model of the PCL. Three worst case configurations of possible ACL–PCL interaction were simulated. These configurations included the maximum size ACL graft model with a $3\, \text{mm}$ shift of the tibial insertion site in the medial, posteromedial, and posterior directions. These configurations all bring the ACL closer to the PCL. However, for the kinematic boundary conditions applied, the results indicated...
Figure 4.4: a) Superior axial view of left tibia showing ACL insertion locations. b) Graphic showing shift of insertion site by radius \( r \) and angle \( \theta \). c) Sagittal plane cross section view of femur showing femoral insertion locations. d) Graphic showing shift of femoral insertion site by radius \( r \) and angle \( \theta \). Radii of 1\text{mm} and 3\text{mm} were used in this investigation. Green circle represents the original insertion site. Offset locations are shown as grey circles, equally spaced in 45° increments from 0° to 360°.
that no ACL–PCL interaction existed for each of these configurations, validating the exclusion of the 3D PCL in this investigation when imposing kinematics as boundary conditions in the finite element simulations.

### 4.4 Results

The contact area and impingement force results from the average size ACL model in the original insertion site configuration compared well with the experimental data from Park et al. (2010). This validation provided a baseline to compare the results from the different configuration simulations against.

To understand how ACL graft size alone affects impingement, the simulations of the three ACL size models at their original insertion sites were compared. The results are presented in Figure 4.5. For the kinematics used in this investigation, the maximum size ACL increased contact area by 27.4%, and impingements force by
48.1%, compared to the average size ACL. The minimum size ACL decreased contact area 31.1% and impingement force by 33.5% compared to the average size ACL.

The impingement effects from a 1 mm shift in the tibial insertion site, seen in Figure 4.6, were highly dependent on the direction of the shift. When comparing the average size ACL models with a 1 mm radial shift to the average size ACL model at the original insertion site, the greatest increase in contact area observed was 8.3% in the antero–lateral (AL) direction. The largest decreases in contact area observed were 31.6% in the postero–medial (PM) and 29.6% in the medial (M) directions. The largest increases in impingement force observed were 41.7% in the AL, 34.7% in the lateral (L), and 16.9% in the postero–lateral (PL) directions. The greatest decrease in impingement force observed was 42.6% in the anterior (A) direction. When comparing the maximum size ACL model with a 1 mm radial shift to the average size ACL model at the original insertion site, an increase in contact area was observed in all directions. The largest increases observed were 34.3% in the AL and 32.1% in the L directions. Large increases in impingement force were also observed from this configuration, corresponding to increases of 101.37% in the AL, 92.0% in the L and 79.9% in the A directions. When comparing the minimum size ACL model with a 1 mm radial shift to the average size ACL model at the original insertion site, decreases in contact area were observed in all directions. The largest decrease observed was 54.8% in the PM direction. Impingement force also decreased in all directions, with the largest observed as a 60.7% decrease in the PM direction.

For the tibial insertion site, simulations with 3 mm of shift produced the most pronounced effects on ACL–intercondylar notch impingement. When comparing the
Figure 4.6: Tibial insertion site shift results. Comparison of the contact area and impingement force data at the end of the time step with a knee flexion angle of 44.8°, valgus angle of 10.0°, and an ER angle of 29.1°. Data is shown for the tibial insertion site configurations with radii ($r$) of $r = 1\, mm$, and $r = 3\, mm$ for the three ACL graft size models.
average size ACL model with a 3\textit{mm} radial shift to the same model at the original insertion site, the greatest increases in contact area observed were 70.7\% in the L and 54.4\% in the AL directions. The greatest decreases in contact area observed were 70.0\% in the PM and 54.4\% in the M directions. The largest increases in impingement force observed were 155.4\% in the AL and 132.8\% in the L directions. The largest decrease in impingement force observed was 85.0\% in the PM and 72.0\% in the M directions. When comparing the maximum size ACL model with a 3\textit{mm} radial shift to the average size ACL model at the original insertion site, the largest increases in contact area observed were 100.4\% in the L and 88.5\% in the AL directions. The largest decrease in contact area observed was 45.4\% in the PM direction. The largest increases in impingement force observed were 242.9\% in the AL, 201.2\% in the L, and 167.2\% in the A directions. When comparing the minimum size ACL model with a 3\textit{mm} radial shift to the average size ACL model at the original insertion site, the largest decrease in contact area observed was 97.4\% in the PM direction. The greatest decrease in impingement force observed was 99.8\% also in the PM direction, effectively removing all contact. It should be noted that a large increase in impingement force of 90.8\% was observed in the AL direction for the minimum size ACL model compared to the average size model at the original insertion site. This is an interesting result because it shows that even if a small ACL graft is selected, significant impingement effects are possible if the graft is not properly located on the tibial plateau.

The impingement effects from a 1\textit{mm} shift in the femoral insertion site, seen in Figure 4.7, were also highly dependent on the direction of the shift. When comparing the average size ACL models with a 1\textit{mm} radial femoral insertion site shift to the
Femoral Insertion Site Shift Results

Figure 4.7: Femoral insertion site shift results. Comparison of the contact area and impingement force data at the end of the time step with a knee flexion angle of 44.8°, valgus angle of 10.0°, and an ER angle of 29.1°. Data is shown for the femoral insertion site configurations with radii (r) of \( r = 1 \text{mm} \), and \( r = 3 \text{mm} \) for the three ACL graft size models.
average size ACL model at the original insertion site, the greatest increase in contact area observed was 22.0% in the antero–proximal (APr) direction. The largest decrease in contact area was 23.3% in the postero–distal (PD) direction. The largest increase in impingement force observed was 31.3% in the APr direction. The greatest decrease in impingement force observed was 27.8% in the PD direction. When comparing the maximum size ACL model with a 1 mm radial shift to the average size ACL model at the original insertion site, an increase in contact area was observed in all directions. The largest increase was 67.2% in the APr direction. This configuration also produced the greatest increase in impingement force, reported at 97.3%. When comparing the minimum size ACL model with a 1 mm radial shift to the average size ACL model at the original insertion site, decreases in contact area were observed in all directions. The largest decrease observed was 44.8% in the PD direction. Impingement force also decreased in all directions, with the largest observed as a 54.8% decrease in the PD direction.

Compared to the 1 mm shift, simulations with 3 mm of shift in the femoral insertion site enhanced the impingement effects between the ACL and the intercondylar notch. When comparing the average size ACL model with a 3 mm radial shift to the same model at the original insertion site, the greatest increase in contact area observed was 112.0% in the APr direction. The greatest decrease in contact area observed was 69.2% in the PD direction. The largest increase in impingement force observed was 292.6% in the APr direction, and the largest decrease in impingement force was 73.3% in the PD direction. When comparing the maximum size ACL model with a 3 mm radial shift to the average size ACL model at the original insertion site, the largest in-
crease in contact area observed was 168.0% in the APr direction. The largest decrease in contact area observed was 100.0% in the distal (D) direction. The largest increase in impingement force observed was 346.2% in the APr direction. When comparing the minimum size ACL model with a 3 mm radial shift to the average size ACL model at the original insertion site, decreases in contact area and impingement force were seen in all but the APr direction. A 3 mm shift in the APr direction increased contact area by 27.8%, and impingement force by 113.8%. The largest decrease in contact area observed was 69.9% in the PD direction. The greatest decrease in impingement force observed was 81.6% also in the PD direction.

Notchplasty was simulated using the maximum size ACL model with 3 mm lateral shift of the tibial insertion site as well as a 3 mm antero–proximal shift in the femoral insertion site. These configurations were selected because they exhibited the greatest impingement of all configurations developed this investigation. To simulate notchplasty, the lateral femoral notch wall surface was isolated, and translated laterally by increments of 1 mm. The same kinematic data was applied to these notchplasty models, and impingement force and contact area were monitored. For the kinematics used in this investigation, it was determined that 4 mm of notchplasty reduced impingement force by 89.4% for the 3 mm lateral shift of the tibial insertion site configuration. 3 mm of notchplasty reduced impingement force by 95.0% for the 3 mm antero–proximal femoral insertion site shift configuration. 5 mm of notchplasty completely removed all impingement for the maximum size ACL model with a 3 mm lateral tibial insertion site shift, while 4 mm of notchplasty completely removed all impingement for the maximum ACL size with a 3 mm antero–proximal femoral insertion.
site shift.

The effects of combined femoral and tibial insertion site shifts were also of interest. One simulation was performed using the maximum size ACL graft with the tibial and femoral insertion sites which produced the greatest impingement (3\text{mm} \, \text{APr} \, \text{for femur and} \, 3\text{mm} \, \text{L for tibia}). The results showed a drastic increase in contact area and impingement force compared to the other simulations. This simulation produced a contact area of 62.1\text{mm}^2, which is 245.0\% greater than the average size ACL model at the original insertion site locations. The impingement force was 273.0\text{N}, which is 678.3\% greater than the average size ACL graft at the original insertion site locations. Notchplasty was also performed for this configuration, and 5\text{mm} \, \text{of notchplasty was sufficient for eliminating all impingement between the ACL and the intercondylar notch.}

Understanding the affects that insertion site shifting may have on knee joint kinematics is important, as kinematic changes could further affect impingement. To determine if insertion site shifting did affect the kinematics of the knee joint, several tests were performed using three configurations of the model: the standard configuration, the 3\text{mm} \, \text{APr femoral shift model, and the} \, 3\text{mm} \, \text{L tibial shift model. Each model was subjected to three loading scenarios: a 2\text{N} \cdot \text{m flexion moment, a} \, 3\text{N} \cdot \text{m ER moment, and a} \, 30\text{N} \cdot \text{m valgus moment, corresponding to the reaction moments associated with knee joint motions applied in Aim 1. The resulting kinematics from each test were compared across the three model configurations, and are shown in Figure 4.8.}

It was observed that minimal differences in kinematics existed between the in-
Figure 4.8: Rotational and displacement degrees of freedom resulting from a 2N·m flexion moment, 3N·m external rotation moment, and a 30N·m valgus moment for the standard, 3mm lateral tibial shift, and 3mm antero-proximal femoral shift models.
sertion site shifting models across the three tests. This result supports the methods applied in this investigation in which the same kinematics were applied to each insertion site shift model.

4.5 Discussion

The FE model was validated with experimental data. A preliminary investigation ensured that the inclusion of a 3D PCL was not necessary, as it did not affect the results. Furthermore, the hyperelastic material model for the ACL was validated through preliminary investigations which showed no benefit to using a visco–hyperelastic material model for the kinematics used in this investigation. After model validation, different configurations of ACL graft size and both tibial and femoral insertion site locations were introduced. These configurations were created based on reported variations in ACL graft size and insertion site location during ACLR (Burkart et al., 2001; Magnussen et al., 2012; Musahl et al., 2003, 1999; Tuman et al., 2007; Wallace et al., 1997; Wilson et al., 1999). Minimum, average, and maximum ACL graft sizes were created, and the original tibial and femoral insertion sites were relocated in the $r$ and $\theta$ directions for each graft size model. It was expected that larger ACL grafts as well as lateral tibial and anterior femoral insertion site shifting would lead to increased contact area and impingement forces, as these shifts would move the ACL closer to the intercondylar notch wall.

The impingement on the intercondylar notch wall is of interest as this is thought to be a major factor influencing ACL injury (Boden et al., 2000b, 2009; Dienst et al.,
In an animal study by Fung et al. (2005), cell death was reported to occur at the impingement site of impinged ACLs compared to unimpinged ACLs. A similar study performed by Gyger et al. (2007) suggest that apoptosis has a role in ligament rupture. This evidence supports the notion that intercondylar notch impingement may weaken the ACL and could be a leading cause of ACL tears.

Increases in impingement were seen in the average size ACL with AL and L tibial insertion site shifts and APr femoral insertion site shifts. Decreased impingement was observed during PM and M tibial insertion site shifts. Large increases in impingement were seen in the maximum size ACL model with AL tibial insertion site shifts and a APr femoral insertion site shifts. The minimum size ACL model lead to decreased impingement with a 3mm medial tibial insertion site shift nearly eliminating all impingement. These results show significant impingement effects may result even with a small ACL graft if the insertion sites are not accurately located.

The results indicate impingement force and contact area are both highly sensitive to minor differences in graft size and both tibial and femoral insertion site location. The results may suggest that notchplasty be performed to reduce the possibility of adverse interactions between the ACL and the intercondylar notch as insertion site variations are inevitable. As an attempt to quantify the amount of notchplasty required to eliminate impingement, notchplasty was simulated on several high impingement configurations. For the knee joint kinematics used in this investigation, it was determined that 5mm of notchplasty removed all impingement for the highest impingement configurations.

In some of the simulations with relatively high levels of impingement, the ACL
was in contact with the femoral intercondylar notch at the start of the simulation. This result may be useful for orthopaedic surgeons performing ACLR. If the ligament is already in contact with the intercondylar notch after surgery, an applied external tibial rotation and/or valgus loading will only increase this interaction.

This study explored only one knee kinematic scenario. However, the kinematic data used simulates high impingement motion associated with athletic cutting maneuvers and ACL injury (Fung and Zhang, 2003; Olsen et al., 2004; Park et al., 2010). Anterior tibial translation is known to increase ACL–intercondylar notch impingement. This motion was not directly applied to the model, however by simulating tibial insertion site shifting in the anterior direction, this motion was essentially simulated. The results show that anterior tibial insertion site translation increased the ACL–intercondylar notch impingement significantly. Along with this, ligament prestrain was not included in this model, unlike the groups previous work (Homyk et al., 2012; Orsi et al., 2015; Yang et al., 2010). This was done in order to compare results with the model from Park et al. (2010), which did not include ligament prestrain. However, to understand how the inclusion of prestrain would affect the results, a 5% prestrain was incorporated into the average ACL at the original insertion site. The results from this simulation only lead to a 2.8% difference increase in contact area compared to the simulation without prestrain. This increase was expected, as tightening the ACL increases the contact pressure between the ACL and the intercondylar notch. Because prestrain did not affect the results significantly, excluding prestrain was considered valid in this investigation.

The effects that surgical variations may have on knee joint kinematics are un-
known. These variations could potentially change the natural motion of the knee joint, further affecting impingement. Because the simulations performed in this investigation were displacement controlled, changes in knee joint kinematics were not included. Future investigations should consider this, as the associated changes in knee joint kinematics may be a contributing factor in the high post-operative ACL re-injury rate.

3\text{mm} of variation during ACL insertion site location should be considered very minimal, yet probable. The results show these minor variations may enhance the contact interactions between the ACL and the femoral intercondylar notch. However, the reported contact area values are relatively small. Because of this, minor changes in contact area between the configurations investigated may produce large percent differences. This should be considered to prevent a misinterpretation of the results. However, the results show that certain configurations do lead to increased impingement between the ACL and the intercondylar notch wall. Most notably was the configuration which combined the highest impingement configuration for the femur and tibia insertion sites which produced extremely large increases in contact area and impingement force. Because of this, several recommendations can be made. If notch-plasty is to be performed, it may be recommended that notchplasty of 4\text{mm} – 5\text{mm} will sufficiently eliminate ACL–intercondylar notch impingement. Also, if the surgeon is to err on the side of caution while locating the ACL graft insertion sites, it may be recommended to avoid anterior and proximal femoral insertion site locations, as well as to avoid anterior and lateral tibial insertion site locations.

This study provides a methodology for analyzing how variations in ACLR affect
ACL impingement force and contact area with the intercondylar notch. Future studies may investigate the effects of other reported surgical variations, such as ACLR technique. Understanding how the differences in ACL graft size and insertion site placement affect the ACL–intercondylar notch interactions is extremely important. The data provided from this investigation, and future similar studies, has the potential to improve outcomes of surgical procedures, reduce medical costs, and improve patient satisfaction.
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Chapter 5

Does The Transepicondylar Axis Location Affects Tibial Articular Cartilage Stress Patterns?
5.1 Abstract

The transepicondylar axis is a reference axis for applying boundary conditions to finite element knee joint models. This study investigates how variability in determining this axis affects model accuracy. Understanding how minor variations in axis detection affect model results will help to understand the requirements for accurately defining this axis. Several configurations of the same finite element knee joint model were constructed by parametrically changing the location of the transepicondylar axis. Each configuration underwent simulations including knee flexion, external femoral rotation and valgus motion. Articular cartilage stress, damage growth, and contact patterns were compared between each configuration. Minor differences in damage growth and contact patterns were observed between the different model configurations. Differences were observed in contact patterns, as 4mm of variation in the transepicondylar axis changed the location of maximum cartilage stress by 6% of the anterior–posterior length of the cartilage. The methods described in this investigation should be considered in all future biomechanical finite element modelling as the results show the importance of locating the reference axes for boundary condition application.

5.2 Introduction

The transepicondylar axis (TEA) is widely used in biomechanical finite element (FE) modelling to approximate the knee flexion axis (Homyk et al., 2012; Orsi et al., 2015; Yang et al., 2010). The TEA is also important clinically as a reference for locating the femoral implant during total knee arthroplasty (TKA). The TEA is determined
through palpation, and is defined as the line between the most prominent point on
the lateral epicondyle and the most prominent point on the medial epicondyle, at the
medial collateral ligament insertion site (Berger et al., 1993; Jerosch et al., 2002). Acc-
curately locating this axis is important clinically to ensure proper implant alignment
during TKA. In computational biomechanics, it remains unknown as to what degree
TEA accuracy affects model results.

Several studies have reported that the process of locating the TEA is prone to
error. Churchill et al. (1998) reported that the standard deviation of the epicondyle
locations is $\pm 2.4\,mm$ in the anterior–posterior direction and $\pm 2.7\,mm$ in the proximal–
distal direction. In a study by Jerosch et al. (2002) in which eight surgeons determined
the epicondyle locations on three separate cadaver specimens, discrepancies of up to
13.8$mm$ on the lateral and 22.3$mm$ on the medial epicondyle locations were reported.
This corresponded to areas of 116$mm^2$ for the lateral epicondyle location and 102$mm^2$
for the medial epicondyle location.

The affects of errors associated with TEA location have not been investigated
extensively. This investigation aims to understand how locating the TEA may affect
articular cartilage stress and damage patterns in a 3D knee joint finite element model.
The results will help verify the accuracy of these FE models and will help guide the
protocol for developing future models by determining the accuracy required for TEA
location.
5.3 Methods

Using the FE knee joint model from Chapter 2, a sensitivity analysis was performed to investigate the effect of errors in the sagittal plane location of the TEA. Four model configurations were developed to study the effect of these errors on the stresses in the cartilage. The TEA was moved parametrically around its original location in the sagittal plane. Each error configuration model displaced the TEA 4 mm in both the anterior–posterior and proximal–distal directions from the original location, creating the 4 error configurations shown in Figure 5.1.

A FE simulation was performed using each of the configurations. The simulation was a 5 step quasi–static sequential loading analysis with the following sequence of boundary conditions;
1. Ligament prestrain

2. $3.6 \times$ body weight

3. $25^\circ$ knee flexion

4. $15^\circ$ external femoral rotation

5. Valgus motion

These boundary conditions were selected because previous work studying ACL injury and concomitant cartilage damage showed that the results from simulations using these boundary conditions correlated well with clinical studies. The preliminary study reported that ACL injuries occurring with the knee in external femoral rotation combined with valgus motion lead to articular cartilage damage in the posterior and lateral portion of the lateral tibial plateau (Orsi et al., 2015). In clinical studies monitoring the cartilage damage during ACL injury, bone bruising and cartilage damage have been reported at the same locations (Faber et al., 1999; Johnson et al., 1998; Vellet et al., 1991).

Knee flexion was applied through the TEA of the femur as this axis has been reported as an adequate approximation of the knee flexion axis (Churchill et al., 1998). The axis of rotation for internal and external femoral rotation was the line normal to the tibial plateau intersecting the midpoint of the femoral TEA. The femur was moved with respect to the tibia by fixing the tibia to the ground as this simulated closed–chain foot–to–ground contact. Valgus motion was applied through the condylar contact point between the femur and tibia. This location was determined as the
centroid of the contact area of the tibial cartilage after body weight application.

Several variables were compared between the configurations including cartilage contact pressure, the path of maximum pressure, location of cartilage damage, and the growth of damage during valgus loading. Cartilage damage was determined using the same method from Orsi et al. (2015), adapted from the work of Atkinson et al. (1998). Atkinson et al. (1998) suggested a critical shear stress of $4.15 \text{MPa}$ is required to initiate articular cartilage fissuring. In this study, when the maximum shear stress in the articular cartilage reached $4.15 \text{MPa}$, cartilage was assumed to have been damaged. This critical shear stress should be considered as the lower bound criterion for damage initiation (Atkinson et al., 1998). It is known that fluid pressure in the cartilage may result in higher critical shear stress for damage. As there is no available critical shear stress for cartilage damage obtained through in–vivo experiments, we used $4.15 \text{MPa}$ as the critical shear stress damage criterion. This limitation may apply to all available mechanical properties obtained through in–vitro experiments.

The extent of articular cartilage damage reported may be inaccurate due to changes in the material properties of damaged cartilage. Currently there is no accurate data regarding the constitutive equations for damaged cartilage. The extent of cartilage damage reported should be considered an underestimation as a damage model was not used. The knee joint motions applied were not affected by cartilage damage because they are displacement driven. Displacement controlled simulations should be considered as a valid method for investigating injuries in future FE modeling based research.
5.4 Results

Several comparisons were performed across the five model configurations. Figure 5.2 shows the contact pressure images for each stage of the simulation for each configuration.

At body weight application, the lateral tibial cartilage is not experiencing high contact pressure, as the medial condyle absorbs more load during this time. At this stage, the peak pressure in the lateral condyle is located centrally on the condyle. As the knee moves into flexion, contact pressure increases, and a band of high contact pressure is located posteriorly. This band is due to the engagement of the meniscus with the cartilage during flexion. During external femoral rotation, the location of maximum pressure on the lateral cartilage moves medially as the femur rotates across the tibial plateau. As the knee moves into valgus, the high pressure region moves to the posterior and lateral portion of the lateral tibial cartilage. During valgus, the highest pressures are located on the lateral tibial cartilage due to the medial condyle lift off which occurs.
Figure 5.2: Superior–axial view, lateral tibial cartilage: contact pressure on lateral tibial cartilage during each stage of the simulation for the five model configurations.
Figure 5.3 shows the path of pressure for each configuration during the knee joint motion simulation.

The location of maximum pressure did not change significantly in the medial–lateral (M–L) direction between configurations. The maximum change relative in this direction was less than 2% of M–L width of the cartilage. In the antero–postero (A–P) direction, the location of maximum pressure exhibited greater model–to–model variations throughout the simulation. Differences in maximum pressure location were reported as high as 6% of the A-P length of the cartilage.

Articular cartilage damage was also compared between each configuration. The cartilage damage plots for each configuration are reported in Figure 5.4. This shows the lateral tibial cartilage damage for each configuration at 8.75° of valgus motion. Figure 5.5 reports the progression of the volume of damage for each configuration.

The location of cartilage damage was very similar across the five configurations.
Figure 5.4: Articular cartilage failure region plot.

Figure 5.5: Damage volume plot for each configuration during valgus loading.
Each configuration showed damage initiation in the postero–lateral region of the lateral tibial cartilage. The growth of the articular cartilage damage volume was also similar across the five configurations. The largest discrepancy in damage volume growth was less than a 10% compared to the standard configuration. This was observed in configuration 2, and corresponded to a difference of only $23\text{mm}^3$.

5.5 Conclusions

This investigation determined the effects of different TEA locations on tibiofemoral cartilage interactions using a subject specific FE model. The analysis showed that $4\text{mm}$ errors in the sagittal plane location of the TEA may only shift the location of high stress in the A-P direction. Overall, these errors produce minimal differences in the cartilage stress pattern results. Across the four error configurations there was minimal variation in the location of cartilage damage. Along with this, the damage volumes were very similar across all error configurations. Cartilage damage was in the lateral and posterior region of the tibial cartilage. It has been well documented that tibial cartilage damage is most common in the posterior and lateral portions of the lateral tibial condyle during ACL injury. The results show that errors in locating TEA should not affect properly simulating tibiofemoral motion with displacement controlled boundary conditions. This study shows that accurately locating the TEA in knee joint finite element models is important. The results should also be considered when performing total knee arthroplasty because the sensitivity of the implant location may change the stress distributions on the implant. Biomechanics profes-
sionals should also understand the sensitivity of these results when locating marker sets during motion analysis.
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Chapter 6

Conclusions and Future Work
6.1 Conclusions

Several 3D finite element (FE) knee joint models were developed throughout the investigations in this thesis. The models were designed to investigate knee joint injury mechanisms and surgical interventions including ACL injury, articular cartilage damage and ACL–femoral intercondylar notch impingement during ACL reconstruction.

The following items were addressed separately throughout the three specific aims of this thesis.

- Develop a failure locus for a 3D ACL
- Investigate concomitant cartilage damage during ACL injury
- Evaluate a simplified FE model for developing ACL failure locus
- Incorporate viscoelastic material properties for the ACL
- Evaluate the effects of viscoelastic material model on the ACL failure locus
- Investigate how surgical variations during ACL reconstruction affect ACL–intercondylar notch impingement
- Simulate and provide recommendations for notchplasty during ACL reconstruction
6.2 Aim 1. Develop a failure locus to understand the relative dangers between different knee motions for ACL and articular cartilage injury.

A complete subject specific FE knee joint model was developed to investigate soft tissue injury. ACL injury and concomitant articular cartilage damage were of interest because this injury combination is one of the most common to occur to the knee joint. The model included 3D structures for the bone, articular cartilage, meniscus and the ACL, and modelled the remaining ligaments as nonlinear spring element sets. A transversely isotropic Mooney–Rivlin material model was incorporated for the ACL. A parametric analysis of knee joint motion including body weight loading, ligament prestrain, knee flexion, internal/external rotation and valgus/varus motion was performed to understand which kinematic combinations are more detrimental to the ACL and articular cartilage.

The results showed ACL failure was most likely to occur in external femoral rotation (ER). ACL failure occurred at lower frontal plane angles in varus compared to valgus. Combined valgus and ER was one of the most detrimental motion combinations for ACL injury. The lowest frontal plane moment required for ACL failure was seen during varus combined with ER.

Concomitant cartilage damage was shown most likely to occur alongside ACL injury during valgus/ER scenarios. In internal femoral rotation (IR), greater amounts of cartilage damage existed at the instance of ACL injury. However, these motions
may be less likely to occur because they require greater frontal plane moments.

During the valgus/ER motion articular cartilage damage was seen in the postero-lateral region of the lateral tibial condyle during ACL injury. This result matched well with clinical studies of ACL injured patients which reported concomitant bone bruising occurring in the same location. Coincidentally, video evidence has also reported this motion combination as a likely mechanism for ACL injury. The results correlate well with both clinical analyses of cartilage damage locations as well as video evidence of ACL injury.

A simplified version of the same knee joint model was developed which incorporated only the bony geometries of the femur and tibia, and the ACL. Excluding the other soft tissues was validated because for the motions investigated, there was no interaction between the ACL and the other ligaments. The simple model predicted similar results compared to the complete knee joint model. While this model is not capable of predicting concomitant cartilage damage, it is useful for determining the failure locus for the ACL alone due to its computationally efficiency, and its ability to rapidly provide results.
6.3 Aim 2. Determine how a rate dependent material model affects the results of the failure locus.

Similar to the simplified model from Aim 1, a subject specific FE model of the knee joint was constructed including the bony geometries of the femur and tibia, and the double bundle ACL. By disregarding tissue not of interest, the model was simplified, increasing the computational efficiency. This model was imported into FEBio, as this software package has a readily available visco-hyperelastic transversely isotropic Mooney–Rivlin material model (Maas et al., 2012). This material model was applied to the ACL to determine the effect of loading rate on ACL failure as ACL injuries are reported to occur during sports activities in short durations.

A parametric analysis of displacement controlled simulations was performed across three different loading rates. The loading rates were selected to simulate the maximum and minimum angular velocities of knee joint motions reported during athletic landing and cutting maneuvers. For the range of physiologically accurate loading rates, it was determined that loading rate had minimal effect on the frontal plane knee angle required for ACL injury. Similar results were obtained from the rate dependent model and the model from Aim 1. The plACL was more susceptible to rupture than the amACL, possibly due to higher prestrain values at full knee extension. Also, the ER simulations required less V/V angle for failure compared to IR simulations.

This study provides a method for determining subject specific ligament injury risk
based on knee joint motion. This is useful clinically as it may help improve subject specific injury prevention. Determining the range of healthy motions for individual athletes provides boundaries for them to train within. The data provided from this study can also help with injury diagnosis. Future studies using similar techniques should be considered to further understand the mechanisms of ACL injury.

6.4 **Aim 3. Understand how surgical variations during ACL reconstruction affect ACL–femoral intercondylar notch impingement.**

The purpose of this study was to understand how differences in ACL graft size and variations in the tibial and femoral insertion site locations affect the ACL–intercondylar notch interactions after anterior cruciate ligament reconstruction (ACLR). This study is important because ACL–intercondylar notch impingement is reported to lead to ACL injury (Boden et al., 2000a,b; Davis et al., 1999; Dienst et al., 2007; Fung et al., 2007; Fung and Zhang, 2003; Park et al., 2010). Understanding how these surgical variations affect impingement has the potential to improve the success rate of ACLR. This study also provides a method for quantifying the amount of notchplasty required to eliminate impingement during ACLR.

A 3D FE knee joint model was constructed and validated with experimental data of ACL–intercondylar notch interactions. The FE model showed good agreement with the cadaver experiment. Several configurations of this model were then introduced
to investigate the effects of surgical variations on ACL–intercondylar notch impingement. Three ACL size models were introduced with polar arrays of tibial and femoral insertion sites.

The results showed that larger graft sizes increased intercondylar notch impingement. The results also showed that anterior, antero–lateral, and lateral shifting of the tibial insertion site increased impingement. Femoral insertion site shifting was more sensitive to increased impingement. It was determined that proximal, anteroproximal and anterior shifting of the femoral insertion site produced the greatest increases in impingement. A worst case surgical misplacement of insertion sites was simulated using the maximum size ACL graft with a 3mm anterior femoral insertion site shift and a 3mm lateral tibial insertion site shift. This configuration lead to nearly a 700% increase in impingement force compared to the average size ACL model at the anatomical insertion sites. Notchplasty was simulated on model configurations exhibiting high impingement by isolating the lateral femoral notch wall and translating it laterally. Under the same kinematic loading, for the highest impingement configuration, 5mm of notchplasty was sufficient for eliminating all impingement.

Understanding how surgical variations affect ACL–intercondylar notch interactions is extremely important and has the potential to improve ACLR outcomes. Also, a better understanding of how notchplasty affects impingement can also improve ACLR. Improving surgical outcomes helps to reduce medical costs, and improves patient satisfaction.
6.5 Limitations

Some limitations do exist within this thesis. For example, each investigation was a subject specific study where only one knee model was used. Because each subject has unique anatomical geometries, the injury results may not be accurate with respect to other subjects. Future work could be performed on a variety of subjects to help understand how different geometrical characteristic affect ACL injury and cartilage damage.

In Aim 1 and Aim 2 a variety of loading conditions were simulated. However, knee flexion was fixed at 25° in each simulation. This was done because this flexion angle is most commonly associated with ACL injury. Future investigations may be conducted to see how the knee flexion angle affects ACL failure and cartilage damage. Also, sequential loading analyses were used, while simultaneous loading simulations may better simulate in–vivo ACL disruptions. Sequential loading was validated as no difference in V/V angle at ACL tear existed between sequential and simultaneous loading simulations. Muscle forces were included only through a reduction model which was simulated with a 3.6× body weight force. Because displacement controlled simulations were used, the results determined the knee joint motions causing knee joint tissue injury.

In Aim 3, only one knee kinematic scenario was used to explore ACL–intercondylar notch impingement. However, the kinematic data used simulated a high impingement motion associated with athletic cutting maneuvers and ACL injury (Fung and Zhang, 2003; Olsen et al., 2004; Park et al., 2010). Using this motion, the model was validated
based on experimental data. Because of this validation, it was possible to compare all surgical variation configurations to this validated model. Anterior tibial translation is a motion known to increase ACL–intercondylar notch impingement. This motion was not directly applied to the model, however it was indirectly simulated due to anterior tibial insertion site shifting. Interestingly, these anterior tibial shift configurations lead to increased ACL–intercondylar notch impingement.

The material properties used throughout this thesis to model knee joint tissues were taken from previous publications (Blankevoort et al., 1991; Donahue et al., 2003, 2002; Fung and Zhang, 2003; Kiapour et al., 2014; Peña et al., 2006; Puso and Weiss, 1998; Wangerin, 2013; Yang et al., 2010b). Bone was modeled as rigid. Previous work determined that modeling bone as rigid versus deformable did not affect cartilage contact, and the greatest variation was less than 2% across all cartilage contact variables (Donahue et al., 2002). While cartilage is known to be a rate dependent material, it was modeled isotropic elastic. This was considered valid because the loading conditions applied in these investigations were much faster than the relaxation time constant of the in-vivo tissue (1,500 sec) (Armstrong et al., 1984; Donzelli et al., 1999; Eberhardt et al., 1990; Kiapour et al., 2014). Viscoelastic material properties were considered for the ACL, however for the range of physiologically accurate loading rates, the failure results did not change. While 3D structures were incorporated for the double bundle ACL structure, this was not incorporated for other ligaments. However, a 3D PCL was incorporated, and it was determined that this did not affect the ACL failure results.

It is known that ACL injury and cartilage damage will change the material prop-
erties of these soft tissues. A damage model was not included, as this is extremely complex requiring careful experimental investigations. Because damage was not included, the cartilage damage initiation results should be considered reliable; however the damage growth may be underestimated. In future investigations, including cartilage damage modelling will improve the accuracy of the concomitant cartilage damage associated with ACL injury.

Even with the existing limitations, the developed methods provide useful data which can be used by orthopaedic clinicians and the biomechanical FE modelling community. The failure locus data can be used to help prevent ACL injuries on a patient specific level. The concomitant articular cartilage damage data matched well with reported clinical data which shows damage occurring on the postero–lateral portion of the lateral tibial plateau. The ACL–intercondylar notch impingement data provides very valuable data explaining how minor variations in ACLR lead to significant changes in intercondylar notch impingement. Future applications using similar techniques can be used to improve other surgical procedures such as patellofemoral realignment and total knee replacement surgery.
6.6 Future Work

6.6.1 Patella Malalignment, and Tibial Tuberosity Relocation

Patella Malalignment

Patellofemoral malalignment is a condition where the patella is located laterally in the trochlear groove. Tibial tuberosity trochlear groove distance (TTTG) is defined by the medial–lateral distance between the most prominent point on the patella and the most anterior portion of the tibial tuberosity, seen in Fig 6.1a. A distance greater than 20 mm is the classical indication for a malaligned patella. Fig 6.1b and Fig 6.1c provide a comparison between a normal and a malaligned patella.
Figure 6.1: a) The red line is the line tangent to the posterior epicondyle. The blue line is the line perpendicular to the red line from the most prominent point of the patella. The green line is the line perpendicular to the red line from the tibial tuberosities most anterior point. TTTG is the distance between the blue and green lines. b) Normally aligned patella. c) Patella exhibiting malalignment.

Patellar instability is exhibited by a reoccurrence of patellar dislocations from the trochlear groove. Patellofemoral malalignment is associated with patellar instability and trochlear dysplasia. It has been observed that 56% of individuals with a history of patellar dislocation have a TTTG greater than 20\text{mm} compared with a mean of 12\text{mm} in a control group (Dejour et al., 1994; Goutallier et al., 1977). Changes in the mechanics of the patellofemoral joint can result in changes in the tibiofemoral compartments (Feller et al., 2007). In 90% of patellar dislocations there is a rupture of the medial patello-femoral ligament (MPFL). This ligament is the primary
restraining structure against patellar lateralization. Repeated patellar dislocation is known to lead to severe arthritis as well as chondromalacia patellae which is a painful, degenerative disease which softens the patellar cartilage.

Tibial tubercle medialization is often performed in symptomatic patients with patellar malalignment. Studies have explored the influence of tibial tuberosity medialization and anteromedialization on patellofemoral contact pressure patterns (Benvenuti et al., 1997; Cohen et al., 2003; Elias et al., 2006, 2004). A study by Mani et al. (2011) demonstrated that medialization of the tibial tuberosity primarily rotated the tibia externally, compared to the lateral malalignment condition. Mani et al. (2011) quantified the variations in tibiofemoral translations and rotations related to tibial tuberosity medialization but did not look at contact pressure. No studies have investigated the effect of progressive increase in TTTG distance on the biomechanics of the patellofemoral joint.

Using 3D FE modelling in conjunction with in vitro testing, this research will study the effect of patella malalignment on patellofemoral contact stress patterns. This investigation will also determine the effectiveness of tibial tubercle management on restoring patellofemoral cartilage stress patterns. Another goal of this study is to determine the effect of isolated MPFL reconstruction versus tibial tuberosity medialization for patients exhibiting patellar instability due to increased TTTG distance. The aim of this investigation will be to determine the TTTG threshold for when MPFL reconstruction should be performed. The results of the study will quantify the amount of tibial tubercle medialization needed to restore proper patellar tracking in a subject specific model.
Finite Element Model Construction

A subject–specific 3D finite element model of a right knee was adapted from the *Open Knee* project developed at the Cleveland Clinic, seen in Figure 6.2 (Erdemir, 2013). Similar models were successfully used in previous investigations (Homyk et al., 2012; Orsi et al., 2015; Yang et al., 2010a, 2009). The *Open Knee* model includes 3D geometries for the following knee joint structures:

- Bone (femur, tibia and patella)
- Cartilage (femoral, patellar, lateral tibial, medial tibial)
- Meniscus (lateral and medial)
- Ligament (ACL, PCL, LCL, MCL)
- Tendon (patellar)

3D CAD models of the *Open Knee* geometries have been imported into ABAQUS and converted to an FE mesh. Improvements were added to this model by including the quadriceps muscle group. The five major components of the quadriceps muscle group include the vastus lateralis obliquus (VLO), the vastus lateralis longus (VLL), the rectus femoris and vastus medialis (RF–VI), vastus medialis longus (VML), and the vastus medialis obliquus (VMO), seen in Figure 6.2a.

Bones were modeled as rigid shells, as they are much stiffer than the soft tissue they interact with. A free meshing technique was used for the cartilage, meniscus and ligament using four–node linear tetrahedral elements. The articular cartilage
Figure 6.2: a) Frontal view, b) medial view of right knee. Modified *Open Knee* model including patella, patella tendon, and the five members of the quadriceps muscle group.
was modeled as isotropic linear elastic and the meniscus was modeled as transversely isotropic linear elastic. Linear elastic material properties were appropriate in this study as similar techniques have been used to model cartilage and meniscus in several previous studies. The menisci were attached to the tibial plateau at the meniscal horns using linear spring element sets, similar to previous methods. A transverse ligament was modeled as a single spring element, which attached the anterior horns of the menisci to each other (Donahue et al., 2003; Fung and Zhang, 2003; Homyk et al., 2012; Orsi et al., 2015; Yang et al., 2010b).

The quadriceps tendon group were modeled as linear elastic shells with stiffness properties taken from previous work (Merican et al., 2009). Preliminary investigations modelled ligament using non-linear tension only springs to increase computational efficiency. Eventually, a transversely isotropic hyperelastic material model will be used, similar to our previous investigations (Orsi et al., 2015).

Several contact interactions exist between the patellar articular cartilage, femoral articular cartilage, the tibial articular cartilage and the medial and lateral menisci as well as the quadriceps muscle group and the femur. All contact was modeled using a frictionless finite-sliding formulation where separation and sliding of finite amplitude and arbitrary rotation of the surfaces are allowed. Contact interaction normal to the contacting surfaces was constrained using the standard penalty enforcement method.

Preliminary simulations have been performed with the model undergoing $0^\circ$–$120^\circ$ of passive knee flexion, mimicking an in-vitro study performed by Merican et al. (2009). In these simulations, the quadriceps tendons undergo a pretension of 175 N along their principal axis. These axes are defined from the literature (Merican et al.,
The distribution of tension across the quadriceps group was as follows; RF–VI: 35%, VLL: 33%, VLO: 9%, VML: 14%, and VMO: 9% (Merican et al., 2009). Quadriceps muscle force was held constant throughout flexion (Merican et al., 2009). The femur is fixed and the tibia was rotated 120° around the TEA. All degrees of freedom were free for the tibia except for flexion. The patella was left unrestricted.

**In Vitro Testing**

Six fresh-frozen cadaveric knees were dissected, and the quadriceps was separated into five components: RF–VI, VLL, VLO, VML, and VMO. The cadaver was fixated to the testing apparatus with set screws which are drilled through the femoral shaft. Tension was applied separately to each of the five muscle groups by a cable, weight, and pulley system in accordance with directions and physiological cross sectional areas as conducted in the FE model, shown in Figure 6.3. A total load of 175N was applied to the muscle groups with the same distribution of tension as in the FE model. A sensor (Tekscan 5040) was placed between the patella and the trochlea through an incision in the superior patellofemoral synovial pouch and secured in position with sutures. The cadaver testing is performed in a motion capture environment with optical trackers placed on the patella, femur and tibia. Patellar motion is post processed and described in relation to the femur.

Several configurations of TTTG distance and MPFL reconstruction will be tested with both the FE model and the cadaver specimens, which are outlined in Table 6.1.

The FE model and the *in–vitro* testing will both model patellar instability, tibial tuberosity medialization, and MPFL reconstruction. To model patellar instability,
the tibial tuberosity will be relocated laterally in the FE model and will be surgically transposed on the cadaver specimens. Tibial tuberosity medialization will be performed in the same way. To model MPFL reconstruction, spring elements of a hamstring graft (30N/mm) will be added to the FE model. To perform MPFL reconstructions \textit{in-vitro}, and a semitendinosus graft will be fixated to the patella, using trans-osseous sutures, and to femur, using interference screws.
Preliminary Results

A baseline FE model has successfully converged at 120° of knee flexion. However, it will be necessary to validate this model based on the in–vitro testing which is currently ongoing. Nevertheless, the preliminary results show the patellofemoral contact patterns throughout knee flexion, which can be seen in Figure 6.4.

In the early stages of knee flexion, 0°–30°, patellar cartilage stress is seen in the
medial facet. From 45°–75°, stress pattern moves to the superior region of both the medial and lateral facets. In deeper knee flexion, 90°–120°, the stresses increase and move distally and medially on the medial facet and distally and laterally on the lateral facet. These stress patterns will be used as a baseline to compare several configurations of patella malalignment against.

Understanding how different anatomical factors and reconstructions both in isolation and in combination affect patellar dislocation can improve treatment in patients with these conditions. Identifying how these factors affect patellar stability may allow physicians to predict which patients are more likely to sustain recurring injuries. Determining which indices correlate with inadequate patellar engagement may allow physicians to use a standardized method to predict patients at greater risk of patellar dislocation. Attempting to link patella malalignments with patellofemoral instability may guide physicians to choose suitable treatments for individual patients.

6.7 Final Words

With an active and healthy young population, the annual number of ACL injuries is expected to rise (Boden et al., 2000b, 2009; Griffin et al., 2006). Roughly 90% of ACL injured patients elect to have ACLR, which combined with rehabilitation creates an annual $1 billion expense in the U.S. (Boden et al., 2000a, 2009; Griffin et al., 2006; Longo et al., 2008; Meredick et al., 2008; Sajovic et al., 2006). These numbers do not account for the associated costs of osteoarthritis linked to ACL injury. Because these injuries are such a heavy burden on the healthcare system, it is necessary to
understand how they occur, to help prevent and better treat them in the future. It is also necessary to understand the effectiveness of current reconstruction and rehabilitation strategies.

Using biomechanical joint modelling is a cost effective way to investigate soft tissue failure mechanisms and surgical interventions. While cadaver testing is necessary for model validation, it is expensive due to the fact that only one failure experiment is possible per cadaver. FE models can be used to investigate several knee joint tissue failure scenarios for individual specimens. Doing so allows for the detection of the kinematic conditions most likely to cause injury for individual subjects. FE models also allow for the simulation of several surgical configurations for individual subjects. Recommendations can be made from these results to improve surgical techniques for individual subjects. This is clinically beneficial because improving surgical success rates improves patient well being and reduces medical costs.

Improvements are needed for FE studies investigating soft tissue injuries and surgical interventions. Because individual subjects exhibit unique physical characteristics (bone size, ligament size, notch width, etc.), the kinematics leading to failure may differ between subjects. A larger sample size of FE models, using subjects with unique physical characteristics, may improve the understanding of failure mechanisms. Also, simulating a wider variety of kinematic conditions, such as varying degrees of knee flexion, will improve the failure mechanisms knowledge. Using larger sample sizes will allow for improved recommendations for surgical interventions. Because of the variety of physical characteristics between subjects, different interventions may be required for specific patients.
3D FE models are useful tools for predicting surgical outcomes as was shown in chapter 4. It is recommended that surgical variations guide biomechanical FE modelling research in the future. This line of research provides a clinically relevant use for 3D FE modelling. A wide variety of surgical reconstruction techniques for various injuries exist which all aim to restore proper biomechanical function after injury. Variability is inevitable in surgery due to human limitations. Understanding the benefits and pitfalls between surgical techniques and understanding how human error affects surgical outcomes are both extremely important. FE investigations of surgical variations have the potential to improve surgical outcomes.

The methods presented in this thesis are useful for orthopaedic professionals. The presented data will benefit orthopaedic surgeons by providing them with an improved understanding of knee joint injury mechanisms. The data provided also has the potential to improve surgical procedures such as ACLR. Physical therapists will benefit from these investigations as the data provides information which can improve rehabilitation techniques and preventative training techniques. Biomechanical engineers will benefit by understanding how to better simulate knee joint injuries and surgical variations to help prevent injuries and improve surgical procedures. As computational power increases, these methods will become more cost effective, increasing our ability to understand even more about the mechanisms of soft tissue injury.
REFERENCES


APPENDIX A

FEBio vs. ABAQUS User Subroutine Validation

ABAQUS and FEBio were used for the different investigations in this thesis. FEBio was used for its convenient integration of rate dependent material models for ligament material (Maas et al., 2012). ABAQUS was used for its robust abilities which aided in convergence of the complete knee model including multiple contact interactions. FEBio includes the transversely isotropic Mooney–Rivlin hyperelastic material in their software package, however ABAQUS does not. Because of this, in order to incorporate this material into ABAQUS, a User Subroutine was used, which was provided by Dr. Estefanía Peña from the University of Zaragoza, Spain based on the constitutive model developed by Weiss et al. (1996). The material coefficients were taken from Peña et al. (2006), and can be seen in Table A.1. Because the same ligament material model was used in two softwares, it was necessary to compare the behavior of the material models between the two. The same material parameters were
Table A.1: Material coefficients for the ACL

<table>
<thead>
<tr>
<th>$C_1 (MPa)$</th>
<th>$C_2$</th>
<th>$C_3 (MPa)$</th>
<th>$C_4$</th>
<th>$C_5 (MPa)$</th>
<th>$\lambda^*$</th>
<th>$K$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.95</td>
<td>0.0</td>
<td>0.0139</td>
<td>116.22</td>
<td>535.039</td>
<td>1.046</td>
<td>1950</td>
</tr>
</tbody>
</table>

incorporated for each software package, and FE simulations were simulated using a one element simulation, and further a complete ACL model.

A single element test was performed using both FEBio and ABAQUS in which both an axial and a shear test were performed. The results from the axial test can be seen in Figure A.1. The results from the shear test can be seen in Figure A.2.

The single element test results show that the FEBio material model and the ABAQUS User Subroutine compare well between each other.

An ACL model was also compared for a simulation involving $15^\circ$ internal rotation with varus loading. The results from this, observed in Fig.A.3, show that the FEBio and ABAQUS model compared well.
Figure A.1: Single element axial stress test.
Figure A.2: Single element shear stress test.
Figure A.3: FEBio vs. ABAQUS ligament stress test.
APPENDIX B

Mesh Sensitivity Analysis

B.1 ACL Mesh Convergence

Three mesh densities were used for the two bundles of the ACL (0.8\text{mm}, 1.0\text{mm}, and 1.2\text{mm} spacing). One simulation was performed for each of these mesh densities. The simplified knee model including only the bones and the ACL was used. The simulation included the appropriate prestrain for each bundle, 25° of knee flexion, and 11.25° of valgus loading. Maximum and average von Mises stress was monitored in the midsubstance of the ACL for each mesh density configuration. Figure B.1 shows the region of interest for each of the ACL bundles.

Figure B.1 shows the von Mises stress in the region of interest for each of the ACL bundles, at each of the mesh densities. The stresses did not differ significantly between the mesh densities, and the results were quantified further.

The results provided in Figure B.3 show that across the three mesh densities, for both bundles of the ACL there was minimal difference in the von Mises stresses.
Figure B.1: a) Double bundle ACL structure showing amACL bundle in blue, and plACL bundle in purple. b) Isolated view of amACL bundle showing the region of interest used for the mesh convergence analysis. c) Isolated view of plACL bundle showing the region of interest used for the mesh convergence analysis.

Figure B.2: Stress profiles of midsubstance cross sectional areas of both bundles for the three mesh densities. The stress profiles exhibit similar results across the three mesh densities.
Figure B.3: a) Mesh convergence for amACL bundle. b) Mesh convergence for plACL bundle

Because of this, it was determined that a mesh density of 1.2mm spacing was adequate for the ACL. In our investigations, each of the mesh densities was used to determine the most efficient run time for our simulations. Using the mesh density of 0.8mm spacing did not significantly increase the simulation time. Because of this, either mesh density can be selected as the 1.2mm did not change the results, and the 0.8mm provides a more accurate representation of the geometry, and it does not significantly increase simulation time.
Three mesh densities were used for the tibial articular cartilage (0.8\text{mm}, 1.0\text{mm}, and 1.2\text{mm} spacing). The complete knee model which included all tissues (cartilage, meniscus, ligament, bone) was used to perform this mesh convergence for the tibial articular cartilage. One simulation was performed for each of these mesh densities. The simulation included the appropriate prestrain for each bundle, and 725N of body weight loading. Tresca stress was monitored in the articular cartilage for each mesh density configuration. Figure B.4 shows the Tresca stress in the lateral tibial articular cartilage for each of the mesh densities.

The bulk of the contact area on the lateral tibial cartilage is showing very similar Tresca stress values, around 1.5\text{MPa}. However, the results provided in Figure B.5 show that across the three mesh densities there were minor differences in the maximum Tresca stress values. The regions exhibiting these high stresses are very small, because of this, it was determined that a mesh density of 1.0\text{mm} spacing was adequate for
Figure B.5: Mesh convergence for lateral tibial articular cartilage. During the time step, from 0 to 1 seconds, the model is undergoing ligament prestrain. From 1 to 2 seconds the model is undergoing body weight loading.

Simulations were performed using all three mesh densities, and it was determined that increasing the mesh density did not significantly increase the computational time. Because of this, the mesh density of 0.8mm spacing was used for the cartilage.
REFERENCES

