Biomechanical and neuromuscular adaptations in those with anterior cruciate ligament reconstruction during functional movements

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Biomechanical and neuromuscular adaptations in those with anterior cruciate ligament reconstruction during functional movements

by

Michelle Hall

A thesis submitted to the graduate faculty
in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

Major: Kinesiology (Biological Basis of Physical Activity)

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Iowa State University
Ames, Iowa
2010

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ABSTRACT

Individuals with anterior cruciate ligament (ACL) reconstruction are at increased risk to develop knee osteoarthritis (OA). Gait analysis including kinetics and electromyography of walking and stair use can provide insight to everyday knee joint dynamic loading. Previously, those with ACL rupture have shown altered gait patterns up to one year post-surgery.

PURPOSE: To compare lower extremity gait patterns of those with ACL reconstruction (>1 yr) to a control group. We hypothesized that the ACL group would have 1) reduced knee extensor strength, 2) reduced knee flexion angles, 3) reduced knee extensor moments, 4) increased hip extensor moments, 5) increased external knee varus moments, 6) reduced knee extensor activity, 7) increased knee flexor activity, 8) increased hip extensor activity, 9) increased quadriceps:hamstring co-contraction, and 10) altered medial and lateral thigh muscle activity patterns when compared to the control group.

METHODS: Eighteen ACL reconstructed individuals (26 ± 6 years, 6 ± 4 years from surgery) and 18 healthy controls (26 ± 4 years) participated in this study. Participants performed three ascending and descending trials on a three step staircase and three walking trials leading with the right and left leg, respectively. Kinematic and kinetic recordings were collected using an 8-camera motion analysis system, and portable force platforms were positioned on the first and second stair steps. Reflective markers were placed on the lower extremities and trunk. Electromyography (EMG) data were collected from the vastus lateralis [VL], vastus medialis [VM], biceps femoris [BF], semimembranosus [SM], and gluteus maximus [GMax]. Using inverse dynamics, internal hip abduction moments, hip extension moments, knee extension moments, and external knee varus moments were calculated during the stance phase of walking for stair ascent and descent (two steps). Maximum
moments were averaged across trials and normalized to body mass. A linear envelope (10 Hz low-pass filter) was used to determine maximum EMG values during the stance phase of each step. Maximum EMG values were averaged across three trials and normalized to the MVIC. Co-contraction ratios were determined for knee extensors and flexors (VL+VM and BF+SM) and for medial and lateral muscle activity (VM and SM, VL and BF, respectively). One-way ANOVAs were used to test for main effects of group (ACL and control) on maximum joint moments and EMG activity. Significance was set at p<0.05.

**RESULTS:** During walking and stair use (on the second step) those with ACL reconstruction exhibited lower knee extensor moments and greater hip extensor moments compared to the control group. Hip extensor EMG activity was greater in the ACL reconstruction group during walking and stair use. Knee flexor activity was increased during walking and the first step of stair ascent. Increased medial thigh co-contraction was found in the ACL group during walking and increased knee extensor and flexor co-contractions was found during the first step of stair ascent.

**CONCLUSION:** Walking and stair ambulation highlight altered knee joint loading patterns in those with ACL reconstruction surgery. Individuals compensate for lower knee extensor moments by increasing hip extensor moments, and this was reflected in EMG data. Reduced knee extensor moments may protect the repaired ACL from excessive strain or may result from impaired neuromuscular control. Those with at least one year post-ACL reconstruction increase medial knee compression during walking.
CHAPTER 1
GENERAL INTRODUCTION

Anterior cruciate ligament (ACL) injuries are among the most common musculoskeletal injuries, occurring more frequently in young adults, especially women. Individuals younger than 30 years sustain ACL ruptures more often compared to the general population (Lohmander et al., 2007; Englund, 2010). Rupture of the ACL is often concurrent with meniscal injuries (Lohmander et al., 2007) and where combined injuries occur, knee osteoarthritis (OA) develops in up to 48% of cases (Louboutin et al., 2009; Lohmander et al., 2007). Knee OA is estimated to develop 5-20 years after initial ACL injury. Therefore, individuals between 35-40 years old are at increased risk of knee OA following ACL rupture (Englund, 2010).

Structural changes associated with ACL injury combined with long-term changes in dynamic loading contribute to the development of knee OA (Lohmander et al., 2007). The focus of this study is to investigate long-term changes (greater than one year post-ACL rupture) in dynamic knee joint loading and to identify any associated neural compensation strategies during walking and stair negotiation. Long-term changes in joint loading alter knee biomechanics, increasing shear forces on medial aspect of the knee (Louboutin et al., 2009).

ACL reconstruction research focuses on gait during level walking (Butler et al., 2009; Knoll et al., 2004b; Timoney et al., 1993; DeVita et al., 1997), with fewer investigating stair ascent and stair descent (Kowalk et al., 1997; Hooper et al., 2002). Stair use is important to consider, as it is a common daily activity associated with increased knee joint stress, particularly during stair descent (Lu and Lu, 2006). Additionally, gait analyses to date have generally focused on the timeline to return to typical gait and how those with ACL deficiency
adapt their gait (Knoll et al., 2004b; Timoney et al., 1993; DeVita et al., 1997; Kowalk et al., 1997; Hooper et al., 2002). A recent study investigating gait of ACL reconstructed individuals five years from surgery focused on possible gait adaptations that attribute to the development of knee OA (Butler et al., 2009).

Reconstructive ACL surgery does not entirely result in typical gait patterns. Individuals 6-12 months post-ACL reconstruction have altered walking patterns as compared to controls, including reduced knee flexion angles at midstance (Timoney et al., 1993; Hooper et al., 2002) and reduced internal knee extensor moments during early stance (DeVita et al., 1997; Timoney et al., 1993). These gait adaptations may lead to degenerative changes in the articular cartilage by altering the loading patterns on the knee joint. Decreased knee flexion angles and internal knee extensor moments have been associated with quadriceps weakness in individuals up to 1 year post-ACL surgery (Lewek et al., 2002; Bush-Joseph et al., 2001). Despite ACL reconstruction and aggressive rehabilitation regimes, weakness in quadriceps musculature exists up to 1 year post-ACL reconstruction surgery (Suter et al., 2001). Large cross-sectional studies have established strong relationships between quadriceps weakness and the onset of knee OA (Becker et al., 2004; Slemenda et al., 1997). It has yet to be determined if quadriceps weakness, reduced knee extensor moments, and reduced knee flexion angles persist long-term (greater than one year) in those with ACL reconstruction.

In addition to movement control and load distribution, muscle activation patterns play a role in stabilizing the knee joint. Following ACL rupture and any associated meniscal damage, greater quadriceps and hamstring co-contraction may be needed to stabilize the knee joint. However, increasing co-contraction increases compression forces on the articular cartilage, perhaps initiating articular degeneration (Schipplein and Andriacchi, 1991). Higher
quadriceps:hamstring co-contraction of has been observed in those with knee OA as compared to a control group (Zeni et al., 2010). Also, medial-to-lateral quadriceps:hamstring co-activation patterns have been found to increase with increasing severity of knee OA (Hubley-Kozey et al., 2009). Zhang and Wang (2001) articulate that lateral quadriceps and hamstring activity is necessary to resist external knee varus moments. Quadriceps:hamstring co-contraction and medial-to-lateral quadriceps:hamstring co-activation patterns may be altered in those with ACL reconstruction in an attempt to stabilize the knee joint. EMG co-activation patterns during walking and stair negotiation may provide insight to how loading is resisted in this population.

Arguably, external knee varus moments are of greatest clinical interest when considering potential knee OA risk factors. Knee varus moments are associated with medial knee joint compression, which is characteristic of OA. Miyazaki et al. (2002) established that the risk of knee OA progression increased by a factor of 6 with a 1% increase in external knee varus moments. More recently, attention has focused on the role of the hip abductors in protecting against knee OA. Impaired hip abduction moment generation causes contra-lateral pelvis drop, increasing knee varus moments (Chang et al., 2005; Bennell et al., 2007). Butler et al. (2009) observed 21% greater knee varus moments during walking in individuals five years following ACL reconstruction when compared to a control group, although no changes in frontal hip mechanics were observed. In contrast, Hooper et al. (2002) found no differences in knee varus moments while walking or using the stairs when comparing the injured and non-injured leg 12 months post-ACL reconstruction. These aforementioned studies suggest that increased knee varus moments may not be present within the first year of ACL rupture, but medial knee joint loading may increase as time progresses. Further investigation is required to assess dynamic knee joint loading post-ACL surgery during walking and stair
ambulation. In particular, those greater than one year post-ACL reconstruction are of interest.

The purpose of this study was to compare gait of ACL reconstructed individuals to a healthy control group during three tasks: stair ascent, stair descent and level walking. The primary focus was to determine if factors associated with the development and/or progression of knee OA were observed in ACL reconstructed individuals when compared to healthy counterparts. We hypothesized that the ACL group would display: 1) reduced knee extensor strength, 2) reduced knee flexion angles, 3) reduced knee extensor moments, 4) increased external knee varus moments, 5) increased quadriceps:hamstring co-contraction, and 6) altered medial and lateral quadriceps:hamstring co-contraction patterns when compared to the control group.
CHAPTER 2
REVIEW OF LITERATURE

1.1. Incidence and Prevalence of Knee Osteoarthritis following ACL Rupture

Anterior cruciate ligament injury (ACL) is among the most common musculoskeletal injuries. Injury often occurs in young individuals while playing sports including soccer, basketball, football and team handball (Lohmander et al., 2007). Increasing evidence indicates that ACL and menisci injuries are associated with knee osteoarthritis (OA) development (Louboutin et al., 2009). Incidence rates are considerably higher in those physically active under 30 years of age when compared to the general population (Lohmander et al., 2007). Due to increasing rates of obesity and cardiovascular disease, governments globally advocate the benefits of being physically active and encourage regular physical activity. Increased participation in physical activity suggests higher incidence of sports related injuries including ACL injury. Therefore, it is imperative that research aims to prevent knee OA onset by identifying causes that initiate knee OA following ACL rupture. (Palmieri-Smith and Thomas, 2009).

Osteoarthritis is a complex degenerative disease of the skeletal system with the tibiofemoral joint being the most common location of OA in the United States (Palmieri-Smith and Thomas, 2009). Knee OA is defined by joint symptoms, and/or radiographic evidence of structural pathology. Radiographic features of OA include: joint space narrowing, osteophytosis, subchondral sclerosis, cyst formation and abnormalities of bone contour (Arden and Nevitt, 2006). Pain, stiffness, and loss of function are commonly reported symptoms.
A systematic review suggests incidence rates of knee OA following an isolated ACL rupture are between 0-13% (Oiestad et al., 2009). However, isolated ACL injuries are uncommon and injury to the ACL is often associated with injuries to the menisci, other ligaments, joint cartilage, and subchondral or cancellous bone (Lohmander et al., 2007). Meniscal injuries at the time of ACL rupture are identified as a primary risk factor for the development of posttraumatic knee OA (Shelbourne and Gray, 2000; Gelber et al., 2000). This is highlighted by knee OA prevalence increasing considerably (21-48%) if ACL injury occurs with meniscal lesions (Neuman et al., 2009; Oiestad et al., 2009). In addition to meniscal damage, a combination of high physical activity levels and knee joint laxity contributes to the onset of knee OA following ACL rupture (Neuman et al., 2009).

Between 5-20 years following the initial injury, the onset of knee OA is likely. Rupture of the ACL is estimated to age the knee by approximately 30 years (Louboutin et al., 2009). In a cohort of female soccer players 12 years after ACL rupture, 75% reported knee pain symptoms that impacted their quality of life while 42% had radiographic evidence of knee OA (Lohmander et al., 2004). Considering the prevalence of knee OA is around 50% when ACL rupture is concurrent with meniscal injuries, the development of knee OA is a reality for many young individuals. Hence, it is necessary to target young individuals sustaining knee injuries in preventing the development of this debilitating disease (Gelber et al., 2000).
1.2 Functional Anatomy

Fig. 1. Overview of the human right knee joint seen from anterior view. This images are from Medicine Net (2004).

1.2.1 The ACL

The knee joint is a complex organ that permits multiple degrees of freedom of joint motion including internal-external rotation, flexion-extension, and varus-valgus movements. The direction of knee joint movement is largely controlled by its ligaments and geometric constraints of the articular surfaces. Ligaments consist of water, non-collagenous proteins, proteoglycans, elastin, and densely packed collagen fibers (Woo et al., 1999).

Proteoglycans and water provide lubrication aiding the gliding function of the joint. Type I collagen fibers run in a longitudinal direction, parallel to the axis of loading (Duthon et al.,
The crimp arrangement of the collagen fibrils enables the ACL to elongate in tension, permitting smooth movement. However, during movements such as running, jumping, and pivoting external applied loads are increased with increased ligament stiffness preventing excessive motion in the joint (Woo et al., 1999). Type II collagen is also found in the ACL specifically at the tibial and femoral sites of attachment, indicating areas of exposure to pressure or shear force (Duthon et al., 2006). The ACL is intra-articular (Fig. 1), connecting the femur to the tibia and resisting anterior tibial translation and rotational loads (Andriacchi et al., 2004). The unique structural properties of the ACL permit it to withstand multi-axial forces ranging between 2100-2500 N (Duthon et al., 2006; Chhabra et al., 2001). The ACL also plays a sensory role in detecting knee joint loading. See Table 1 for ACL innervations.

<table>
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<tr>
<th>Receptors of Nerve Fibers</th>
<th>Sensitivity &amp; Location</th>
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<tr>
<td>Ruffini Receptors</td>
<td>Sensitive to stretching, located predominantly on the femoral portion where the deformations are the greatest</td>
</tr>
<tr>
<td>Vater-Pacini Receptors</td>
<td>Sensitive to rapid movement, located at the femoral and tibial ends of the ACL</td>
</tr>
<tr>
<td>Golgi-like Receptors</td>
<td>Sensitive to tension, located near the attachment of the ACL and the surface</td>
</tr>
<tr>
<td>Free-nerve endings</td>
<td>Act as nociceptors, but also release neuropeptides with vasoactive function</td>
</tr>
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**Table 1. ACL Innervations**  
Reproduced from Duthon et al. (2006)

The primary role of the ACL is to resist anterior tibial translation (Duthon et al., 2006). Under normal conditions, the quadriceps extensor mechanism (by way of patella tendon) pulls the
tibia anteriorly and the ACL restrains anterior translation of the tibia (Fig. 1) (Chhabra et al., 2001). When the ACL is ruptured, the remaining structures to restrain anterior translation include the medial tibial plateau, the posterior horn of the meniscus, the posterior ligament-capsular structures and the hamstrings (Louboutin et al., 2009; Ageberg et al., 2009). In chronic ACL deficient knees, anterior translation of the tibia relative to the femur is up to four times greater than in typical knees (Beynnon et al., 2002), highlighting the contribution of the ACL to knee joint stability in the sagittal plane. In addition, the ACL functions as a major secondary restraint to internal rotation, particularly when the knee is near full extension (Duthon et al., 2006). At 20° of knee flexion, an internal rotation moment of 10 Nm produced 100 N force in the ACL, while an external rotation moment of 10 Nm produced 50 N force in the ACL (Woo et al., 1999). It is apparent that the ACL is critically involved in knee joint stability.

1.2.2. Articular Cartilage

Joint mechanics is considered a critical component initiating knee OA, as biomechanical factors mediate the gross and chemical structure of articular cartilage (Shakoor and Moisio, 2004). Osteoarthritis is characterized by articular cartilage degeneration, subsequently followed by joint space narrowing, pain, and impaired function. Specifically, articular cartilage functions to absorb loading and protect the bone ends from degradation. Sensitivity of articular cartilage is characterized by its response to mechanical loading. Optimal articular cartilage health can be maintained through exposure of cyclical loads within a physiological zone; however, cartilage integrity is compromised by reduced or excessive loading (Whiting and Zernicke, 2008).
In a review, Arokoski et al. (2000) details the response of articular cartilage to mechanical loading. Articular cartilage is composed of an interacting solid matrix and interstitial water. The solid matrix includes primarily Type II collagen and proteoglycans (PGs). Swelling of the articular cartilage results from an electromechanical force that is created by an interaction between the interstitial water and the matrix. Specifically, PGs repel each other due to their negative charge and attract interstitial water into the matrix causing the articular cartilage to swell. Collagen resists shearing and tension by retaining its shape, developing tension, and transmitting the articular cartilage swelling. Arokoski et al. (1999) estimates intrinsic cartilage stiffness at 0.5-1.0 MPa, whereas dynamic articular stiffness can be 10 times the intrinsic modulus (Jurvelin et al., 1997). Articular cartilage is a complex tissue, with properties to attenuate considerable loads that would otherwise be imposed on bone.

1.3 Injury to the ACL

A combination of biochemical alterations and long-term changes in dynamic joint loading contribute to the development of OA (Louboutin et al., 2009; Lohmander et al., 2007). Once a knee joint injury occurs, a series of intra-articular pathogenic processes take place in an attempt to repair the damage. Within days of injury to ACL or meniscus, damage to the type II collagen network, aggrecan, and other matrix components of the joint cartilage are present. In a cohort of more than 250 individuals with ACL and/or meniscus injuries, the concentration of aggrecan fragments (biomarkers, indicative of cartilage synthesis) was increased from initial injury to 20 years post injury when compared to a control group (Lohmander et al., 1999). Biomarker levels generally decrease with time following knee injury; however, levels are similar to those in an osteoarthritic joint (Lohmander et al., 2007). It is likely that biochemical disorganization contributes to knee OA development despite a successful ACL reconstruction (Lohmander et al., 2007).
Overall knee joint stability depends on the contributions of individual ligaments including the ACL, posterior cruciate ligament, medial collateral, and lateral collateral ligaments (Fig. 1) as well as the interaction of between ligaments (Woo et al., 1999). Injury to the ACL compromises knee joint integrity by increasing stress on secondary restraints of anterior translation and rotation, including the menisci and other ligaments (Louboutin et al., 2009; Chhabra et al., 2001).

In addition to initial injury to knee joint structures, long-term changes induce alterations in the knee biomechanics that increase shear forces on the articular cartilage. Shear forces, particularly on the medial side of the knee, increase in the absence on the ACL (Louboutin et al., 2009). The posterior horn of the medial meniscus is a static restraint to anterior translation in the absence of the ACL. The integrity of this restraint becomes increasingly impaired as longitudinal splits form due to the meniscus being wedged between the tibia and the posterior condyle of the femur (Louboutin et al., 2009). Increasing shear forces lead to loss of meniscus and ultimately loss of articular cartilage. Hence, meniscal injuries are considered a significant risk factor for the development of knee OA after ACL injury (Oiestad et al., 2009).

Injury to the knee joint including ACL injury influences the congruency and laxity of the joint, which alters contact stress and joint motion and consequently impacts the mechanical environment of the cartilage (Andriacchi et al., 2004). Similar to muscle, cartilage degenerates if not subjected to ample use or stress. Joint surfaces are congruent in young people. Congruity maintains stability, physiologic loading, and joint nutrition (Bullough, 2004). The response of cartilage to load is dependent on the ability to maintain equilibrium between degeneration and synthesis of collagen fibrils and proteoglycans (Andriacchi et al., 2004). Evidence suggests that abnormal motion causes redistribution of loading on the
cartilage and initiates joint degeneration as the cartilage cannot remodel according to the redistribution loads (Bullough, 2004). A model-based study speculated that overloading specific regions of the joint, either because of altered contact mechanics or disrupted joint stability, may initiate knee OA following ACL rupture (Wu et al., 2000). It is possible that ACL injury disrupts joint loading through a combination of translational changes and rotational changes (Andriacchi et al., 2004). It is generally accepted that ACL reconstruction surgery restores anterior-posterior translation stability (Lohmander et al., 2007), although less is known about the restoration of normal rotational alignment. Ristanis et al. (2003) showed greater tibial rotation during a pivoting movement in the ACL reconstructed knee compared to intact knee one year after ACL reconstruction surgery.

1.4 Gait Analysis

As a measuring tool, gait analysis is considered a practical and reliable technique to indirectly assess dynamic loads on the lower extremity (Shakoor and Moisio, 2004). Dynamic loading is referred to loads placed on the joints during physiological activity (Shakoor and Moisio, 2004). Gait analysis is used clinically to establish pathological gait patterns and subsequently used to develop and improve rehabilitation techniques. Indeed, gait analysis and EMG have recently been used to identify biomechanical changes associated with various levels of knee OA severity (Astephen et al., 2008). The ACL reconstruction literature often focuses on analyzing gait during level walking (Butler et al., 2009; Knoll et al., 2004b; Timoney et al., 1993; DeVita et al., 1997), with fewer investigating stair ascent (Kowalk et al., 1997; Hooper et al., 2002) and stair descent (Hooper et al., 2002). Stair use is a focus of this study considering that it is a common daily activity associated with increased knee joint stress, particularly during stair descent (Lu and Lu, 2006). Generally, gait analyses of ACL injuries focus on the timeline to return to typical gait
and how those with ACL deficiencies adapt their gait (Knoll et al., 2004b; Timoney et al., 1993; DeVita et al., 1997; Kowalk et al., 1997; Hooper et al., 2002). As a result of the increased risk of knee OA following ACL injuries after 5-20 years, it is of interest to assess dynamic knee joint loading in those with ACL reconstruction at least one year from surgery. Reports on dynamic loading during walking in this population group are limited to one study (Butler et al., 2009).

1.5 Neuromuscular Adaptations Following ACL Injury Leading to Knee OA

As mentioned previously, the long-term changes in dynamic loading contribute to the development of knee OA following ACL rupture. Therefore, it is the purpose of this study to identify kinetic risk factors that are associated with ACL injury that may initiate the onset of knee OA. A second objective is to compare neuromuscular control in those with ACL reconstruction to a healthy comparable population. In particular, neuromuscular impairments that alter joint loading at the knee are the focus of this section.

1.5.1. Quadriceps Weakness

Quadriceps weakness has been associated with the development and progression of knee OA (Palmieri-Smith and Thomas, 2009). Large cross-sectional studies have established strong relationships between knee extensor weakness and the onset of tibiofemoral and patellofemoral knee OA for both men and women (Becker et al., 2004; Slemenda et al., 1997). Quadriceps function is important for walking (Palmieri-Smith and Thomas, 2009), standing, and climbing stairs (Becker et al., 2004), and plays an integral role in protecting the cartilage of the knee from excessive loads. Quadriceps muscles function to attenuate shock and to distribute loads across the joint articular surfaces. Eccentric control of the quadriceps while the knee is flexed immediately after foot ground contact attenuates the
ground-reaction force which otherwise would travel through the articular surface of the knee (Becker et al., 2004). Quadriceps muscles load the articular cartilage as the knee extends (Palmieri-Smith and Thomas, 2009). Weak quadriceps muscles contribute to joint degeneration due the inability of extensor muscles to attenuate shock and distribute loads across the joint (Palmieri-Smith and Thomas, 2009). It is unknown if changes in joint loading or loss of muscular control causes the onset and progression of joint degeneration (Herzog et al., 2003). However, it is apparent that muscle weakness and altered joint loading is associated with ACL rupture (Palmieri-Smith and Thomas, 2009).

Quadriceps weakness persists in those with ACL reconstruction, and it is generally attributed to neural activation deficits. Despite ACL reconstruction and aggressive rehabilitation regimes, weakness in knee extensor musculature exists three months (Drechsler et al., 2006) and up to one year post-ACL reconstruction surgery (Suter et al., 2001). Lewek and colleagues (2002) observed 80% of quadriceps strength in the involved limb compared to the uninvolved limb in patients three months after ACL reconstruction. Additionally, extensor weakness exists in both the injured limb and in the healthy contralateral limb (Suter et al., 2001). Quadriceps weakness is caused by a combination of muscle atrophy and reduced voluntary muscle activation (Petterson et al., 2008). Quadriceps activation deficits are defined as a decrease in voluntary isometric quadriceps knee extension moments compared with the moments produced during superimposition of an electrical stimulus on a maximum voluntary isometric contraction (Petterson et al., 2008). Voluntary quadriceps activation is reduced in individuals with knee pain and radiographic evidence of OA, compared to healthy subjects or non-diseased contralateral limbs (Becker et al., 2004). Decreased muscle activation results from arthrogenic muscle inhibition, which is a natural response designed to protect the joint from further damage (Hopkins et al., 2001).
Altered neuromuscular control of the quadriceps is likely to affect gait patterns. Chmielewski et al. (2004) conducted a prospective analysis of muscle inhibition in a consecutive sample of 100 patients with ACL rupture. Six weeks following injury, quadriceps inhibition of the injured limb was observed in 33% of cases and interestingly 31% of cases also demonstrated inhibition in the uninjured limb. Suter et al. (2001) also assessed muscle inhibition and concluded persistent inhibition may be responsible for quadriceps weakness. Furthermore, extensor inhibition is suggested to alter kinetic and kinematic variables during walking that are likely to be associated with premature articular degeneration (Suter et al., 2001).

Similar to Chmielewski et al. (2004), Lewek et al. (2002) observed inhibition in both the injured and uninjured limb. These findings have implications not only for changes in the dynamic loading patterns, but also in the design of gait analysis studies that use the non-injured leg as a control.

Gait analysis of walking has indicated alterations in gait during weight acceptance, including reduced knee flexion at midstance (Timoney et al., 1993; Hooper et al., 2002) and reduced internal knee extensor moments during early stance compared to controls (DeVita et al., 1997; Timoney et al., 1993). These studies did not perform knee extensor strength assessment. The aforementioned studies performed analysis on subjects between 6-12 month post-ACL reconstruction, and it has yet to be determined if similar findings persist long-term (greater than one year).

Quadriceps weakness is associated with decreased knee flexion angles and decreased internal knee extensor moments in individuals up to one year post ACL surgery (Lewek et al., 2002; Bush-Joseph et al., 2001). Evidence suggests a reduction in knee flexion angle
may increase the risk of inducing degenerative changes to the articular cartilage as it
impairs the ability of the knee to absorb shock during weight acceptance (Cook et al., 1997).

During common daily activities, altered kinetics and kinematics have been found in
individuals with ACL reconstruction. Hooper et al. (2002) reported decreased knee flexion
angles at heel strike during stair ascent, and no differences were observed while
descending stairs. Stair ascent analyses demonstrate reductions in peak internal extensor
moments, power, and work performed at the injured knee highlighting gait asymmetries in
those with ACL reconstruction (Kowalk et al., 1997). A reduction in peak knee extensor
moments is suggestive of impaired quadriceps function altering loading patterns. In addition
to data from stair use, reductions in knee extensor moments have been observed in
subjects with ACL reconstruction during functional activities such as single-leg vertical jump
and lateral step-up (Ernst et al., 2000). Hooper et al. (2002) suggest that hip and ankle
extensors compensate for the decrease in knee extensor moments. However, no
 electromyography data are available to support this observation.

Neuromuscular control is likely to influence knee extensor moments. Although reduced knee
extensor moments were observed during weight acceptance when walking, vastus lateralis
activity was not different between the involved and uninvolved side (Lewek et al., 2002). The
generalization of this finding is hindered because comparisons were made between the
involved and uninvolved legs of those with ACL injury rather than between injured and non-
injured groups. However, Knoll et al. (2004a) observed that EMG patterns of individuals 8
months after ACL surgery were similar to activation patterns of a control group.

It is apparent that various studies have focused on documenting the time it takes to recover
(return to typical gait) following ACL rupture. It is also clear that quadriceps weakness is
ubiquitous following ACL reconstruction and that quadriceps weakness is associated with
gait alterations including reduced knee flexion angles and internal knee extensor moments. These gait adaptations may lead to degenerative changes by altering the loading patterns of the knee joint, which is a potential mechanism for the development of knee OA. In addition, changes in knee kinetics have been observed early in knee OA (Astephen et al., 2008).

1.5.2. Frontal Plane Mechanics at the Knee

Loading in the frontal plane is of significant interest considering the association of external knee varus moments and knee osteoarthritis. Miyazaki et al. (2002) found knee varus moments at the knee during gait strongly predicted the progression of the medial knee OA. The risk of progression of knee OA increased by a factor of six with a 1% increase in knee varus moments.

Soft tissue structures of the knee joint provide most of the frontal plane stability as the quadriceps musculature predominantly acts in the sagittal plane. Nonetheless, the quadriceps aid in the control of varus and valgus moments at the knee joint (Palmieri-Smith and Thomas, 2009). Additionally, co-contraction of quadriceps and hamstring muscles provides stability for counteracting the external knee varus moment during walking (Shelbourne and Klotz, 2006). In particular, the hamstrings are antagonists to the ACL and these muscles aid to limit anterior-posterior and rotational displacement (Ageberg et al., 2009). Therefore, rehabilitation of hamstring and quadriceps muscles after ACL surgery is vital.

Alterations in hamstring/quadriceps (H/Q) strength ratio affect knee stabilization. Imbalances have been observed in ACL reconstructed individuals at least one year following surgery (Hiemstra et al., 2004). In particular, the H/Q ratio favors the knee flexors near full knee
extension (as in heel strike), perhaps signifying the role of the hamstring muscles in knee stabilization when the ACL is under greatest strain.

Injury to the knee increases the laxity of passive tissues within the knee joint, and greater quadriceps and hamstring co-contraction is needed to stabilize the joint. However, increasing co-contraction increases the compression forces on the articular cartilage, perhaps initiating articular degeneration (Schipplein and Andriacchi, 1991). Higher quadriceps:hamstring co-contraction has been observed in individuals with knee OA as compared to a control group (Zeni et al., 2010). Quadriceps:hamstring co-contraction has not been reported in those with ACL reconstruction surgery greater than one year. Medial quadriceps:hamstring co-activation patterns have been found to significantly increase with increasing severity of knee OA (Hubley-Kozey et al., 2009).

Zhang and Wang (2001) articulate that medial quadriceps and hamstring activity provide resistance to external knee valgus loads and that lateral quadriceps and hamstring activity is necessary to resist varus loads on the knee. In healthy recreationally active females medial-to-lateral quadriceps:hamstring co-contraction was found to be unbalanced, limiting the ability of the knee to resist valgus loads (Palmieri-Smith et al., 2009). Medial-to-lateral thigh muscle activation patterns have not been established in individuals post-ACL reconstruction and may provide insight to how frontal loading is resisted in this population group.

Butler et al. (2009) observed 21% greater knee varus moments during gait for individuals approximately 5 years after ACL reconstruction compared to a control group. However, Hooper et al. (2002) observed no differences in knee varus moments while walking or using the stairs when comparing the injured and non-injured leg 12 months post-ACL reconstruction. Increased hip extensor moments were found, compensating for reduced
knee extensor moments (Hooper et al., 2002). As noted earlier, rupturing the ACL can affect the neuromuscular ability of the both the injured and non-injured limb, which perhaps hinders an analysis of knee varus moment differences within an individual. Additionally, much more time elapsed in the previous study compared to the latter.

1.5.2. Frontal Plane Mechanics at the Hip

The knee joint does not function in isolation. It is likely that hip and ankle/foot mechanics may influence knee joint loading during weight bearing activities (Chang et al., 2005). Recalling the findings of Hooper et al. (2002) and Kowalk et al. (1997), it has been observed that the hip and ankle compensate for knee impairments in the sagittal plane. Additionally, few of the knee muscles control frontal plane stability; therefore, it has been suggested that hip musculature may aid in providing frontal plane stability. Hip abductor moments have been established as a protective mechanism against medial tibiofemoral knee OA progression assessed over an 18 month period (Chang et al., 2005).

The gluteus medius is the primary hip abductor. During gait, frontal plane control of the pelvis relies heavily on the internal hip abduction moment produced by the gluteus medius to stabilize and position the pelvis over the femur (Bennell et al., 2007). The hip abductors produce a force at least two times a person’s body weight to prevent contra-lateral pelvic drop in the frontal plane and aid in propelling the pelvis forward in the transverse plane (Wilson, 2005). Impaired hip abduction moment generation increases contra-lateral pelvic drop during the swing phase of gait. Pelvic drop alters the position of the center of mass away from the knee joint center, increases the external knee varus moment, and increases compression in the medial knee joint (Chang et al., 2005; Bennell et al., 2007). Unlike the established association between the internal knee extensor moment and quadriceps strength, Rutherford and Hubley-Kozey (2009) found no association between hip abductor
strength and internal hip abduction moment magnitude during gait in healthy subjects. However, in a cohort of subjects with generalized anterior knee pain, the gluteus medius activation had delayed onset and shorter duration during stair descent, indicating a compensatory mechanism due to knee pain (Brindle et al., 2003).

Internal hip abduction moments are becoming increasingly associated with medial knee joint loading and knee OA progression (Bennell et al., 2007). To date, only one study focused on frontal mechanics at the hip. Butler et al. (2009) found no alterations in hip mechanics in subjects 5 years post-ACL reconstruction surgery. More studies are needed to verify this finding and should include electromyography to aid in identifying a compensatory mechanism that might explain increased external knee varus moments.

To summarize, systematic reviews suggest that approximately half of those sustaining ACL rupture with meniscal injuries will develop knee OA. Knee OA is a debilitative disease, degrading the joint and inflicting the individual with considerable pain and impaired daily function. By the modest age of 35-40 years, knee OA is a reality for many of those who ruptured an ACL during early adulthood. Gait analysis can be used to identify alterations in factors associated with knee OA including reduced knee extensor moments, reduced hip abductor moments, and increased external knee varus moments. EMG measures can provide insight to neuromuscular control. Few studies have focused on individuals greater than one year from ACL reconstruction surgery. Research must aim to reduce the risk of knee OA development and improve current treatment and management practices to slow the rate of knee OA progression.
References


CHAPTER 3: GAIT ANALYSIS POST ANTERIOR CRUCIATE LIGAMENT RECONSTRUCTION: KNEE OSTEOARTHRITIS PERSPECTIVE

A paper to be submitted to Gait and Posture.
Michelle Hall, Catherine A. Stevermer, and Jason C. Gillette.

ABSTRACT

Individuals with anterior cruciate ligament (ACL) reconstruction are at increased risk to develop knee osteoarthritis (OA). Gait analysis describing kinetics of the lower extremity during walking and stair use (stair ascent and stair descent) can provide insight to everyday knee joint dynamic loading. In this study we compared lower extremity gait patterns of those with ACL reconstruction (>1 yr) to a control group. We hypothesized that the ACL group would have: 1) reduced knee extensor strength, 2) reduced knee flexion angles, 3) reduced knee extensor moments, 4) increased hip extensor moments, and 5) increased external knee varus moments when compared to the control group. Seventeen ACL reconstructed individuals and 17 healthy controls participated in this study. Knee extensor strength and knee flexor strength were recorded using a hand-held dynamometer. Kinematic and kinetic recordings were collected using an 8-camera motion analysis system, and portable force platforms were positioned on the first and second stair steps. Using inverse dynamics, internal hip abduction moments, hip extension moments, knee extension moments, ankle plantar flexion moments, and external knee varus moments were calculated during the stance phase of walking and during two steps of stair ascent and descent. Moments were normalized to body mass, and peak values were averaged across trials. One-way ANOVAs were used to test for main effects of group (ACL and control). Significance was set at p<0.05. During walking and stair use (on the second step), those with ACL reconstruction exhibited reduced knee extensor moments and increased hip extensor moments compared
to the control group. No differences were found in knee extensor strength or flexor strength between the two groups. Walking and stair ambulation highlighted altered joint loading in those with ACL reconstruction surgery. Individuals appeared to compensate for lower knee extensor moments by increasing hip extensor moments. Reduced knee extensor moments may protect the repaired ACL from excessive strain and/or may result from impaired neuromuscular control.

INTRODUCTION

Anterior cruciate ligament (ACL) injuries are among the most common musculoskeletal injuries, occurring more frequently in young adults, especially women. Individuals younger than 30 years sustain ACL ruptures more often compared to the general population (Lohmander et al., 2007; Englund, 2010). ACL ruptures are often concurrent with meniscal injuries (Lohmander et al., 2007) and where combined injury has occurred, knee osteoarthritis (OA) has been shown in up to 48% of cases (Louboutin et al., 2009; Lohmander et al., 2007). Knee OA is estimated to develop 5-20 years after initial ACL injury. Therefore, individuals between 35-40 years old are at increased risk of knee OA following ACL rupture (Englund, 2010).

Structural changes associated with ACL injury combined with long-term changes in dynamic loading contribute to the development of knee OA (Lohmander et al., 2007). As a measuring tool, gait analysis is considered a practical and reliable technique to indirectly assess dynamic loads on the lower extremity (Shakoor and Moisio, 2004). Investigating lower extremity joint moments during walking and stair negotiation can provide insight into everyday repetitive loads placed on the knee joint.
Lower extremity kinetic accommodations at the knee (DeVita et al., 1997; Timoney et al., 1993; Hooper et al., 2002) and hip (Ferber et al., 2004; Hooper et al., 2002) have been reported following ACL reconstruction. Individuals 6-12 months post-ACL reconstruction have altered walking patterns as compared to controls, including reduced knee flexion angles at midstance (Timoney et al., 1993; Hooper et al., 2002) and reduced internal knee extensor moments during early stance (DeVita et al., 1997; Timoney et al., 1993). These gait adaptations may lead to degenerative changes in the articular cartilage by altering the loading patterns on the knee joint. Decreased knee flexion angles and internal knee extensor moments have been associated with quadriceps weakness in individuals up to 1 year post-ACL surgery (Lewek et al., 2002; Bush-Joseph et al., 2001). Large cross-sectional studies have established strong relationships between quadriceps weakness and the onset of knee OA (Becker et al., 2004; Slemenda et al., 1997). Persistent quadriceps weakness developed through the arthrogenic inhibition mechanism leads to altered kinetics and kinematics after knee injury (Hart et al., 2010). Despite ACL reconstruction and aggressive rehabilitation regimes, weakness in quadriceps musculature exists up to one year post-ACL reconstruction surgery (Suter et al., 2001). Theoretically, weak quadriceps reduces the ability to generate force necessary to provide efficient eccentric control of joint loading during the loading phase of gait (Hart et al., 2010). It has yet to be determined if quadriceps weakness along with reduced knee extensor moments and knee flexion angles persist long-term (greater than one year after ACL reconstruction).

Arguably, external knee varus moment has the greatest clinical consequence when considering potential knee OA risk factors. Knee varus moments are associated with medial compression, the compartment most often affected with OA. Miyazaki et al. (2002) established that the risk of knee OA progression increased by a factor of 6 with a 1% increase in external knee varus moment. More recently, attention has focused on the role of
the hip abductors in protecting against knee OA. Impaired hip abduction moment generation causes contralateral pelvis drop, increasing knee varus moments and compression in the medial knee (Chang et al., 2005; Bennell et al., 2007). Hooper et al. (2002) observed no differences in knee varus moments while walking or using the stairs when comparing the injured and non-injured leg one year post-ACL reconstruction. However, Butler et al. (2009) observed 21% greater knee varus moments during walking in individuals five years following ACL reconstruction when compared to a control group, although no changes in frontal hip mechanics were observed. Evidence suggests that increased knee varus moments may not be present within the first year of ACL rupture, but medial knee loading may increase as time progresses. Further study is required to assess whether dynamic knee joint loading is greater for individuals post-ACL surgery during walking and stair ambulation.

The purpose of this study was to compare gait of those with ACL reconstruction to a comparable healthy group during three tasks: stair ascent, stair descent and level walking. The primary focus was to determine if factors associated with the development and/or progression of knee OA were observed in ACL reconstructed individuals when compared to healthy counterparts. We hypothesized that the ACL group would display: 1) reduced knee extensor strength, 2) reduced knee flexion angles, 3) reduced knee extension moments, 4) increased hip extension moments, and 5) increased external knee varus moments when compared to the control group.

**METHODS**

Seventeen subjects who had previous ACL reconstruction surgery (Table 2) and 17 healthy controls participated. Individuals were excluded if they had any history of musculoskeletal or neurological conditions that would preclude safe walking and stair negotiation. This study
was approved by the Institutional Review Board at Iowa State University, and all subjects gave their written consent to participate.

The walkway used in this experiment was 6 m in length, and the experimental staircase consisted of three steps (step height 18.5 cm, tread depth 29.5 cm). Kinematic data were collected using an 8-camera, three-dimensional motion analysis system (Vicon Nexus, Los Angeles, CA, USA). Kinetic data were recorded using two portable force platforms positioned on the second and third step of the stairs and one in-ground force platform (AMTI, Watertown, MA, USA). Kinematic data were collected at a sampling rate of 160Hz, while force platform data were collected at a rate of 1600Hz.

After subjects signing the informed consent document, their age, height, weight, medical history and physical activity levels (Tegner scale) were recorded. Knee extensor and knee flexor strength data were acquired using a hand-held dynamometer (Lafayette Instrument Company, Lafayette, Indiana, USA). While seated upright with the knee flexed approximately 90°, participants performed three maximal knee flexion and knee extension contractions against manual resistance. High inter-rater reliability and good intra-rater reliability was achieved (ICC = 0.89-0.97, ICC =0.76, respectively). The peak dynamometer force measured over the three trials was used for analysis.

Retro-reflective markers (1.9 cm in diameter) were then placed bilaterally on the medial and lateral malleoli, heels, fifth metatarsal heads, dorsal foot, anterior shank, medial and lateral femoral condyles, anterior thighs, greater trochanters, anterior superior iliac spines, posterior superior iliac spines and acromion processes. Individual markers were placed on the sacrum and cervicale. Following a static trial, markers including heels, medial malleoli, medial knee joints, and posterior superior iliac spines were removed. Removed markers were recreated during dynamic trials using transforms derived from the static trial. Three tasks of interest in
this study were stair ascent, stair descent, and level walking. Participants performed three trials leading with each leg for a total of six trials for each task. Individuals descended and ascended the stairs using a step-over-step technique. They wore their preferred shoes and performed all tasks at a self chosen pace.

All data analyzed were from the stance phase of walking and the first and second step of both stair ascent and descent. For stair negotiation, stance phase was defined as the period from when vertical ground reaction force reached 20% of body weight (BW) to the point when vertical ground reaction force dropped below 20% BW. Stance phase for walking was initiated at 5% BW and terminated when vertical ground reaction force dropped below 5% BW. Stance time was calculated for each of the three tasks: stair ascent, stair descent and walking. Gait analysis was performed on both legs of all participants. Noise was reduced in kinematic and kinetic data using a fourth order, symmetric Butterworth filter with a cut-off frequency of 6Hz. Maximum knee angles were calculated using the following order of rotations: flexion/extension, abduction/adduction, and internal/external rotation. Using inverse dynamics, maximum internal ankle plantarflexion, knee extension, hip extension, hip abduction, and external knee varus moments were calculated during each stance phase, transformed to the distal segment coordinate system, and normalized by body mass. Peak moments were averaged across the three trials of each movement condition. Kinetic and kinematic data were processed using custom code written in Matlab™ version 9.0 (The Mathworks Inc., Natick, MA, USA).

Between group (post-ACL versus control) differences for age, height, body mass, physical activity levels, and stance time were investigated using t-tests. One-way ANOVAs were used to test for main effects of group. Statistical analyses were performed using SPSS for Windows (Version 12.0; SPSS Chicago, IL, USA). A statistical significance level was set at $p$
≤ 0.05. An average of right and left leg was determined for the control group and used to compare with the injured leg of the ACL group.

**RESULTS**

There were no differences between controls and the ACL group for age, height, and body mass (Table 1). There were no differences in Tegner scores between the groups, verifying that all ACL individuals had returned to typical physical activity levels. No differences were found between the ACL group and control group for stance time during stair ascent, stair descent or walking (Table 1). This variable was excluded as a possible confounder in our subsequent results.

Knee extensor strength did not differ between the ACL and the control group (382 ± 167N and 402 ± 137N, \( p = 0.702 \)). Also, no differences were found for knee flexor strength between the ACL and control group, (248 ± 53N and 271 ± 44N, \( p = 0.184 \)).

Initial stance knee flexion angle during stair ascent did not differ between the ACL group and the control group on step one (60.6 ± 6.4° and 60.4 ± 3.6°, \( p = 0.939 \)) or step two (64.4 ± 5.6° and 63.2 ± 3.7°, \( p = 0.485 \)). Similarly, no significant differences between the ACL and control group were found in initial stance knee flexion angle differences during step one of stair descent (11.7 ± 3.8° and 13.11 ± 3.2°, \( p = 0.246 \)) or step two of stair descent (11.9 ± 4.0° and 12.9 ± 3.4°, \( p = 0.419 \)). During walking, initial stance knee flexion angles were similar when comparing ACL individuals to the control group (41.2 ± 3.4° and 41.2 ± 5.7°, \( p = 0.663 \)).

Joint moments at the hip, knee, and ankle did not differ between the groups during the first step of stair ascent or descent (Table 3). However, hip extensor moments differed between the ACL and control group during stair ascent and descent on the second step and during walking (Figure 1). Peak hip extensor moments for the ACL group were 31% greater during
stair ascent ($p = 0.007$), 90% greater stair during descent ($p = 0.031$), and 43% greater during level walking compared to controls ($p = 0.002$). In contrast, the ACL group displayed reduced knee extensor moments on the second step of stair ascent and descent; 23% less during stair ascent ($p = 0.014$) and 22% less during stair descent ($p = 0.002$). Hip abduction moments were greater in the ACL group during stair ascent on the first step when compared to controls, with the difference approaching significance ($p = 0.077$). Ankle plantar flexion moments and external knee varus moments did not differ between the ACL and control group during any of the three tasks.

**DISCUSSION**

This study aimed to determine if established biomechanical parameters associated with the development and/or progression of knee OA existed in a young population over a year post-ACL reconstruction. Knee injuries, including ACL ruptures, increase the risk for early-onset knee OA by a 10-fold compared to the general, uninjured population (Gillquist et al., 1999). Focusing research on this should provide insight in the early onset of knee OA pathogenesis.

As hypothesized, reduced knee extensor moments were found in the ACL group, specifically during stair use. Decreased knee extensor moments, also known as quadriceps avoidance, have been observed in ACL reconstructed individuals up to one year post surgery during stair ascent (Kowalk et al., 1997; Hooper et al., 2002), stair descent, and walking (Lewek et al., 2002). Knee extensor moments are indicative of quadriceps and hamstring neuromuscular function (Hart et al., 2010) and have been attributed to reduced quadriceps strength (Lewek et al., 2002; Bush-Joseph et al., 2001) and reduced knee flexion angle in those with ACL injury (Lewek et al., 2002).
Table 1: Descriptive statistics comparing ACL reconstruction individuals and healthy controls

<table>
<thead>
<tr>
<th>Variable</th>
<th>ACL (n=17)</th>
<th>Control (n=17)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (M/F)</td>
<td>8M/9F</td>
<td>7M/10F</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>26 ± 6</td>
<td>26 ± 4</td>
<td>0.145</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>174 ± 14</td>
<td>170 ± 12</td>
<td>0.360</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
<td>75 ± 16</td>
<td>68 ± 12</td>
<td>0.135</td>
</tr>
<tr>
<td>Tegner Activity Level</td>
<td>7 ± 2</td>
<td>6 ± 1</td>
<td>0.112</td>
</tr>
<tr>
<td>Stance time (s)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stair Ascent</td>
<td>1.43 ± 0.11</td>
<td>1.40 ± 0.12</td>
<td>0.485</td>
</tr>
<tr>
<td>Stair Descent</td>
<td>1.22 ± 0.13</td>
<td>1.28 ± 0.11</td>
<td>0.172</td>
</tr>
<tr>
<td>Walking</td>
<td>0.72 ± 0.05</td>
<td>0.71 ± 0.05</td>
<td>0.795</td>
</tr>
</tbody>
</table>

Table 2: ACL group characteristics

<table>
<thead>
<tr>
<th>Injury Characteristics</th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Reconstruction Type (n = 17)</strong></td>
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</tr>
<tr>
<td>Hamstring</td>
<td></td>
<td>41 %</td>
</tr>
<tr>
<td>Patellar Tendon</td>
<td></td>
<td>41 %</td>
</tr>
<tr>
<td>Cadaver</td>
<td></td>
<td>6 %</td>
</tr>
<tr>
<td>Cadaver &amp; Hamstring</td>
<td></td>
<td>12 %</td>
</tr>
<tr>
<td><strong>Nature of Rupture (n = 17)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contact</td>
<td></td>
<td>41 %</td>
</tr>
<tr>
<td>Non-Contact</td>
<td></td>
<td>59 %</td>
</tr>
<tr>
<td><strong>Severity of Injury</strong></td>
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<td></td>
</tr>
<tr>
<td>Isolated ACL rupture (n = 5)</td>
<td></td>
<td>29 %</td>
</tr>
<tr>
<td>ACL rupture with meniscus (n = 12)</td>
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<td>71 %</td>
</tr>
<tr>
<td><strong>Time from Injury</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average (yrs)</td>
<td></td>
<td>5.6 years</td>
</tr>
<tr>
<td>Range</td>
<td></td>
<td>1-18 years</td>
</tr>
</tbody>
</table>
Table 3: Lower extremity joint moments during stair ascent, stair descent, and walking. Significant differences (p < 0.05) highlighted in bold. Results are in mean ± SD.

| Peak Moments (Nm · kg⁻¹) | Stair Ascent | | | Stair Descent | | | | Walking | | |
|-------------------------|-------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|------------------|-------------------|------------------|------------------|
|                         | ACL n = 17  | Control n = 17    | *p*-value         | ACL n = 17        | Control n = 17    | *p*-value         | ACL n = 15        | Control n = 16   | *p*-value         |
| Hip Extension           |             |                   |                   |                   |                   |                   |                   |                   |                   |
| Step 1                  | 0.90 ± 0.26 | 0.72 ± 0.26       | 0.061             | 0.28 ± 0.40       | 0.17 ± 0.24       | 0.327             | 1.07 ± 0.25       | 0.75 ± 0.28       | **0.002**         |
| Step 2                  | 0.98 ± 0.26 | 0.75 ± 0.20       | **0.007**         | 0.40 ± 0.24       | 0.21 ± 0.24       | **0.031**         |                   |                   |                   |
| Hip Abduction           |             |                   |                   |                   |                   |                   |                   |                   |                   |
| Step 1                  | 0.92 ± 0.50 | 0.69 ± 0.15       | 0.077             | 1.01 ± 0.55       | 0.94 ± 0.40       | 0.653             | 1.00 ± 0.16       | 0.94 ± 0.12       | 0.202             |
| Step 2                  | 0.62 ± 0.23 | 0.61 ± 0.23       | 0.926             | 1.13 ± 0.31       | 1.06 ± 0.18       | 0.412             |                   |                   |                   |
| Knee Extension          |             |                   |                   |                   |                   |                   |                   |                   |                   |
| Step 1                  | 1.02 ± 0.33 | 1.20 ± 0.29       | 0.101             | 1.14 ± 0.48       | 1.17 ± 0.30       | 0.799             | 0.48 ± 0.28       | 0.44 ± 0.12       | 0.576             |
| Step 2                  | 0.81 ± 0.25 | 1.05 ± 0.29       | **0.014**         | 0.86 ± 0.21       | 1.11 ± 0.23       | **0.002**         |                   |                   |                   |
| Knee Varus              |             |                   |                   |                   |                   |                   |                   |                   |                   |
| Step 1                  | 0.68 ± 0.60 | 0.52 ± 0.10       | 0.261             | 0.27 ± 0.38       | 0.17 ± 0.10       | 0.274             | 0.57 ± 0.30       | 0.45 ± 0.08       | 0.164             |
| Step 2                  | 0.45 ± 0.27 | 0.47 ± 0.23       | 0.830             | 0.33 ± 0.24       | 0.31 ± 0.12       | 0.728             |                   |                   |                   |
| Ankle Plantar Flexion   |             |                   |                   |                   |                   |                   |                   |                   |                   |
| Step 1                  | 1.44 ± 0.20 | 1.42 ± 0.14       | 0.650             | 1.21 ± 0.28       | 1.21 ± 0.16       | 0.962             | 1.53 ± 0.54       | 1.42 ± 0.6        | 0.420             |
| Step 2                  | 1.44 ± 0.25 | 1.46 ± 0.10       | 0.793             | 1.29 ± 0.22       | 1.26 ± 0.16       | 0.671             |                   |                   |                   |
Figure 1. Peak hip extensor moments during the stance phase. Step two of stair ascent and stair descent is included with walking. * indicates significant differences (p < 0.05).

Figure 2. Peak knee extensor moment during stance phase. Step two of stair ascent and stair descent is included with walking. * indicates significant differences (p < 0.05).
Quadriceps weakness following ACL reconstruction is believed to contribute to likelihood of early-onset knee OA (Roos, 2005; Palmieri-Smith and Thomas, 2009). Interestingly, no difference in knee extensor strength between the ACL and control group was found. Despite not exhibiting weakened knee extensors or reduced peak knee flexion angles, it is possible that impaired quadriceps muscle activity contributed to reduced knee extensor moments. Impaired neural activity of the quadriceps, likely caused by arthrogenic muscle inhibition (Palmieri-Smith and Thomas, 2009), has been observed up to two years following ACL surgery (Urbach et al., 2001). Arthrogenic muscle inhibition is considered a natural response to protect the joint from further damage (Hopkins et al., 2001). Alternatively, a reduced knee extensor moment in the ACL group may be the result of increased hamstring activity. Internal knee extensor moments are a net joint moment calculation (Winter, 1990) that does not account for quadriceps and hamstring contributions separately. Electromyography data of the thigh musculature would provide insight into the mechanism of reduced knee extensor strength in the ACL group, affirming if quadriceps activity was reduced or indeed if hamstring activity was increased. Regardless of the reduced knee extensor moment mechanism, loading on the knee joint is altered in the ACL group during stair use as compared to the control group, which may initiate joint surface damage. Due concern must be given to neuromuscular impairments such as weakness and inhibition when aiming to prevent post-traumatic knee OA.

As a compensation for reduced knee extensor moments, ACL reconstructed participants were found to produce greater hip extensor moments during stair use. During walking, the least challenging of the tasks analyzed in this study, those with ACL reconstruction demonstrated increased hip extensor moments despite not having reduced knee extensor moments. Increased hip extensor moments during the stance phase of walking have also been found in individuals 6-month post-ACL reconstruction (DeVita et al., 1998). In the
present study, particularly during the second step of stair descent, the ACL group generated a hip extensor moment 90% greater than the control group. Studies have found increased hip extensor moments combined with decreased knee extensor moments during closed-chain exercises in individuals within and following one year post-ACL reconstruction (Osternig et al., 2000; Salem et al., 2003). The ratio of hip:knee extensor moments is significantly related to the magnitude of anterior tibia shear in those at least one year from ACL reconstruction surgery (Osternig et al., 2000). To avoid placing strain on the repaired ACL, individuals increase hip extensor moments and decrease knee extensor moments. Interestingly, this phenomenon of increasing hip extensor moments to compensate for reduced knee extensor moments has not been observed with other knee pathologies (e.g., patellofemoral pain), where individuals also exhibit reduced knee extensor moments during stair use. A change in the ratio of hip:knee extensor moments will likely affect the area of loading on the articular cartilage, which may relate to the initiation of knee OA.

The ACL group did not adapt hip or knee frontal plane mechanics during any of the three tasks as compared to the control group. Knee varus moments are closely associated with knee osteoarthritis and have been reported to be up to 21% greater during walking in a similar study sample (Butler et al., 2009). In the current study the average time from surgery was 5 years, similar to that of Butler et al. (2009), suggesting that additional factors may have influenced knee joint kinetics. For example, type of ACL reconstruction surgery, rehabilitation program, time from injury to surgery, and physical activity level may influence knee OA risk factors. Reconstructions using the bone-patellar tendon-bone technique have resulted in lower magnitude moments when compared to hamstring autograft type reconstructions (Webster et al., 2005). However, a systematic review suggests that there is no clear bias between these two types of reconstruction techniques regarding kinetic measures (Hart et al., 2010).
In conclusion, our findings suggest that the knee joint experiences altered dynamic loading during everyday activities, including walking and stair use. Altered joint loading occurred primarily in the sagittal plane, specifically increased hip extension moments and reduced knee extension moments. We speculate that altered hip and knee joint loading during repetitive everyday movements may contribute to the development of knee OA.
REFERENCES


ABSTRACT

Individuals with anterior cruciate ligament (ACL) reconstruction are at increased risk to develop knee osteoarthritis (OA). Electromyography of walking and stair use can provide insight to everyday knee joint dynamic loading. Those with ACL reconstruction have altered neuromuscular patterns during dynamic activities. The purpose of this study was to compare lower extremity neuromuscular control strategies of those with ACL reconstruction (>1yr) to a control group. Eighteen ACL reconstructed individuals (26 ± 6 years, 6 ± 4 years from surgery) and 18 healthy controls (26 ± 4 years) participated in this study. Participants performed three ascending and descending trials on a three step staircase and three walking trials leading with the right and left leg, respectively. Ground reaction forces were collected using two portable force platforms that were positioned on the first and second stair steps and an in-ground force platform. Electromyography (EMG) data were collected from the vastus lateralis [VL], vastus medialis [VM], biceps femoris [BF], semimembranosus [SM], and gluteus maximus [GMax]. A linear envelope (10 Hz low-pass filter) was used to determine maximum EMG values during the stance phase of each step. Maximum EMG values were averaged across three trials and normalized to maximum voluntary isometric contractions MVIC. Co-contraction indices were determined for knee extensors/flexors (VL+VM,BF+SM), for medial thigh muscles (VM,SM), and for lateral thigh muscles (VL,BF). One-way ANOVAs were used to test for main effects of group (post-ACL vs. control) on EMG activity and co-contraction indices. Significance was set at p<0.05. Hip extensor
(GMax+BF+SM) activity was greater in the post-ACL group during stair ascent, stair descent, and walking. Knee flexor (BF+SM) activity was greater during the first step of stair ascent and during walking in the post-ACL group. No differences were found for knee extensor (VL+VM) activity between the post-ACL and control groups. Increased medial co-contraction was found in the post-ACL group during walking, and increased knee extensor/flexor co-contraction was found during the first step of stair ascent.

Those with ACL reconstruction displayed long-term changes in neuromuscular control during walking and stair ambulation. The adaptations observed may act to control anterior tibial translation, prevent excessive strain on the ACL, and/or increase overall knee joint stability.

**INTRODUCTION**

Anterior cruciate ligament rupture (ACL) is among the most frequent musculoskeletal injuries sustained by a predominantly young population. Those with ACL injury are considered at higher risk to develop knee osteoarthritis (OA), as almost 50% of those with ACL injury develop the degenerative condition (Lohmander et al., 2007). Neuromuscular impairments of the knee joint and associated musculature have been found in persons with ACL reconstruction (Hart et al., 2010a; Hart et al., 2010b; Drechsler et al., 2006). Impairments include reduced knee extensor strength, increased knee extensor inhibition, and increased co-contraction between quadriceps and hamstring muscles. These alterations in neuromuscular strategies are also associated with knee OA, with changes in muscle force distribution ultimately affecting the mechanical environment of the knee (Hubley-Kozey et al. 2009). Using electromyography (EMG), the presence of altered neuromuscular control can be identified in those with ACL reconstruction during walking and stair use.
Research on persons with ACL reconstruction has indicated kinetic and kinematic alterations during walking (Timoney et al., 1993; DeVita et al., 1997) and stair use (Kowalk et al., 1997; Hooper et al., 2002). The predominant alterations include reduced internal knee extensor moments (Lewek et al. 2002; Bush-Joseph et al., 2001), increased hip extensor moments (Hooper et al. 2002; Ernst et al., 2000; Nyland et al., 2010), and reduced knee flexion angles (Timoney et al., 1993; Lewek et al., 2002) during dynamic activity.

Understanding of the mechanisms resulting in reduced knee extensor moments is limited when only using kinetic analysis. Reduced quadriceps activity and/or strength are suspected to be contributing factors to reduced knee extensor moments (Webster et al., 2005; Ernst et al., 2000). Therefore, analyzing EMG magnitudes enable us to determine if those with ACL reconstruction have reduced quadriceps activity or have increased hamstring activity as compared to a control group.

Increased anterior tibial translation has been identified as the principal cause for developing knee OA following ACL rupture (Louboutin et al., 2009). Injury to the knee increases the laxity of passive tissues within the knee joint and greater quadriceps and hamstring co-contraction is needed to stabilize the joint. Therefore, quadriceps and hamstring co-contraction aids in stabilizing the knee joint during walking by controlling anterior tibial translations and counteracting knee varus moments (Shelbourne and Klotz, 2006).

Increased quadriceps and hamstring co-contraction results in greater knee joint contact forces (Lu et al., 1997), which translates into increased compression forces on the knee joint surface, perhaps initiating articular degeneration (Schipplein and Andriacchi, 1991). Consistent with this view, higher quadriceps and hamstring co-contraction has been observed in individuals with knee OA as compared to a control group (Zeni et al., 2010).

Quadriceps and hamstring co-contraction has not been reported during stair use in individuals more than one year post-ACL reconstruction surgery.
Increased knee varus moments have been found during walking in those with ACL reconstruction as compared to healthy control individuals (Butler et al., 2009). Knee varus moments are associated with medial knee joint compression, which is often implicated in the progression of knee OA (Miyazaki et al., 2002). Soft tissue structures of the knee joint provide most of the frontal plane stability as the quadriceps and hamstring musculature acts predominantly in the sagittal plane. Nonetheless, the quadriceps aid in the control of valgus and varus moments at the knee joint (Palmieri-Smith and Thomas, 2009). Zhang and Wang (2001) explain that medial quadriceps and hamstring activity provide resistance to knee valgus loads and that lateral quadriceps and hamstring activity is necessary to resist varus loads on the knee. In healthy recreationally active females, medial and lateral quadriceps/hamstring co-contractions were found to be unbalanced, limiting the ability to resist knee valgus loads (Palmieri-Smith et al., 2009). Medial and lateral quadriceps/hamstring activation patterns have not been established in individuals post-ACL reconstruction and may provide insight to how frontal loading is resisted.

Identifying neuromuscular compensatory strategies in the years following ACL reconstruction and understanding their potential effects on long-term joint health will lead to an improved understanding of likely mechanisms contributing to altered gait patterns and knee joint degeneration. Therefore, the purpose of this study was to determine whether neuromuscular responses differ during dynamic loading (stair use, walking) in those with ACL reconstruction compared to a healthy population. We hypothesized that the ACL group would have: i) reduced knee extensor activity, ii) greater hamstring muscle activity, iii) increased hip extensor activity, iv) increased co-contraction between knee extensors and knee flexors, and v) altered medial and lateral quadriceps/hamstring co-contraction as compared to a healthy control group.
METHODS

Two groups were recruited for this study: a healthy control group and those with ACL reconstruction. Both control and ACL participants were recruited from the general community. Control subjects had no history of musculoskeletal or neurological conditions. Those in the ACL group had previous ACL reconstruction more than one year previously and presented no current conditions that precluded safe walking and stair use. This study was approved by the Institutional Review Board at Iowa State University, and all subjects gave their written consent to participate.

The walkway used in this experiment was 6 m in length, and the experimental staircase consisted of three steps (step height 18.5 cm, tread depth 29.5 cm). Muscle activity signals were collected from a wireless EMG system (Delsys, Boston, MA, USA). Force platform and EMG data were collected at a rate of 1600Hz. Two portable force platforms on the second and third step of the stairs and one in-ground force platform (AMTI, Watertown, MA, USA) were used to determine the stance phases of walking and stair ambulation.

Subject age, height, weight, medical history, and physical activity levels (Tegner scale) were recorded. The participant's skin was shaved (when needed), cleaned, and slightly abraded before surface electrodes were placed. The electrodes were placed bilaterally over the muscle belly in line with muscle fibers of the gluteus maximus [GMax], rectus femoris [RF], vastus lateralis [VL], vastus medialis [VM], biceps femoris [BF], and semimembranosus [SM]. The VL and VM were selected to represent knee extensors, with SM and BF chosen to represent knee flexors. These are the muscles that potentially lead to the greatest knee joint compression. Hip extensors were represented by the GMax, SM, and BF. The hamstring muscles were included based on their bi-articular functions as hip extensors. A
reference electrode was placed over the electrically neutral tissue of the right anterior superior iliac spine.

All subjects performed three trials of 5-second maximum voluntary isometric contractions (MVIC) in order to normalize EMG data from walking and stair use. For the GMax MVIC test, participants stood upright with arms supported on a table and extended the hip towards the researcher who resisted the movement by placing hands on the lower thigh. Knee extensor (VL and VM) and knee flexor (BF and SM) strength data were acquired during MVIC using a hand-held dynamometer (Lafayette Instrument Company, Lafayette, Indiana, USA). While seated upright with the knee flexed approximately 90°, participants performed three maximal knee flexion and knee extension contractions against manual resistance. All subjects were given verbal encouragement. The peak dynamometer force measured over the three trials was used for analysis.

Participants performed three tasks: stair ascent, stair descent and walking. Individuals descended and ascended the stairs using a step-over-step technique, and all tasks were performed at a self-selected pace. Participants performed three trials leading with right and left leg, for a total of six trials for each task.

All data were analyzed during the stance phase of walking and on the first and second step of both stair ascent and descent. For stair analyses, the stance phase was defined as the period from when the vertical ground reaction force reached 20% of body weight (BW) to the point where vertical ground reaction force dropped below 20% BW. For walking analyses, step detection was initiated at 5% of BW and terminated when the vertical ground reaction force dropped below 5% BW. It was necessary to increase the threshold of step detection for stair negotiation to avoid potential cross-talk between the portable force platforms.
Stance time was calculated for each of the stair negotiation and walking stance phases using the thresholds defined above.

Raw EMG data for the MVICs and the three tasks were bandpass filtered between 10-450Hz with a fourth order, dual-pass Butterworth filter, and bias was removed by subtracting the mean from each point. The data were then rectified and filtered using a low-pass filter at 10Hz to create a linear envelope. Maximum EMG amplitudes during the MVICs were defined as the maximum point within the linear envelope. The maximum EMG amplitude during the stance phase of stair ascent, stair descent, and walking were normalized to the peak MVIC values. The maximum EMG amplitudes were then combined by muscle function and divided by the number of muscles in each group:

\[ EMG_{knee\, extensors} = \frac{VL + VM}{2} \quad 1a. \]

\[ EMG_{knee\, flexors} = \frac{BF + SM}{2} \quad 1b. \]

\[ EMG_{hip\, extensors} = \frac{G\text{Max} + BF + SM}{3} \quad 1c. \]

Co-contraction indices (CCI) were calculated by adapting the equation described by Rudolph et al. (2001) for maximum EMG amplitudes and multiple muscle groups:

\[ CCI_{flexors:extensors} = \frac{\min (VL + VM, BF + SM)}{\max (VL + VM, BF + SM)} \times (VL + VM + BF + SM) \quad 2a. \]

\[ CCI_{mediat} = \frac{\min (VM, SM)}{\max (VM, SM)} \times (VM + SM) \quad 2b. \]

\[ CCI_{laterat} = \frac{\min (VL, BF)}{\max (VL, BF)} \times (VL + BF) \quad 2c. \]

In the equations, \( \min \) represents the maximum EMG amplitudes from the less active muscle group, and \( \max \) represents the EMG values of the more active muscle group. To investigate
medial and lateral neuromuscular strategies, we grouped VM and SM to represent medial thigh muscle activity and VL and BF to represent lateral thigh muscle activity. All data were processed using custom code written in Matlab™ version 9.0 (The Mathworks Inc., Natick, MA, USA).

Gait analysis was performed on both legs of all participants. For control participants, data from the right and left leg were combined and compared to the injured leg of the ACL group. Between group (post-ACL vs. control) differences for age, height, body mass, physical activity levels and movement speeds were investigated using t-tests. One-way ANOVAs were used to test for main effects of group (post-ACL vs. control). Statistical analyses were performed using SPSS for Windows (Version 19, SPSS Chicago, IL, USA). The statistical significance level was set at p<0.05.

RESULTS

Eighteen participants were recruited for the post-ACL group (9 males and 9 females, age 26 ± 6 years, height 1.7 ± 0.1m, mass 73.4 ± 15.8kg, Tegner score 6.5 ± 1.5), and 18 participants were recruited as healthy controls (8 males and 10 females, 26 ± 4 years, height 1.7 ± 0.1m, mass 68.5 ± 12.1kg, Tegner score 6.1 ± 0.9). There were no differences between controls and the post-ACL group for age, height, weight, and physical activity levels (p > 0.05). No differences were found between the post-ACL group and control group for stance time during stair ascent (1.41 ± 0.11s vs. 1.40 ± 0.12s, p > 0.05), stair descent (1.19 ± 0.12s vs. 1.28 ± 0.11s, p > 0.05), or walking (0.71 ± 0.04s vs. 0.71 ± 0.05s, p > 0.05). Knee extensor strength did not differ between the ACL and the control group (382 ± 167N and 402 ± 137N, p = 0.702) Also, no differences were found for knee flexor strength between the ACL and control group (248 ± 53N and 271 ± 44N, p = 0.184).
Depending on the task, participants were excluded from the analysis if EMG amplitude outliers occurred. These outliers were likely caused by excessive skin movement, disrupted wireless signals, or compromised skin contact. Analysis for stair ascent included 10 post-ACL participants and 14 controls, stair descent included 12 post-ACL participants and 16 controls, and walking included 13 post-ACL participants and 16 controls.

Maximum EMG amplitudes for hip extensors, knee extensors, and knee flexors are shown in Table 1. The post-ACL group demonstrated significantly increased hip extensor activity (p < 0.05) during both steps of stair ascent, both steps of stair descent, and during walking compared to the control group (Fig. 1). There were no significant differences in knee extensor activity (p > 0.05) between the two groups during any of the three tasks analyzed (Fig. 2). The post-ACL group demonstrated significantly increased knee flexor activity (p < 0.05) during the first step of stair ascent and during walking as compared to the control group (Fig 3.). No significant differences for knee flexor activity (p > 0.05) were found between the post-ACL and control groups during stair descent.

Knee extensor/flexor co-contraction indices are shown in Table 2. Knee extensor/flexor CCI was significantly increased on the first step of stair ascent for the post-ACL group as compared to the control group (p < 0.05). Knee extensor/flexor CCI did not significantly change between the post-ACL and control groups during stair descent or walking (p > 0.05).

Co-contraction indices for medial thigh muscle activity and lateral muscle thigh activity are shown in Table 2. Medial thigh CCI was significantly increased in the post-ACL group during walking (p < 0.05). In addition, increases in medial thigh CCI approached significance (p = 0.064) in the post-ACL group during the second step of stair descent when compared to controls. There were no significant differences for lateral thigh CCI between those with ACL reconstruction and the control group for any of the three tasks (p > 0.05).
Table 1: Maximum EMG amplitudes for hip extensors, knee extensors, and knee flexors. Significant differences (p < 0.05) highlighted in bold. Results are in mean ± SD.

| EMG Amplitude (MVIC) | Stair Ascent | | | Stair Descent | | | Walking | |
|-----------------------|--------------|-----------------|----------------|--------------|-----------------|---|---|---|---|---|
|                       | ACL n = 10   | Control n = 14  | p-value        | ACL n = 12   | Control n = 16  | p-value         | ACL n = 13 | Control n = 16 | p-value |
| Hip Extensors         |              |                 |                |              |                 |                |              |                 |        |
| Step 1                | 0.49 ± 0.17  | 0.36 ± 0.07     | **0.014**      | 0.26 ± 0.10  | 0.18 ± 0.07     | **0.033**      | 0.31 ± 0.12  | 0.21 ± 0.08     | **0.009** |
| Step 2                | 0.50 ± 0.14  | 0.38 ± 0.08     | **0.021**      | 0.25 ± 0.09  | 0.18 ± 0.07     | **0.034**      |              |                 |          |
| Knee Extensors        |              |                 |                |              |                 |                |              |                 |        |
| Step 1                | 0.90 ± 0.23  | 0.82 ± 0.20     | 0.375          | 0.43 ± 0.16  | 0.45 ± 0.15     | 0.652          | 0.31 ± 0.26  | 0.22 ± 0.11     | 0.213   |
| Step 2                | 0.93 ± 0.25  | 0.79 ± 0.18     | 0.130          | 0.36 ± 0.15  | 0.40 ± 0.14     | 0.498          |              |                 |          |
| Knee Flexors          |              |                 |                |              |                 |                |              |                 |        |
| Step 1                | 0.36 ± 0.11  | 0.27 ± 0.06     | **0.017**      | 0.19 ± 0.10  | 0.15 ± 0.07     | 0.208          | 0.27 ± 0.15  | 0.16 ± 0.08     | **0.021** |
| Step 2                | 0.36 ± 0.10  | 0.29 ± 0.07     | 0.084          | 0.19 ± 0.11  | 0.15 ± 0.04     | 0.136          |              |                 |          |
Table 2: Co-contraction indices for knee extensors/flexors, medial thigh muscles, and lateral thigh muscles. Significant differences ($p < 0.05$) highlighted in bold. Results are in mean ± SD.

<table>
<thead>
<tr>
<th>Co-contraction Indices</th>
<th>Stair Ascent</th>
<th>Stair Descent</th>
<th>Walking</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ACL</td>
<td>Control</td>
<td>$p$-value</td>
</tr>
<tr>
<td>Knee Extensors/Flexors (VL+VM,BF+SM)</td>
<td>Step 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.03 ± 0.35</td>
<td>0.74 ± 0.18</td>
<td><strong>0.014</strong></td>
</tr>
<tr>
<td></td>
<td>1.03 ± 0.38</td>
<td>0.81 ± 0.24</td>
<td>0.103</td>
</tr>
<tr>
<td>Medial Thigh Muscles (VM,SM)</td>
<td>Step 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.31 ± 0.11</td>
<td>0.24 ± 0.12</td>
<td>0.151</td>
</tr>
<tr>
<td></td>
<td>0.30 ± 0.13</td>
<td>0.25 ± 0.18</td>
<td>0.315</td>
</tr>
<tr>
<td>Lateral Thigh Muscles (VL,BF)</td>
<td>Step 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.36 ± 0.24</td>
<td>0.28 ± 0.12</td>
<td>0.347</td>
</tr>
<tr>
<td></td>
<td>0.46 ± 0.49</td>
<td>0.32 ± 0.12</td>
<td>0.291</td>
</tr>
</tbody>
</table>
Figure 1. Maximum hip extensor EMG magnitudes during stair ascent, stair descent, and walking.

Figure 2. Maximum knee extensor EMG magnitudes during stair ascent, stair descent, and walking.
Figure 3. Maximum knee flexor EMG magnitudes during stair ascent, stair descent, and walking.
DISCUSSION

This purpose of this study was to determine if individuals with ACL reconstruction altered their muscle coordination strategies at the knee and hip during the stance phase of stair use and walking. Muscle coordination strategies were characterized by peak muscle activity and co-contraction indices during the stance phase of three everyday tasks. Individual muscles were grouped based on their function as hip extensors, knee extensors, and knee flexors. Quantifying neuromuscular control in this cohort provides insight into how the knee is stabilized during dynamic loading. Control of the knee and hip musculature influences loads placed on the knee joint. Altered loading patterns may contribute to the onset of early posttraumatic knee OA in those with a reconstructed ACL.

Our hypothesis that quadriceps muscle activity would be impaired in the ACL group was not supported. Previous studies have reported persistent neuromuscular impairments in persons with ACL reconstruction including quadriceps weakness due to arthrogenic inhibition (Hart et al., 2010b). In that study, the presence of arthrogenic inhibition suggested impaired neural activity of the quadriceps. Quadriceps weakness and inhibition have been associated with altered kinetic and kinematic gait patterns in those with ACL reconstruction. In our study, however no significant differences were observed in knee extensor strength or the magnitude of quadriceps activity for the ACL reconstruction group as compared to the healthy control group. Previously, no difference in vastus lateralis muscle activity during walking or jogging in those with ACL reconstruction has been documented (Lewek et al., 2002). Our results indicate that the magnitude of quadriceps activity is not altered during stair use and walking when evaluated more than one year following ACL reconstruction surgery. Normalized EMG activity is fundamentally different than the central activation ratio which is often used to determine the presence of inhibition. Central activation ratio is the
ratio between volitional activation and the magnitude of activation achieved with volitional
effort and electrical stimulation (Hart et al., 2010a). Perhaps those with ACL reconstruction
have central activation ratio deficits; however, the magnitude of EMG activity required to
walk or use stairs was not sufficient to detect neural deficits related to maximal activation.

Movements such as jump landing require greater quadriceps activity to balance the
associated increase in external knee flexion moment. For example, during propulsion of a
single-leg countermovement jump where greater demand is placed on the knee extensors,
vastus medialis activity was reduced in those on average 5 years post-ACL reconstruction
(Nyland et al., 2010). Our study suggests that during daily activities of stair use and walking,
individuals with ACL reconstruction do not alter the magnitude of knee extensor muscle
activity as compared to a healthy control group. Perhaps persons with ACL reconstruction
have unaffected knee extensor activity under a certain threshold of knee joint loading.

Cadaver studies have established that during loaded isometric flexion, hamstring co-
contraction reduces strain on the ACL, preventing anterior tibial translation and internal
rotation of the knee (MacWilliams et al., 1999; Hirokawa et al., 1991). Our hypothesis that
knee flexor activity would increase in the post-ACL group was partially supported. The
increased hamstring activity found during stair ascent and walking in the current study is
likely a natural adaptation of the neuromuscular system to protect the reconstructed ACL
(MacWilliams et al., 1999). Increased hamstring activity is consistent with a strategy to
prevent strain on the ACL and reduce anterior tibial translation. However, the increased
hamstring force intensifies compressive loads and alters the patterns of shear in the
tibiofemoral joint (MacWilliams et al., 1999). These altered loading patterns affect the
morphology of the articular cartilage and may contribute to joint degeneration (Arokoski et
al., 2000). Since the properties of articular cartilage are influenced by mechanical loading of
the joint, altered knee joint loading as influenced by increased hamstring activity is an important factor when considering long-term joint health.

Our hypothesis that knee extensor/flexor co-contraction would increase for individuals post-ACL reconstruction was partially supported. Knee extensor/flexor co-contraction was significantly increased during the first step of stair ascent. Stair ascent is considered the most challenging of the three tasks in terms of increased knee extensor moment requirements (Yu et al., 1997). Increased co-contractions of the knee extensors and knee flexors is likely to provide additional knee stability as stair ascent requires increased control of the knee in the sagittal plane. We suggest that greater co-contractions of the knee extensors and flexors contribute to preventing anterior tibial displacement and to control external varus loads at the knee joint. Increased knee extensor/flexor co-contractions may be a result of increased knee joint laxity, but would increase knee joint compressive forces and may induce further joint space narrowing. Longitudinal studies are needed to determine the role of increased co-contraction in the development of articular degeneration (Zeni et al., 2010).

Previous research groups have found increased hamstring activity, although few researchers account for the gluteus maximus as a hip extensor. The results of this study support our hypothesis that hip extensor activity is increased in persons with ACL reconstruction. The gluteus maximus has the potential to exert a posteriorly directed force on the proximal femur to extend the knee while the pelvis and foot are stabilized (Ernst et al., 2000). A combination of the knee flexors with the gluteus maximus allows the quantification of increased hip extensor activity during walking and stair use. Increased gluteus maximus activity has been reported in persons with ACL reconstruction during a single leg countermovement jump (Nyland et al., 2010). Persons with ACL reconstruction
adopt a strategy to increase knee flexor and hip extensor muscle activity. These findings concur with kinetic data that indicate those with ACL reconstruction have increased hip extensor moments, increased knee flexor moments, and reduced knee extensor moments (Nyland et al., 2010).

The contribution of the quadriceps to frontal plane stability is debated. Zeni et al. (2010) explains that the influence of the quadriceps in frontal plane control is insignificant. This research group suggests that the quadriceps act through the patellar tendon resulting in a muscle orientation vector that is anterior to the knee joint center and midway between the medial and lateral plateaus. However, Zhang and Wang (2001) suggest that a combination of medial quadriceps and hamstring activity provide resistance to knee valgus loads and that lateral quadriceps and hamstring activity is necessary to resist varus loads on the knee. When considering the insertions of the hamstrings, it is conceivable that they contribute to frontal plane stability. The hamstring muscles are inserted posterior to the knee at varied locations. The SM is posterior and medial to the knee joint center, enabling an internal flexion moment and an internal knee varus moment. In the current study, our hypothesis that medial and lateral thigh muscle co-contractions would be altered in the ACL reconstruction group was partially supported. Co-contraction of the VM and SM was increased during walking in those with ACL reconstruction as compared to the control group. This finding suggests that the load distribution is altered in the knee joint and that medial compartment compression may be increased in the post-ACL group. This is clinically relevant when considering that an everyday repetitive movement such as walking may increase medial compression in the knee joint, the compartment most affected with knee OA. Knee OA patients have been found to increase lateral thigh muscle activation to aid in stabilizing increased knee varus moments (Heiden et al., 2009), but there were no significant increases in lateral thigh muscle CCI in the current study.
There are several limitations of this study. The methodology of using MVIC to determine individual muscle activity and CCI is a limitation. Differences in quality of individual MVICs may inflate or reduce group differences in muscle activity during the tasks and in CCI calculations (Zeni et al., 2010). Despite all participants being encouraged to maximally contract their muscles, inconsistencies in effort and neuromuscular control may be have been introduced during the MVIC collection. Participants were not restrained to a seated position during the knee extensor and flexor MVICs and therefore some minor differences hip and pelvic alignment may have affected the measurements. Additionally, using two researchers to provide manual resistance may influence the accuracy of the strength data despite high inter-rater reliability and good intra-rater reliability (ICC = 0.89-0.97, ICC = 0.76, respectively). Nonetheless, we believe the use of MVIC during our data collection provided a valid and repeatable measure of participant force generating abilities. Furthermore, previous research has confirmed no differences in voluntary activation or knee extensor strength in persons with previous ACL reconstruction (Lewek et al., 2002).

This study focused on the stance phase, which is the period where the knee joint is most dynamically loaded. Characterizing neuromuscular strategies throughout the entire gait cycle is considered valuable when assessing muscle response to changes in joint dynamics (Hubley-Kozey et al., 2009). Additionally, the interpretation of individual muscle activity was limited to magnitude as the onset and offset timing of the muscles was not determined in this study. Previously, Knoll et al. (2004) observed no changes in the timing of muscle firing in those with ACL reconstruction one year following surgery. However, altered timing patterns of the lower extremity in knee OA patients have been identified, and this is a promising area for further study within a post-ACL reconstruction group.
To summarize, the results of this study indicate that persons with ACL reconstruction increase hip extensor muscle activity as assessed through EMG magnitudes for the gluteus maximus, semimembranosus, and biceps femoris. These neuromuscular adaptations were observed during stair ascent, stair descent, and walking for individuals who were at least one year post-ACL reconstruction. These adaptations may help to maintain dynamic stability and prevent anterior tibial translation. The concern is that these adaptations may lead to increased knee joint compression, knee joint degeneration, and the development of knee OA. This study provides a step toward understanding the long-term muscle responses of those with ACL reconstruction during dynamic tasks of stair use and walking. The results of this investigation into neuromuscular control may provide insight for the development of interventions aimed at correcting the loading distribution on the knee joint.
REFERENCES


CHAPTER 5: GENERAL CONCLUSIONS

The two manuscripts presented in this thesis highlight biomechanical and neuromuscular adaptations that those with ACL reconstruction make to successfully perform activities of daily living.

The presence of meniscal damage at time of acute injury cannot be underestimated when considering the early onset of posttraumatic knee osteoarthritis. While it is thought that almost 50% of those with ACL injury will develop knee OA, the range within the literature is substantial (1-100% Oiestad et al., 2009). Selmi et al. (2006) followed 103 patients with ACL reconstruction 17 years post-surgery and found 52% had normal radiographic knees, and 28% had early onset of posttraumatic knee OA. Interestingly, if the meniscus was normal at the time of initial reconstruction or was successfully repaired, then the rate of those with knee OA was 14%. The incidence rose to 37% in individuals who had a medial meniscectomy. Future studies should consider the presence of meniscal injuries when focusing on the development of knee OA following ACL injury.

Reduced knee extensor moments are often considered a result of strength and neural deficits in the quadriceps musculature. We contend that, although net knee extensor moments were reduced in the ACL group, the ability of the quadriceps to generate an extensor moment (as assessed through strength measures and neural activity) during stair use or walking was not impaired. Rather, it is the increased knee flexor activity that reduced the net knee extensor moments. In addition, net hip extensor moments were increased as a compensatory adjustment to the reduced net knee extensor moments. Increased knee flexor activity would help prevent excessive anterior tibial translation and
strain on the reconstructed ACL. Protection of the reconstructed ACL through quadriceps/hamstring co-contraction likely comes at a cost. Increased quadriceps/hamstring co-contraction would be expected to increase compressive forces within the menisci and articular cartilage of the knee joint. It is likely that loading on the articular cartilage is altered beyond what it was physiologically accustomed to and therefore has developed abnormal repair attempts.

We acknowledge that hand-held dynamometry is not the gold standard for assessing strength, and that may influence the sensitivity of our strength data. Additionally, using two researchers as testers may also influence the reliability of the measure. However, our intraclass correlation coefficient measured was 0.77, and inter-rater reliability ranged between 0.89 and 0.97. As a consistent and repeatable measure that is sensitive enough to detect a clinically relevant gap in strength, no differences were found between the ACL reconstruction and healthy control groups.

The combined neuromuscular and biomechanical lower extremity differences between a control group and individuals an average of 6 years after ACL reconstruction suggest that long-term compensatory adaptations persist. Although relative knee stability is achieved through reconstruction, individuals continue to use neuromuscular adaptations to maintain dynamic stability. Findings from the present study agree with those found with other tasks including single-leg countermovement jump (Nyland et al., 2010) that those with ACL reconstruction attempt to reduce anterior tibial translation by favoring increased hip extensor moments.
REFERENCES


APPENDIX: MAXIMUM VOLUNTARY CONTRACTION MEASURES

Figure 1: Knee extensor MVIC and strength assessment. Participants sat upright with knee flexed approximately 80°. Participant maximally extended the leg against resistance from the researcher.

Figure 2: Knee flexor MVIC and strength assessment. Participants sat upright with knee flexed approximately 90°. Participant maximally flexed the leg against resistance from the researcher.
Figure 3. Hip extensor MVIC assessment. Participant stood upright, supported by table and extended the hip against resistance by researcher.

Figure 4. Hip abductor MVIC assessment. Participant stood upright, supported by wall and abducted the hip against resistance by researcher.
Figure 5. Ankle plantar flexion MVIC assessment. Participant sat and plantar flexed the foot against the adjacent wall.
ACKNOWLEDGEMENTS

Prof. Jason Gillette, I am indebted to you for taking me on as your Master’s student. I have learned so much from your professional approach and thoroughly enjoyed this whole experience. I thrived on the environment you create, and sincerely thank you for your patience and time you spent with me.

Dr. Tim Derrick, it has been fantastic to work with you, take your classes and have your expertise at close hand. I thank you in particular for introducing me to Matlab and having tremendous patience in doing so!

Dr. Gary Mirka, it has been a pleasure to have taken your classes. Your enthusiastic approach is hugely motivating. I particularly enjoyed the IE 671 project and learned a great deal from that experience.

Dr. Mark Gleason, whoever thought I would be including an acknowledgment for a Plant Pathology Professor! Your enthusiasm and genuine interest in your students and employees is infectious. Interacting with you and observing you has taught me so much.

Dr. Katie Stevermer, I appreciate and enjoy your enthusiastic, applied perspective, thank you for sharing it. To all the study participants for making this research possible, thank you for your time and interest in our project. Fellow graduate students and friends thanks for making the past two years so much fun! Finally, I would not be close to be sitting where I am today without the unconditional love and inspiration of my parents, David and Caroline and siblings, Amy and Wayne. Your guidance and influence has catapulted me through a life of endless opportunities and possibilities, for which I immensely enjoy and am grateful for.