THE RELATIONSHIP BETWEEN GLUTEUS MEDIUS ACTIVATION AND FRONTAL PLANE KNEE STABILITY

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ABSTRACT

Excessive knee abduction moment and knee valgus in a weight bearing limb are well known biomechanical risk factors of chronic knee pain such as patellofemoral pain (PFP) or knee osteoarthritis (OA). Neuromuscular control of the hip abductors is important to prevent excessive knee abduction moment and knee valgus. Potential associations between altered neuromuscular control of gluteus medius (GMED) and PFP has been frequently suggested; however, there is limited literature on how neuromuscular control of the GMED is related to the knee abduction moment or knee valgus.

The primary objective of the present study was to examine whether GMED onset and activation magnitude are related to the knee abduction moment and knee valgus. The secondary objective was to investigate the relationship between hip abductor strength and knee abduction moment and valgus.

20 healthy females (22.6 ± 2.5 yrs) performed 15 Single Limb Mini Squats (SLMS) on each leg. Correlations between the GMED activation parameters, hip abductor strength, and frontal plane knee angle and moment were examined separately for each limb in three different phases of the SLMS: Double to single limb transition, single limb stabilization, and descending phase. As secondary analyses, the relationships among frontal plane hip kinematics, kinetics, pelvic obliquity, and frontal plane knee angle and moment were examined separately for each limb in the specific movement phases.

Greater GMED activation magnitude was significantly correlated with a decrease of the knee abduction moment during the single limb stabilization phase in the non-dominant limb only.
The non-dominant limbs experienced significantly greater reduction of the knee abduction moment than the dominant limbs during the single limb stabilization phase. Greater hip abduction strength was correlated with less knee valgus only in the dominant limb during the double to single limb transition phase. Limb dominance may be an important factor when considering the neuromuscular control of GMED for controlling knee abduction moment. These results can provide useful insights for developing strategies for preventing chronic knee pain.
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Dr. Sandra Webber not only provided her own input for designing this study but also helped me to get connected with other physical therapists so that this thesis has practical inputs. She was also a good mentor for me whenever I needed guidance for building academic experiences into my career.

Kirstin Kendel helped me as a research assistant during the entire data collection. She always brought positive energy to the data collection laboratory, and did an excellent job of making participants feel comfortable.

I cannot thank our participants enough. This thesis would not even exist without their help.

Last but not least I would like to acknowledge the colleagues in biomechanics and motor control lab group who inspired me with their diligence and creativity.
DEDICATION

I would like to dedicate this thesis to my parents for their unending support and for their unconditional love.

I also want to dedicate this thesis to Dr. Junggi Hong and Guido Van Ryssegem who truly motivated me to jump into research.
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<td>Pelvic drop</td>
<td>Frontal plane rotation of the pelvis towards unsupported side of the body</td>
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<tr>
<td>Pelvic obliquity</td>
<td>Frontal plane rotation of the pelvis towards supported side of the body</td>
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<td>Frontal plane knee stability</td>
<td>Ability to prevent excessive side to side movement and/or moment of the knee during a weight bearing activity</td>
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LIST OF ABBREVIATIONS

2D Two Dimensional
3D Three Dimensional
ABD Abduction
ADD Adduction
AJC Ankle Joint Centre
ASIS Anterior Superior Iliac Spine
BOS Base of Support
BPM Beats per Minute
COM Centre of Mass
COP Centre of Pressure
EMG Electromyography
ER External Rotation
F/E Flexion / Extension
GMED Gluteus Medius
GRF Ground Reaction Force
HJC Hip Joint Centre
IEMG Integrated EMG signal
IR Internal Rotation
KJC Knee Joint Centre
Mp Net moment acting at proximal joint
msec Milliseconds
OA Osteoarthritis
PCSA Physiological Cross Sectional Area
<table>
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<tr>
<th>Abbreviation</th>
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<tr>
<td>PFP</td>
<td>Patellofemoral Pain</td>
</tr>
<tr>
<td>PSIS</td>
<td>Posterior Superior Iliac Spine</td>
</tr>
<tr>
<td>ROM</td>
<td>Range of Motion</td>
</tr>
<tr>
<td>SD</td>
<td>Standard Deviation</td>
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<tr>
<td>SLMS</td>
<td>Single Limb Mini Squat</td>
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<td>TO</td>
<td>Toe-off of a non-supporting limb</td>
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INTRODUCTION

Chronic knee pain syndromes such as patellofemoral pain (PFP) and knee osteoarthritis (OA) are receiving growing attention in primary health care (Bennell, Bartam, Crossley, & Green, 2000; Ellis, Hing, & Reid, 2007; Tveit, Rosengren, Nilsson, & Karlsson, 2011) because of their strong impact on functional disability, high incidence rates, and health care costs (Lawrence et al., 1998; Ruffin & Kiningham, 1993; Thomas, Wood, Selfe, & Peat, 2010; Waal, Terwee, Windt, Bouter, & Dekker, 2005). Preventing as well as treating knee pain is critical for reducing the burden of medical costs and improving public health (Arroll, Ellis-Pegler, Edwards, & Sutcliffe, 1997; Kettunen et al., 2011).

Although there are multiple risk factors of PFP (Waryasz & McDermott, 2008) and knee OA (Cooper et al., 2000), identifying modifiable risk factors is fundamental to avoid symptomatic pain and further intensification of the pain. Excessive knee abduction and medial collapse of the knee in a weight bearing limb have been consistently reported as biomechanical risk factors of PFP (Aminaka, Pietrosimone, Armstrong, Meszaros, & Gribble, 2011; Paoloni et al., 2010; Stefanyshyn, Stergiou, Lun, Meeuwisse, & Worobets, 2006) and knee OA (Chang et al., 2005). Previous studies (Chang et al., 2005; Conneely & Sullivan, 2008; Noehren, Pohl, Sanchez, Cunningham, & Lattermann, 2011) further investigated neuromuscular determinants of the above mentioned improper mechanics of knee movement. Excessive hip adduction and unilateral drop of the pelvis in single limb stance can significantly increase knee abduction moment and medial collapse of the knee (Chang et al., 2005; Paoloni et al., 2010).

Proper functioning of the hip abductors is important in resisting excessive hip adduction and unilateral drop of the pelvis (Neumann, 2010; Perry & Burnfield, 2010; Powers, 2010). The
majority of attention has been given to gluteus medius (GMED) (Brindle, Mattacola, & McCrory, 2003; French, Dunleavy, & Cusack, 2010; Jacobs, Uhl, Mattacola, Shapiro, & Rayens, 2007; O'Sullivan, Smith, & Sainsbury, 2010) due to its role as a primary hip abductor (Clark & Haynor, 1987) as well as previously observed deficits in activation parameters of the GMED in patients with PFP (Bolgla, Malone, Umberger, & Uhl, 2011). For example, delayed GMED activation onset during stair negotiation was found among patients with PFP when compared to healthy controls (Aminaka et al., 2011; Cowan, Crossley, & Bennell, 2009).

Women are more susceptible to PFP and knee OA than men (Srikanth et al., 2005; Taunton et al., 2002). High-risk movement patterns such as excessive knee abduction, medial collapse of the knee, hip adduction, and pelvic drop have also been observed more frequently in females than males (Chumanov, Wall-Scheffler, & Heiderscheit, 2008; Earl, Monteiro, & Snyder, 2007; Ferber, Davis, & Williams, 2003). Authors of the previous research (Heiderscheit, 2010; Jacobs et al., 2007; Noehren, Davis, & Hamill, 2007) emphasized the need for further studies on the influence of GMED activation in females on unsafe hip and knee movement patterns.

To date, only two studies (Hollman et al., 2009; Willson, Kernozek, Arndt, Reznichek, & Scott Straker, 2011) have examined the biomechanical relationship between the GMED activation parameters and lower limb kinematics among females. The generalizability and applicability of these studies is limited due to the recruitment of participants who already developed PFP and comparison of GMED activation parameters and a kinematic event without careful consideration of the relative timing of the events. In order to provide more knowledge for developing prevention strategies for PFP and knee OA, this study aims to investigate how GMED activation timing and magnitude are associated with frontal plane knee movement among
healthy females with careful consideration of functional relevance between GMED activation and kinematic and kinetic events.
1.1 Patellofemoral pain and knee osteoarthritis: Growing concerns for primary health care

Chronic knee pain syndromes such as patellofemoral pain (PFP) and knee osteoarthritis (OA) are receiving growing attention in primary health care (Bennell et al., 2000; Ellis et al., 2007; Tveit et al., 2011). These knee pain syndromes can begin as a periodic discomfort triggered by specific movements such as running or jumping which may then develop into more chronic disorders (J. P. Fulkerson, 2002; Tveit et al., 2011). They are becoming one of the major concerns for primary health care due to their strong impact on functional disability, high incidence rates, and health care costs. It was reported that knee OA has a substantial influence on decreasing health-related quality of life most likely due to the reduction of physical activity levels (Waal et al., 2005). Previous literature described that approximately 25% of non-athletic young adults had PFP (Ruffin & Kiningham, 1993) and more than 12% of older adults were affected by OA in North America (Lawrence et al., 1998). In addition, an increasing number of concerns are being raised that a non-degenerative knee pain condition such as PFP may be a precursor to a degenerative disorder such as patellofemoral OA (Thomas et al., 2010). Preventing non-traumatic knee pain as well as treating the pain itself is therefore critical for reducing the burden of the medical costs and promoting public health (Arroll et al., 1997; Kettunen et al., 2011).
1.2 *Excessive knee abduction moment: A common risk factor of PFP and knee OA*

It is consistently argued that excessive knee abduction moment plays an important role in development of the chronic knee pain such as PFP and knee OA (Chang et al., 2005; Powers, 2010). In this section, a brief description of the knee abduction moment is first provided followed by details on how the knee abduction moment is involved in the development of PFP and knee OA.

1.2.1 *Internal knee abduction moment: An opposing force of external knee adduction moment*

When our body is in contact with the ground, muscle forces and ground contact forces induce an equal and opposite ground reaction force (GRF) applied on our body (Zatsiorsky & Prilutsky, 2012). This GRF creates torques around our joints, which are named external joint moments (Powers, 2010; Zatsiorsky & Prilutsky, 2012). An external joint moment is opposed by an internal joint moment created by the muscles around the joints and the non-contractile tissues such as ligaments and articulation cartilages (Zatsiorsky & Prilutsky, 2012).

During weight bearing activities such as walking or stair negotiation, the GRF vector is usually oriented medial to the knee joint centre of the weight-bearing limb, thereby creating an external adduction moment at the knee (Powers, 2010; Winter, 2009). An external knee adduction moment is expected to increase the tensile strain on the non-contractile tissues around the knee including the iliotibial band and lateral collateral ligament (Lavine, 2010; Powers, 2010) and to create greater compressive forces within the medial compartment of the tibiofemoral joint (Chang et al., 2005; Miyazaki et al., 2002; Schipplein & Andriacchi, 1991; Sharma et al., 1998). Because the internal knee abduction moment is often considered a major
opposing force of the external adduction moment (Winter, 2009), biomechanical studies for investigating risk factors of chronic knee pain often examine the internal knee abduction moment. Because it is generally thought that an internal knee abduction moment is almost equal and opposite to an external knee adduction moment (Robertson, 2004), a greater internal knee abduction moment is considered to increase the risk for knee pain. Because the internal joint moment, rather than the external joint moment, is a commonly used term, the internal knee abduction moment will be simply termed a knee abduction moment throughout this thesis.

1.2.2 Evidence of excessive knee abduction moment among patients with PFP

Research suggests that PFP commonly develops on the lateral aspect of the patella (Cutbill, Ladly, Bray, Thorne, & Verhoef, 1997; J. Fulkerson & Shea, 1990). Based on these findings, it is suggested that knee abduction moments may play an important role in the development of PFP (Paoloni et al., 2010). Paoloni et al. (2010) compared frontal plane kinetic patterns of the knee between young adults with PFP and age-matched healthy controls while the participants were walking 10 m on a level surface at a self-selected speed. Using a three-dimensional (3D) kinetic analysis, the study found that the patients with PFP displayed a significantly greater knee abduction moment than the healthy controls during loading of the stance leg. An increased knee abduction moment among patients with PFP was also confirmed in the study by Aminaka et al. (2011) that examined the 3D knee kinetics during stair ambulation. A knee abduction moment and a knee abduction impulse (knee abduction moment integrated over time) were examined (Aminaka et al., 2011). When compared to age-matched healthy young adults, the patients with PFP demonstrated a significantly higher peak knee abduction moment during the stair ascent and a significantly increased knee abduction impulse during both stair ascent and descent (Aminaka
et al., 2011). The association between excessive knee abduction moment and PFP has also been supported by a prospective study by Stefanyshyn et al. (2006). In their study, the participants who developed PFP after six months of running showed a significantly greater knee abduction impulse during the baseline measurement compared to that of the age-matched group who did not develop PFP.

1.2.3 Evidence of excessive knee abduction moment among patients with knee OA

Increased abduction moment at the knee has been associated with development and progression of knee OA at the medial compartment of the tibiofemoral joint (Chang et al., 2005; Miyazaki et al., 2002; Schipplein & Andriacchi, 1991; Sharma et al., 1998). According to Chang et al. (2005), the medial tibio-femoral compartment is the most common site of knee OA, mainly because the body weight is generally loaded more on the medial than lateral compartment of the tibial plateaus during the normal gait cycle. Using a statistically determinate model, Schipplein and Andriacchi (1991) found that the knee abduction moment is a major determinant of the biased loading at the medial compartment of the knee. Furthermore, two studies (Miyazaki et al., 2002; Sharma et al., 1998) discovered that a greater knee abduction moment during normal walking was associated with an increased likelihood of medial tibiofemoral OA progression. Sharma et al. (1998) measured lower extremity kinetics of normal gait and disease severity of 54 patients diagnosed with medial tibiofemoral OA. The amount of osteophyte formation and narrowing of the joint space were used to evaluate the severity of the OA in the study (Sharma et al., 1998). When controlled for age, sex, and pain level, the severity of medial tibiofemoral OA was moderately correlated with knee abduction moments that were normalized by weight and height (Sharma et al., 1998). The association between the knee abduction moment and medial
tibiofemoral OA progression found by Sharma et al. (1998) was later confirmed in a longitudinal study by Miyazaki et al. (2002) who measured the severity of medial tibiofemoral OA and the knee abduction moment of 106 patients older than 50 years of age during 1991 - 1993 and at a six year follow up. This study showed that the risk of progression of the medial tibiofemoral OA increased 6.46 times with a 1% increase in abduction moment (Miyazaki et al., 2002).

1.3 Importance of hip control for preventing excessive knee abduction moment

It has been suggested that controlling frontal plane pelvis and femur movement is important to prevent an excessive knee abduction moment (Chang et al., 2005; Paoloni et al., 2010). In this section, hip control mechanisms for preventing excessive knee abduction moment are reviewed.

1.3.1 Association between pelvic drop and increased knee abduction moment

During gait, the lower extremities experience a series of double to single leg transitions. To achieve postural stability during single-leg stance, the vertical projection of the centre of mass (COM) must be shifted from a location between both feet (during double-leg stance) towards a base of support (BOS) underneath a single foot (Horak, 2006). Standing or moving on one leg; however, removes support to the trunk and pelvis on that side of the body causing pelvic drop towards the unsupported side of the body (Patla, 2003). This contralateral pelvic drop can cause the position of the COM to move medial to the supporting limb knee (Chang et al., 2005; Patla, 2003), which increases the knee abduction moment of the supporting limb (Chang et al., 2005; Perry & Burnfield, 2010; Simonsen, Dyhre-Poulsen, Voigt, Aagaard, & Fallentins, 1997). In this thesis, drop of the unsupported side of the pelvis is termed “pelvic drop”, and rise of the contralateral pelvis is termed “pelvic obliquity”. Clinical studies have indicated that pelvic drop
is possibly associated to PFP (Willson, Binder-Macleod, & Davis, 2008; Willson & Davis, 2008). In a study by Willson et al. (2008), young adult females with PFP demonstrated a greater amount of supporting-limb pelvic drop at the end of five consecutive single-legged jumps (during the landing phase) compared with the control group. Willson and Davis (2008) also reported 3.5° greater pelvic drop in the non-supporting limb in a PFP group for various functional movements including single leg squats, running, and repetitive single leg jumps. Chang et al. (2005) also theorized that excessive pelvic drop would accelerate the progression of knee OA by increasing the forces across the medial compartment cartilage of the stance limb knee.

**1.3.2 Association between hip adduction and increased knee abduction moment**

When a limb prepares to accept the body weight during a gait cycle, a moment is created that forces the hip to adduct (Paoloni et al., 2010; Powers, 2010). In order to unload the non-supporting limb and load the supporting limb, the stance limb hip adductors generate a propulsive impulse to initiate a lateral movement of the COM (Rogers & Pai, 1993). According to Stefanyshyn et al. (2006), hip adduction of the stance limb during running may increase the external knee adduction moment by increasing the lever arm (distance between the GRF line of action and the knee joint centre). An increased external knee adduction moment must be counteracted by an internal knee abduction moment. Therefore the net result of increased hip adduction is increased knee abduction moment in a stance limb.
1.4 **Pelvic drop and hip adduction: Mechanisms for poor knee alignment**

In addition to increasing the knee abduction moment, hip adduction and pelvic drop also have an adverse effect on knee joint alignment. In particular, excessive hip adduction is known to substantially contribute to a medial collapse of the knee joint in a weight bearing limb, termed a “dynamic knee valgus” (Ireland, 1999, p. 383). A dynamic knee valgus is receiving growing attention from the investigators of PFP due to its impact on increasing the forces that pull the patella laterally (Powers, 2010; Schulthies, Francis, Fisher, & Van De Graaff, 1995). Pain at the patellofemoral joint is known to be aggravated by the contact pressure between the posterior surface of the patella and the lateral femoral condyle when the patella is positioned on the lateral aspect of the distal femur (Bolgla, Malone, Umberger, & Uhl, 2008; Ferber et al., 2003). In a weight bearing limb, the quadriceps pull the patella towards the tibia (Souza, Draper, Fredericson, & Powers, 2010). In this situation, dynamic knee valgus can substantially increase laterally oriented forces of the quadriceps, which can trigger the pain on the posterior surface of the patella (Powers, Ward, Fredericson, Guillet, & Shellock, 2003).

Pelvic drop is also thought to increase the lateral forces exerted on the patella. Pelvic drop is hypothesized to elongate the iliotibial band in the supporting limb (Willson & Davis, 2008). Elongation of the iliotibial band increases the tension in its structure, which can lead to greater forces that shift the patella laterally (Puniello, 1993; C. C. Wu & Shih, 2004).

Clinicians can assess that a patient is susceptible to dynamic knee valgus when showing a consistent pattern of excessive pelvic drop, hip adduction, hip internal rotation (or knee abduction), or a combination of these signs when performing a single-leg squat or step down (Earl & Vetter, 2007). Two studies have actually found the above mentioned movement patterns that contribute to dynamic valgus among patients with PFP (Salsich & Long-Rossi, 2010;
Willson et al., 2008). Salsich and Long-Rossi (2010) reported that young female adults with PFP demonstrated significantly greater knee abduction in their stance limb during fast speed walking (2 m/sec) when compared to age and gender matched pain-free controls. Willson and Davis (2008) reported that females with PFP demonstrated significantly greater average hip adduction (3.5°) compared to asymptomatic participants during running, hopping, and single-limb squatting.

1.5 The role of neuromuscular control of hip abductors for maintaining knee stability

Based on the aforementioned influences of excessive hip adduction and pelvic drop on increasing the risk factors of PFP and knee OA, proper functioning of the hip abductors has been emphasized (Aminaka et al., 2011; Brindle et al., 2003; Chang et al., 2005). Hip abductors function as antagonists of the hip adductors and are therefore important for preventing excessive hip adduction. Generating a hip abduction moment at the stance limb has also been emphasized for preventing the pelvic drop (Chang et al., 2005). Theoretically, the hip abduction moment generated by hip abductor muscles can resist excessive pelvic drop to facilitate COM movement towards the supporting leg, thereby preventing an excessive knee abduction moment (Neumann, 2010). Chang et al. (2005) prospectively investigated the relationship between baseline hip internal abduction moment during gait and the medial tibiofemoral OA progression after 18 months of follow-up. The study concluded that the hip abduction moment was protective against the progression of medial knee OA (Chang et al., 2005).
1.6 The role of hip abductor strength on knee stability is unclear

Comparisons between people with and without knee disorders reveal relative weakness in hip abduction in people with the PFP (Bolgla et al., 2011). In accordance with these retrospective findings, therapeutic exercises for strengthening hip abductors are reported to be effective in reducing knee pain (Dolak et al., 2011; Ferber, Kendall, & Farr, 2011; Khayambashi, Mohammadkhani, Ghaznavi, Lyle, & Powers, 2011). These findings suggest that hip abductor weakness contributes to excessive hip adduction and pelvic drop and subsequent increase of a knee abduction moment (Ferber et al., 2003).

Numerous investigations (Bolgla et al., 2008; Rutherford & Hubley-Kozey, 2009; Thijs, Pattyn, Van Tiggelen, Rombaut, & Witvrouw, 2011; Thijs, Van Tiggelen, Willems, De Clercq, & Witvrouw, 2007) have disputed the presumed association between hip abductor strength and the biomechanical risk factors of knee pain. Bolgla et al. (2008) investigated if hip muscle weakness in patients with PFP was related to excessive hip adduction and dynamic knee valgus during stair descent. When the isometric hip abduction strength (normalized to body weight) was compared, females diagnosed with PFP showed 26% less strength than the healthy controls. However, no between-group differences were found for average hip and knee frontal plane angles during stair descent. The study suggested that hip abductor weakness may not be the contributing factor for altered hip and knee kinematics (Bolgla et al., 2008). Rutherford and Hubley-Kozey (2009) investigated the relationship between hip abductor muscle strength and frontal plane hip moments during gait in 22 healthy individuals. When hip moment and strength were normalized to body weight, the maximal isometric hip abductor strength could not explain the variability in the internal hip abduction moment in both initial and mid-stance phases. The study concluded that hip abductor strength was not significantly related to the hip abductor
moment (Rutherford & Hubley-Kozey, 2009). Thijs et al. (2007) examined the relationship between hip muscle strength and frontal plane knee kinematics of healthy young adults. Isometric strength of hip abductor, adductor, flexor, extensor, and external and internal rotator muscle groups was measured. Peak knee valgus or varus angles for each participant during the three forward lunge movements were measured using a 2D kinematic analysis. The study did not find a significant relationship between frontal plane knee angle and any of the assessed hip muscle strength values. Moreover, a recent prospective study (Thijs et al., 2011) also disputed an association between weakness of hip abductors and PFP. The study investigated if hip muscle weakness is a predisposing factor for the development of PFP. The investigators compared the base line hip strength between the groups who did and did not develop PFP after a 10-week running program. Isometric hip flexor, extensor, abductor, adductor, and external and internal rotator muscle strength was measured in 77 healthy female novice runners, and normalized to their body weight. The study found no significant differences in any of the assessed hip strength profiles between the runners who did and did not develop PFP (Thijs et al., 2011).

1.7 The role of neuromuscular control of hip abductors in knee pain

In response to the unclear relationship between hip abductor weakness and abnormal knee mechanics, it has been suggested that research should focus more on altered neuromuscular control of the hip abductors rather than the strength of the muscle (Heiderscheit, 2010). Particular attention has been paid to gluteus medius (GMED) in studies investigating how the neuromuscular control of hip abductors is related to the development of knee pain (Aminaka et al., 2011; Brindle et al., 2003; Cowan et al., 2009; Willson et al., 2011). In this
section, the anatomical function of GMED will be explained. Then the two major characteristics of neuromuscular control of the GMED will be described based on the mechanical challenges on dynamic stability during both single and double limb stance.

GMED is the most superficial and the largest muscle among the primary hip abductors including gluteus minimus and tensor fascia latae (Clark & Haynor, 1987; Kendall, McCreary, & Kendall, 1983). The GMED provides a longer lever arm for hip abduction than gluteus minimus and a larger cross-sectional area for force production than tensor fascia latae (Kendall et al., 1983; Neumann, 2010). Therefore the GMED is often recognized as the most efficient hip abductor for controlling both femur-on-pelvis and pelvis-on-femur motion (Kendall et al., 1983; Neumann, 2010; Ward, Winters, & Blemker, 2010).

One characteristic of neuromuscular control of the hip abductors is the activation magnitude. During single limb weight bearing activities, the hip abductor moment accounts for most of the forces resisting a contralateral drop of the pelvis (Hurwitz, Foucher, & Andriacchi, 2003). In order to achieve frontal plane stability, hip abductors are expected to produce at least twice as much force as the body weight (Neumann, 2010), because the moment arm used by the hip abductor muscles is about half of the moment arm used by gravity (Neumann, Soderberg, & Cook, 1988). The hip abductors, except tensor fascia latae, have large physiological cross sectional areas (PCSA), enabling a relatively large number of sarcomeres to generate the abduction force simultaneously (Ward et al., 2010). In order to get the true benefit of the large PCSA of the hip abductors, a considerable amount of motor unit recruitment is critical (Perry & Burnfield, 2010). Only two studies (Brindle et al., 2003; Willson et al., 2011) examined if the magnitude of gluteus medius (GMED) activation was altered among patients with PFP: Neither
found a significant association between GMED activation magnitude and PFP. The association between the magnitude of hip abductor activation and knee OA has not yet been investigated.

Another characteristic of neuromuscular control of the hip abductors is the timing of activation. The body COM during the single limb stance phase of walking remains medial to the base of support (BOS) (Patla, 2003). A fall is prevented by the forward stepping of the swing limb which forms a transient double limb BOS. Each time a single limb support phase begins, the COM shifts towards the unsupported side (Winter, 2009). The lateral shift of COM causes the external adduction moment about the hip to drastically increase in the single limb stance phase of gait (Neumann, 2010). This external hip adduction moment is “the destabilizing force” (Lepers & Brenière, 1995, p. 123) that leads the hip to adduct, the contralateral pelvis to drop, and the knee to a valgus position (Earl & Vetter, 2007; Paoloni et al., 2010; Powers, 2010). Because there exists an inevitable delay between the onset of electrical activity and force development of a muscle group (Cavanagh & Komi, 1979), the hip abductors should be activated before the destabilizing forces begin to take effect. It is well established that our central nervous system can predict the timing of known destabilizing forces and preserve joint and postural stability using proactive neuromuscular strategies (Patla, 2003). Several studies (Aminaka et al., 2011; Brindle et al., 2003; Cowan et al., 2009; Willson et al., 2011) have found a delayed onset of GMED among patients with PFP, and suggest that the timing of the proactive onset of hip abductors may be related to frontal plane hip and knee kinematics and kinetics. Brindle et al. (2003) and Aminaka et al. (2011) examined GMED onset relative to when the stance foot had initial contact while the participants were stepping up and down stairs. These studies found that patients with PFP had significantly slower onset of GMED compared to the healthy participants in both stair ascent and descent. In the study by Brindle et al. (2003), the
PFP group showed GMED onset 88.1 ± 110.3 msec prior to toe-contact while ascending stairs, whereas the healthy controls showed GMED onset 182.1 ± 110.5 msec prior to toe-contact. The PFP group also showed slower GMED onset during the stair descent (-238.3 ± 202.4 msec for healthy controls, -333.9 ± 144.9 msec for PFP group) (Brindle et al., 2003). In the study by Aminaka et al. (2011), the PFP group showed GMED onset 38.38 ± 49.05 after toe-contact while descending stairs, whereas the healthy participants showed GMED onset 69.95 ± 88.20 prior to toe-contact. Cowan et al. (2009) also examined GMED onset relative to foot contact as participants were stepping up onto a force platform at their maximum speed. The patients with PFP showed GMED onset after foot contact, while the healthy participants activated their GMED prior to foot contact (Cowan et al., 2009). Willson et al. (2011) compared GMED onset prior to foot contact during running between patients with PFP and healthy participants. The study reported significantly slower GMED onset among the PFP group (59.7 ± 32.6 msec for healthy group, 35.2 ± 32.3 msec for PFP group).

1.8 Female biased biomechanical risk factors of knee pain

Incidence rates of PFP (Boling et al., 2010) and knee OA (Srikanth et al., 2005) are known to be higher among females than males. A 2.5 year of longitudinal study by Boling et al. (2010) on 1525 participants from the United States Naval Academy reported a 2.23 times higher incidence rate of PFP among females compared to males. A review by Taunton et al. (2002) reported that in England, female runners had an approximately two times greater incidence rate of PFP than male runners in 2002. A meta analysis by Srikanth et al. (2005) revealed a 37% higher incidence rate of knee OA in Australian females than males who were younger than 55 years old.
Studies investigating possible causations for the gender discrepancy have identified that females tend to have a greater knee valgus angle and femoral anteversion in a static standing position when compared to males (Ireland, 1999; Sheehan, Derasari, Fine, Brindle, & Alter, 2010). The aforementioned anatomical risk factors may lead females to adduct their hips and abduct their knees during weight bearing movement (Ireland, 1999). Females have shown greater hip adduction angle, and greater knee abduction moment in the stance limb during weight bearing activities such as walking, running, single leg landing from a jump, or walking down the stairs when compared to male participants (Chumanov et al., 2008; Decker, Torry, Wyland, Sterett, & Richard Steadman, 2003; Earl et al., 2007; Ferber et al., 2003; Ford, Myer, & Hewett, 2003; Malinzak, Colby, Kirkendall, Yu, & Garrett, 2001; Sigward & Powers, 2006). In accordance with the previously suggested relationship between hip abductor function and frontal plane knee mechanics (Powers, 2010), previous studies also found a lower activation level of the hip abductors (Chimera, Swanik, Swanik, & Straub, 2004) and a weaker hip abductor strength (Claiborne, Armstrong, Gandhi, & Pincivero, 2006) among females.

Neuromuscular risk factors are potentially modifiable risk factors, whereas anatomical risk factors are considered non-modifiable risk factors (Ireland, 1999). For this reason, authors of the previous research (Heiderscheit, 2010; Jacobs et al., 2007; Noehren et al., 2007) encouraged more studies on the influence of the neuromuscular control of GMED on the high-risk frontal plane knee movements in females for developing prevention strategies for chronic knee pain.
1.9 **Gaps of the current literature and strategies for filling the gap**

While the growing body of literature suggests potential associations between neuromuscular control of GMED and knee pain such as PFP, only two studies (Hollman et al., 2009; Willson et al., 2011) have directly examined the relationship between GMED activation and frontal plane knee kinematics. In this section, the gap of the current knowledge will be identified including the limitations of each study. Based on the gaps, recommendations for more meticulous investigations will be provided.

Hollman et al. (2009) found no significant relationship between GMED activation magnitude and two dimensional (2D) frontal plane hip and knee angles while healthy female participants were performing single-limb step-downs. However, the method used in the study may not be appropriate to examine the association between the muscular activation and related movement patterns: In their study, the GMED activation magnitude was calculated by taking the average electromyography (EMG) activity level during a 500 msec epoch surrounding peak activation level. Then the correlation between the calculated GMED activation magnitude and the maximum frontal plane angle of hip and knee was tested. No explanation was provided if the peak GMED activation level occurred before or after the timing of the peak frontal plane joint excursion. Without knowing the timing of the peak GMED activation relative to the peak joint excursion, we cannot be sure if the GMED activation magnitude calculated in the study was functionally relevant to the examined joint kinematics.

Use of a 2D method for calculating the frontal plane knee angles is another limitation of the study by Hollman et al. (2009). Ageberg et al. (2010) suggested caution when interpreting weight bearing limb kinematic data collected by a 2D method, because hip internal rotation can significantly contribute to increased hip adduction in a 2D model. Therefore, the result of no
significant relationship between GMED activation and hip adduction shown by (Hollman et al., 2009) might have been different if a 3D method was used.

Willson et al. (2011) examined the relationship between GMED activation and 3D frontal plane hip and knee kinematics while female participants with PFP were running. The timing of GMED activation was calculated relative to foot contact on the ground. Two different magnitudes of the GMED activation were calculated by taking the peak and the average EMG activity levels occurring between onset and offset. Hip and knee frontal plane angles were calculated from the time of initial foot contact to the time of peak vertical GRF. The results showed that only hip adduction excursion was related to the onset of GMED. Although this study found a potential relationship between GMED activation timing and frontal plane hip kinematics, a major limitation was pointed out by the authors themselves: The relationship was examined among the participants who already developed PFP. Because there is a possibility that the expectation of pain might have delayed onset of the GMED (Moseley, Nicholas, & Hodges, 2004; Willson et al., 2011), it is not clear whether or not the observed relationship existed prior to development of PFP. Considering current emphasis on developing prevention strategies for knee pain, it is necessary to examine if there is a relationship between GMED activation and hip and knee kinematics among healthy participants.

While all of the previous investigations (Aminaka et al., 2011; Brindle et al., 2003; Cowan et al., 2009) on the association between GMED onset and knee pain examined stair walking, the study by Willson et al. (2011) examined the relationship between GMED onset and the knee kinematics in running. It is well known that running and walking are two different motor patterns having different invariant activation timing of the lower limb muscles (Cappellini, Ivanenko, Poppele, & Lacquaniti, 2006). Therefore the biomechanical study by
Willson et al. (2011) might not be directly applicable to the previous clinical studies (Aminaka et al., 2011; Brindle et al., 2003; Cowan et al., 2009).

The Single Limb Mini Squat (SLMS) is an appropriate movement for a laboratory based biomechanical study because it is a commonly used screening tool for clinical assessment of frontal plane hip and knee movement during weight bearing movement that is functionally similar to stair negotiation (Ageberg et al., 2010). To perform the SLMS, patients stand on one leg and descend their body by flexing hip, knee, and ankle. Physical therapists or orthopedic physicians pay particular attention to the lower extremity joint behavior during the descent because increased knee valgus and hip adduction during the descending movement of the SLMS has been observed in individuals with chronic knee pain such as PFP (Ageberg et al., 2010; Boling, Bolgla, Mattacola, Uhl, & Hosey, 2006).

The SLMS is not only a movement screening tool but also an effective rehabilitation exercise for chronic knee pain (Boling et al., 2006; French et al., 2010). One of the major purposes of the SLMS exercise is to enhance the activity of the GMED (French et al., 2010). Recently, it was reported that the quality of SLMS performance can reliably represent the functional quality of GMED activation (Crossley, Zhang, Schache, Bryant, & Cowan, 2011). For example, a participant whose SLMS was rated as poor showed relatively slower onset of the GMED compared to another participant whose SLMS was rated as good (Crossley et al., 2011). In the study, ratings for the performance of SLMS were qualitatively measured by experienced clinicians. Part of the criteria for the qualitative measurements included the extent of knee valgus, hip adduction, and pelvic obliquity, which are known to be modifiable biomechanical risk factors of the chronic knee pain (Powers, 2010). Therefore examining how GMED activation is related to frontal plane hip and knee kinematics and kinetics during a SLMS will
provide useful biomechanical and clinical insights for understanding the mechanism of GMED control of knee stability and for the development of prevention strategies.

Careful consideration of functional demands of each movement phase will be required when examining GMED control of knee stability. The biomechanical study by Willson et al. (2011) calculated average GMED activation magnitude over the entire stance phase of running to examine its relationship with maximum frontal plane hip and knee joint excursion. According to Novacheck (1998), lower limb muscles are mainly responsible for impact absorption during the beginning half of the stance phase of running and for propulsive energy generation during the latter half of the stance phase. These different functional demands would require different neuromuscular responses from the GMED of the stance limb. Maximum hip adduction and knee valgus are likely to occur in response to the impact absorption not the propulsion (Brophy, Silvers, Gonzales, & Mandelbaum, 2010; L. Y. Griffin et al., 2000). Therefore an average of GMED activation magnitude over the entire stance phase of running might have not been functionally relevant to the maximum hip adduction and knee valgus occurring during the stance phase of running.

An electromechanical delay between the onset of GMED and the development of hip abduction force should also be taken into account. Buchanan, Lloyd, Manal, and Besier (2004) suggested that there always exists a delay between electrical activity and muscle force production even in already activated muscles. Therefore it is not appropriate to assume that a change of GMED activation magnitude within a certain phase is expected to take immediate effect on the kinematic or kinetic change of the hip or knee within the same phase. For example, in case of the study by Willson et al. (2011), it might not be appropriate to only examine the relationship between GMED activation magnitude and the kinematic changes during the impact.
absorption part of the stance phase. For a more appropriate investigation, it would be necessary to also examine how GMED activation magnitude of one movement phase can be related to the kinematic and kinetic patterns of the hip and knee in the next movement phase.

Another noticeable gap in the literature is that the knee abduction moment was not examined in the studies investigating the influence of GMED activation on the biomechanical risk factors of the knee pain (Hollman et al., 2009; Willson et al., 2011). Many biomechanical and clinical studies have suggested that an excessive knee abduction moment plays an important role in the development of PFP (Aminaka et al., 2011; Paoloni et al., 2010; Stefanyshyn et al., 2006) and knee OA (Chang et al., 2005; Miyazaki et al., 2002; Schipplein & Andriacchi, 1991; Sharma et al., 1998). Therefore analysis of the relationship between GMED activation and knee abduction moment may provide insight for developing strategies to prevent or minimize knee pain.

Choosing an appropriate reference event for detecting GMED activation is also very important for a careful investigation into the relationship between GMED activation and knee abduction moment. Most of the previous studies (Aminaka et al., 2011; Brindle et al., 2003; Cowan et al., 2009; Willson et al., 2011) reported the time of GMED onset relative to foot contact which is a movement event representing the beginning of weight acceptance on a stance limb. At foot contact, the participants were in double limb stance phase in which the pelvis had bilateral support from the lower limbs. It has been consistently proposed that pelvic drop substantially increases the knee abduction moment of a stance limb (Chang et al., 2005; Powers, 2010; Willson et al., 2008). It is expected that the pelvis is under the maximal influence of the destabilizing force that causes the pelvic drop when it loses the support from one leg. Therefore, using a reference movement event that represents the initiation of single limb stance rather than
the beginning of a limb loading would be more appropriate for investigating the association between GMED onset and frontal plane knee kinetics. It is often recommended to maintain the pelvis level at toe-off of the unloading limb while performing SLMS to minimize the knee abduction moment before going into the descending movement (Medicine, 2010). Therefore toe-off of the unloading limb at the end of the double to single limb transition of SLMS may be a good reference event to detect GMED onset.

Both Hollman et al. (2009) and Willson et al. (2011) examined the kinematic and neuromuscular patterns in only one leg of each participant. A significant effect of limb dominance on hip and knee frontal plane kinematics has been reported (Ford et al., 2003; Zifchock & Davis, 2008). In addition, Brophy et al. (2010) suggested that there is a possibility that an ability to coordinate neuromuscular control might be different between dominant and non-dominant legs, because our daily activities usually impose different functional demands on each limb. For example, according to Grouios, Hatzitaki, Kollias, and Koidou (2009), dominant limbs are usually used for manipulating objects, while non-dominant limbs are mostly responsible for providing support during the activities. Therefore it is necessary to analyze the data of both dominant and non-dominant legs.

1.9.1 Strategies for further study

Considering the gaps of the literature identified above, more meticulous investigations on the association between GMED activation characteristics and frontal plane knee stability are required. An ideal investigation should include measurement of 3D kinematics and kinetic patterns of both dominant and non-dominant legs of female participants who do not have any indication of knee pain. The use of the SLMS is recommended as it is a commonly used clinical
screening tool that is functionally similar to stair negotiation. The phases of SLMS should be identified based on the functional goal of each phase. Based on the identified movement phases, the influence of GMED activation on the frontal plane knee kinematics and kinetics in the next phase, as well as that within the same phase should be investigated. Specifically, proactive GMED onset relative to toe-off of the unloading limb should be measured, because the toe-off timing represents the initiation of single limb stance.

1.10 Summary

Chronic knee pain such as patellofemoral pain and knee osteoarthritis is a growing concern for primary health care providers. Excessive knee abduction moments and dynamic knee valgus are noticeable biomechanical mechanisms for the development and progression of knee pain. Proper functioning of hip abductors is important for preventing excessive knee abduction moments and dynamic knee valgus. Neuromuscular control of the GMED has been emphasized as a muscle of interest due to the potential association between delayed GMED onset and PFP. Therefore investigating how neuromuscular control of GMED affects the management of knee abduction moments and dynamic knee valgus will provide a useful insight for developing prevention strategies for knee pain.

There is limited literature on the association between GMED activation and frontal plane knee kinetics and kinematics. The existing literature has many limitations, which present significant gaps in the literature. First, functional relevance between kinematic and kinetic events and GMED activation has not been carefully considered. Second, the movements investigated in the biomechanical studies had different neuromuscular control requirements
than the movements in the previous clinical studies which reported a potential association between delayed GMED onset and knee pain. Third, including participants who already developed patellofemoral pain limits the applicability of the study for the development of a prevention strategy. Finally, investigating only one leg may not be the best approach of elucidating the GMED mechanism for kinematic and kinetic control of the knee.

Considering that females are more susceptible for both the above mentioned chronic knee pain and proposed biomechanical risk factors, this study has been designed to further investigate how GMED activation timing and magnitude are related to the frontal plane kinematics and kinetics of the knee among females. GMED activation and 3D knee kinematics and kinetic patterns will be examined in both dominant and non-dominant limbs during a Single Limb Mini Squat (SLMS). The GMED onset will be detected relative to toe-off timing during the double to single limb transition. The association between the GMED activation timing and magnitude, and kinematic and kinetic knee patterns will be examined within and between functionally unique movement phases of the SLMS.

1.11 Objectives

The primary objective is to investigate how GMED activation timing and magnitude are related to knee abduction moment and dynamic knee valgus during the performance of a SLMS. The secondary objective is to clarify if hip abductor strength is related to knee abduction moment and dynamic knee valgus during the performance of a single limb mini squat. If a significant relationship between GMED activation parameters and the frontal plane knee kinetics and
kinematics is identified, it will be investigated if the observed relationship is influenced by hip adduction or pelvic drop as the tertiary objective.

1.12 Hypothesis

Excessive knee abduction moment and knee valgus are well known biomechanical factors that contribute to chronic knee pain syndromes such as PFP and knee OA. Because past research has shown a potential association between slower onset and lower activation magnitude of GMED and PFP, it is hypothesized that slower onset and lower magnitude of GMED activation will be significantly related to increased knee abduction moment and increased dynamic knee valgus.
2 METHODOLOGY

2.1 Protocol overview
The study was a cross-sectional design which required a single visit of the participants to the laboratories. The movement data was collected in the Musculoskeletal Biomechanics Lab using a three dimensional (3D) motion capture system, a high-speed video camera, two force platforms, and surface electromyography (EMG). The strength data was collected in the Motor Control Lab using an isokinetic dynamometer. Both of the laboratories are located in the Physical Activity Complex on the University of Saskatchewan campus.

2.2 Participants
Twenty female participants between the ages of 18 to 39 years were recruited from the University of Saskatchewan. An a priori calculation to achieve a statistical power of 0.70 indicated that a sample size of 20 would be necessary to detect correlation coefficients of 0.50 or greater at $\alpha = 0.05$. A gender limitation was set to exclude any gender effect on lower body movement patterns as it is well known that a female bias exists regarding specific hip and knee movement patterns related to chronic knee pain (Bišèeviæ, Tomiæ, Starc, & Smrke, 2005; Zeller, McCrory, Kibler, & Uhl, 2003). An age limitation was set based on previous epidemiologic observations that listed postmenopausal hormone deficiency as a risk factor for knee OA and knee pain in women (Van Saase, Van Romunde, Cats, Vandenbroucke, & Valkenburg, 1989). Because pain, injuries and knowledge of movement mechanics may modify the movement pattern of the participants, participant selection was limited to healthy females without a history of knee and ankle ligament injuries, indication of chronic knee pain, low back pain, and/or the
knowledge of proper squat technique. Potential participants were introduced to the thesis project with a verbal presentation by the student researcher and/or electronic advertisement. Interested participants were required to contact the student researcher. Before coming to the laboratory for data collection, an initial pre-screening was performed using questionnaires (Appendices A & B) to exclude participants based on age, gender, and the above mentioned exclusion criteria.

2.3 Instrumentation

2.3.1 Visual Analogue Scale
A visual analogue scale is commonly used for the self-assessment of the indication of patellofemoral pain PFP (Chesworth, Culham, Tata, & Peat, 1989). The self-assessment is a series of 10 cm numeric weighting scales identifying the extent of knee pain while performing activities including walking, running, stair ascent and descent, prolonged sitting, kneeling, squatting, participating in a sport, resting following the sport activities. Going from left to right, the scale changes from “no pain” to “worst imaginable pain”. Following the criteria used in the previous studies (Boling et al., 2006; Cowan et al., 2009; Willson et al., 2011) examining the association between the GMED activity characteristics and the knee kinematics, volunteers who experienced a pain scale of more than 3cm in two or more of the above mentioned activities during the past week were excluded from the present study.

2.3.2 Modified Waterloo Footedness Questionnaire
Limb dominance of a participant was identified with the Modified Waterloo Footedness Questionnaire (Elias, Bryden, & Bulman-Fleming, 1998) (Appendix C). This questionnaire is
designed to assess the limb preference for two types of tasks: The first half of the questionnaire assessed which limb is preferred for manipulating an object including kicking a ball, picking up a marble, etc. The other half assessed which limb is preferred for providing support during activities including standing on one foot, balancing on a stool etc. The scoring system was adapted from Grouios et al. (2009): Responses of (i) left-always, (ii) left-usually, (iii) equal, (iv) right-usually, and (v) right always were scored on a scale from +2 to 2 giving a range of scores from +20 (left limb is the most preferred) to -20 (right limb is the most preferred). Participants who received scores of -7 to -20 were considered left-footed, those with scores between -6 and +6 were considered mixed-footed, and those with scores from +7 to +20 were considered right-footed (Grouios et al., 2009). If a participant was mixed-footed, the foot that was most frequently answered for the questionnaires of the latter half was considered a non-dominant limb.

2.3.3 Motion capture system

The three-dimensional (3D) kinematics of the hips and lower limbs were recorded using a commercial motion capture system (Vicon Nexus, Vicon Motion Systems, CO). The motion capture system consists of eight specialized high speed video cameras that can track and resolve the 3D coordinates of reflective markers attached to the body. Small spherical markers of 10 and 14 mm in diameter were attached to the body. Some of the markers were attached to clinical plastic platforms which were adhered to the body. The specific location of the body for the marker setup is described in detail in section 2.4.3. Motion data were collected at a sampling rate of 100 Hz.
2.3.4 **Force platform**

Two force platforms (OR6, AMTI, MA) measured the 3D ground reaction forces on each foot while the participants performed SLMS. The force platforms were synchronized with the 3D motion capture system. The force data enabled the determination of joint moments through the calculation of inverse dynamics. The sampling rate for both force platforms was set at 2000 Hz.

2.3.5 **Surface electromyography**

A surface electromyography (EMG) system (2400GT2, Noraxon Inc., AZ) was used to record participants’ muscle activation patterns. The electrical signals detected by the electrodes (Vermed® A10043) were transmitted wirelessly to an analog output receiver via a small battery-powered amplifier and transmitter attached to a belt worn by the participants. This wireless setting allowed free movement of participants and eliminated any risk of tripping. The analog output receiver was connected to the main data collection computer and synchronized with the other data collection instruments. EMG was collected at a sampling rate of 2000 Hz.

2.3.6 **High-speed video**

A high-speed digital video camera (A602fc, Basler Vision Tech., Germany), synchronized with the rest of the data collection system, recorded the SLMS movements of participants. Video from this camera was recorded at 100Hz.
2.3.7 Isokinetic dynamometer

An isokinetic dynamometer (HUMAC NORM, CSMi, Stoughton, MA) was used to measure participants’ hip and knee strength. The isokinetic dynamometer allows participants to apply maximal muscular force to the device at all points throughout the joint range of motion by leading the limbs to move at a constant velocity (Baltzopoulos & Brodie, 1989).

2.4 Procedures

2.4.1 Participant preparation

All participants were required to read and sign an informed consent form approved by the University of Saskatchewan Research Ethics Board before participating. Following the informed consent process, participants were asked questions about their current pain or injuries, and completed a visual analogue scale of knee pain (Appendix B) to confirm the eligibility and safety of the participation. Leg dominance was also identified using a Modified Waterloo Footedness Questionnaire (Appendix C), because it may affect neuromuscular response of the hip and knee to the stability demands (Brophy et al., 2010). Each participant wore athletic shorts, a short sleeved shirt and removed their shoes and socks. Anthropometric data (age, mass, and height) was measured and recorded. After being familiarized to the lab, electrodes and markers for EMG and motion capture were placed on the participant.

2.4.2 Surface EMG preparation

EMG electrodes were placed bilaterally on the gluteus medius (GMED), adductor longus, biceps femoris, and vastus medialis. It was important to monitor the activation pattern of adductor longus, because it is an antagonist of GMED which was the primary EMG site.
Moreover, it was reported in previous research that patients with PFP displayed earlier onset of adductor longus activity compared to healthy participants during stair ascent (Aminaka et al., 2011). An important potential influence of biceps femoris and vastus medialis activation on frontal plane knee movement mechanics was also suggested previously by Palmieri-Smith, Wojtys, and Ashton-Miller (2008). The skin surface at each EMG electrode site was shaved and cleaned with a medical grade alcohol swab (70%v/v isopropyl alcohol) to enhance the quality of the signal, and to minimize discomfort when removing the electrode. The electrodes were placed parallel to the muscle fibre orientation along the line of action of the muscle, and the distance between the electrodes was approximately 20mm. A female research assistant did the shaving and electrode placement in a closed laboratory for the privacy of the participants. To insure reliability of the EMG data, the sensor locations of each muscle were determined following published guidelines [(Hermens, Freriks, Desselhorst-Klug, & Rau, 2002), (Cram (2011)] with the participants in a standing position.

2.4.3 Motion capture preparation

After the EMG electrodes were attached, the motion capture markers were placed on the participants. A total of 62 markers were used during calibration, 10 of which were used for calibration only (medial and lateral femoral epicondyles, medial and lateral maleolli, and the distal end of the second metatarsals) and removed for data collection. Upper body markers included shoulder, upper arm, elbow, forearm, wrist, and a marker on the C7 vertebrae. For the head marker set, participants wore a head band on which three head markers were placed. The head and upper body markers were used for visualization only. A rectangular plastic platform
with clusters of four markers at each corner was attached to the posterior pelvis. Markers were also placed on anterior superior iliac spine (ASIS), posterior superior iliac spine (PSIS). Rectangular plastic platforms with clusters of four markers were attached to the lateral aspects of the thighs and shanks. Clusters of three markers were directly adhered to the skin in a triangular shape over the dorsal aspect of 3\textsuperscript{rd}, 4\textsuperscript{th}, and 5\textsuperscript{th} metatarsals of each foot. Markers were also placed on the heel at the base of the Achilles tendon. A double-sided non-allergenic wig tape was used to attach the markers and the plastic platforms on the skin. The plastic platforms were also secured by fabric straps. Detailed illustrations of the marker set-up are provided in Appendix G.

2.4.4 Equipment verification

After all the electrodes and the reflective markers were placed, the participants were asked to perform hip abduction, adduction, knee extension, and flexion against the student researcher’s resistance while the EMG signals from each channel were visually monitored. The above mentioned movements were performed in a standing position with the student researcher’s resistance applied on the distal femur for hip abduction and adduction, and on the ankle for knee flexion and extension. Standing position was used to verify the EMG signal in an extended hip posture (Delp, Hess, Hungerford, & Jones, 1999). To ensure that the participants maintained their standing balance and stabilized uninvolved body segments, they were asked to stand against the wall sideways and grab a research assistant’s shoulders. This ensured good quality EMG signals and identified and minimized cross talk between the channels. Adjustments were made when needed.
2.4.5 **Static posture calibration**

After the equipment verification was completed, the participant was asked to stand quietly for a two to three seconds in the centre of the data collection area for a static posture calibration trial. Data from the static posture trial was used to calibrate the marker-tracking algorithm in the motion capture system and to obtain reference data for the participant’s neutral posture. The neutral posture required that the participant stand on a wooden platform equipped with a heel ridge that allowed the feet to be 20cm apart on the same line. The wooden platform was not placed on the force platform, and the weight of the participant did not affect the data for the static posture calibration. Markers on the wooden platform were used to identify the heel alignment line. The arms were slightly apart from the body in an anatomical posture.

2.4.6 **Quiet standing EMG recording**

After the static posture calibration, the participant was asked to stand quietly on the floor for one minute while the EMG signals were recorded. The participant was asked to maintain a similar static posture as in the static posture calibration, except her arms were hanging relaxed. The quiet standing EMG signals of each channel were used to identify baseline noise level and to calculate the muscular activation onset threshold. Calculation of the onset threshold is explained more in detail in section 2.7.

2.4.7 **Joint centre calibration**

Following the quiet standing EMG recording, dynamic functional movement data were captured to estimate the hip and knee joint centres. The joint centre calibration using functional movement data is a well established method to minimize the errors of estimating the centre of rotation in the joints (Leardini et al., 1999) and more appropriate for determining participant-specific locations of the joint centre than using anthropometry-based prediction equations.
 Movements performed by the participant for the hip calibration were ~60 degrees of flexion and ~40 degrees of extension followed by ~30 degrees of abduction and ~50 degrees of adduction. For the knee calibration ~80 degrees of flexion was used. The aforementioned ROM was approximation only, and was also dependent of the flexibility of the participant. The functional calibration method is explained more detail in section 2.5.1.1

2.4.8 Movement (Single Leg Mini Squat) data collection

After the functional calibration, the participant performed Single Leg Mini Squat (SLMS) while the movement data were captured by the 3D motion capture system, the force platforms, and the EMG. The SLMS, in this protocol, was broken down into five sequential phases: 1) double leg standing at the start; 2) single leg standing; 3) descending movement; 4) ascending movement and; 5) double leg standing at the end. In order to standardize the pace of the movement, the participant was required to perform each phase of the movement following a metronome at 80 beats per minute (BPM). At the first beat, the participant lifted her toe off the force platform to make the transition from double to sing leg stance. At the next beat she descended her body to the lowest position of the SLMS. At the third beat, she ascended her body, and put her foot down at the fourth beat. 80 BPM was selected because it was identified, during pilot testing, as a rhythm that resembles a brisk but comfortable pace of stepping up and down the stairs.

The starting double leg position required the participant to place her feet separately on each force platform with feet hip width apart. She was also instructed not to lean on one side during
double leg standing at the start. She was also instructed to go into single leg standing by flexing the non-supporting knee without flexing the non-supporting hip. Flexion angle of the non-supporting side was not controlled directly but it was emphasized that the non-supporting side foot should not touch the force platform before completing the entire SLMS. Following an established clinical standard of performing SLMS (Bremander, Dahl, & Roos, 2007), the participant was required to flex her knee about 50 degrees during the descending movement. The SLMS was demonstrated to the participant so that she understood how deep she needed to go down. However, flexing the knee exactly 50 degrees was not the purpose during the data collection. Lest the participant should use arm swings for the balance, she was also asked to cross her arms on her chest during the whole movement phases.

A total of 32 SLMS trials were performed (16 trials on each leg). The order of the supporting leg was selected randomly with the maximum number of consecutive trial on the same leg being limited to four repetitions. The participant was asked to start each SLMS trial from the double leg standing position, and was also instructed to walk off the force platforms after returning to the double leg standing phase at the end of each trial. This was a necessary step to collect the baseline noise level in the force platforms. The participant was given instructions for the SLMS and allowed to practice the movement at least five times for familiarization before starting. If the participant needed more than ten practices, at least one minute of rest was given after the ten practices in order to prevent fatigue. She was also allowed to rest between trials to prevent fatigue.
2.4.9 **Strength measurement**

The participant’s hip and knee strength was measured after at least five minutes of rest after the movement data collection. Following the five minutes of rest, the participant performed dynamic stretching (two sets of 20 repetitions of hip and knee swing) lead by a student researcher to recover the range of motion (ROM) at the hip and knee. The strength tests included concentric and eccentric hip abduction and adduction and knee extension and flexion. The angular velocity of the testing was set at 90°/sec for concentric knee contraction, at 75°/sec for concentric hip contraction, and at 30°/sec for eccentric contraction of both hip and knee. These velocities were chosen to provide appropriate resistance to the testing motions as verified during pilot testing.

Following a protocol used by de Marche Baldon et al. (2009), isokinetic hip abduction and adduction strength was measured in a side-lying position with both participants’ hip and knee extended. The axis of the dynamometer lever arm was aligned at the posterior aspect of the hip joint (just superior and medial to the greater trochanter), and the lever arm pad was strapped to the lower third of the thigh on the medial and lateral aspects of the testing leg. To ensure stability, additional straps were placed around the distal thigh of the non-testing leg and around the iliac crest of the testing limb side. The set-up for isokinetic dynamometer for hip strength testing is illustrated in Appendix H. Hip adduction/abduction ROM will was set from 0° hip abduction to 30° hip abduction. However, if the participant had to tilt her pelvis to reach 30° hip abduction, maximum hip abduction just before the pelvic tilt was set as the hip abduction ROM. The ROM was measured by passively adducting / abducting the participant’s hip while monitoring a goniometer. Strength can be over-estimated when the limb goes downward (e.g., hip adduction and knee flexion), and under-estimated when the limb goes upward (e.g., hip abduction and knee flexion).
extension) because of gravity. A built-in gravity correction function in the dynamometer mathematically cancels out the effect of gravity and permits reliable strength measurements. Static torque measurement at 30° of hip abduction (or maximum hip abduction before the pelvic tilt) was used for gravity correction.

Isokinetic knee extension and flexion strength was measured in a seated position with the lever arm axis aligned at the lateral femoral epicondyle of the tested knee. Velcro straps were applied across each shoulder and over the tested leg, and the lever arm pad was strapped just above the ankle malleoli. The isokinetic dynamometry set-up for knee strength testing is illustrated in Appendix H. Knee extension/flexion ROM was set from 10° flexion to 90° flexion. Gravity correction torque was measured at 10° of knee flexion.

Before the start of testing, the participant was familiarized with the movement by repeating the movement four times consecutively at a sub-maximal intensity. Actual testing began after she felt sufficiently able to perform maximal contractions and the researchers were sure that the participant could safely and successfully exert maximal effort (i.e., axis of rotation properly aligned with the joint throughout the ROM). For the actual testing, the participant was instructed to perform the full ROM as fast and as hard as possible for three successive unilateral repetitions on both limbs. A minimum of two minutes of rest was provided between strength testing for each movement.


2.5 Data processing and analysis

The data processing was accomplished by using Visual 3D (C-Motion, Inc., Kingston, ON) for kinematics and kinetic analysis and custom Matlab (R2006b for PC, The Mathworks, MA) routines for EMG analysis.

2.5.1 Kinematics

Kinematic data analysis was comprised of three sequential steps. First, joint centre location was identified at the hip, knee and ankle. Second, the anatomical orthogonal coordinate systems for the pelvis, the femur, the tibia and the foot were defined. Finally, planar joint motions were described by the Cardan sequences (G. Wu et al., 2002) where z is the positive vertical axis and y is the positive anterior direction.

2.5.1.1 Step 1 – Locating the joint centres

The hip joint centre was identified using the functional hip calibration data following the method described by Cappello, Cappozzo, La Palombara, Lucchetti, and Leardini (1997). As the participant was moving her femur relative to her pelvis, the global trajectories of markers located on both pelvis and femur cluster were reconstructed so that the instantaneous positions of the femur markers were represented in the pelvic anatomical frame. Then the transformed trajectories of the femur markers were fed to a mathematical algorithm which estimated the centre of rotation.

As for the identification of the knee joint centre, both static and functional calibration data were used. Using the static calibration data, the midpoint between the lateral and medial femoral
epicondyle markers was calculated to provide an initial approximation of the knee joint centre. From the flexion and extension (F/E) movement performed in the dynamic calibration session, a mean F/E axis was estimated following the method described by Cappello et al. (1997). The initial approximation of the knee joint centre was then perpendicularly projected on the mean F/E axis (Hagemeister et al., 2005).

The ankle joint centre was calculated by taking a midpoint between the lateral and medial malleoli using data captured during the static calibration session (G. Wu et al., 2002).

2.5.1.2 Step 2 – Anatomical coordinates

The joint coordinate system was created using the static calibration data. Defining the joint coordinate system was important, because the joint angle and moment in the present study represented the orientation of a target segment relative to the local coordinate system.

2.5.1.3 Step 3 – Analysis of planar motions

Marker trajectories were low-pass filtered using a 4th order Butterworth digital filter at a cutoff frequency of 8 Hz. Using the Cardan sequence method (Davis, Tyburski, & Gage, 1991; Grood & Suntay, 1983) 3D hip and knee angles were calculated for each participant. The joint angle was defined as the orientation of a target segment relative to the local coordinate system. Following a standard proposed by Cole, Nigg, Ronsky, and Yeadon (1993), x-y-z cardan sequence was used, where x was the flexion and extension (F/E) axis, y was the abduction/adduction (ABD/ADD) axis, and z was the internal rotation/external rotation (IR/ER)
axis. Knee flexion was seen as a negative angle. Positive rotation about y- and z-axis always represented ADD and IR regardless of the limbs.

Frontal plane knee angle calculated by the 3D method was not considered reliable due to cross-talk between different planes; therefore, the frontal plane knee angle was calculated using a 2D method. The 2D angle was calculated between the line connecting the HJC and KJC and the line joining the KJC and AJC. As shown in the Fig 2.1, a decrease of the 2D angle represented increased knee valgus. The 2D knee angles were calculated up to the 2\textsuperscript{nd} movement phase (single limb stabilization), and calculation of the 2D knee angles for the 3\textsuperscript{rd} movement phase is ongoing. A detailed explanation of the movement phases is provided in section 2.6.

![Figure 2.1: An example of increased knee valgus](image)

The focus of analysis was on describing frontal plane knee, hip, and ankle angles on the supporting limb. The sagittal plane knee angle of the supporting limb was mainly used for defining movement phases and providing a normalizing factor for frontal plane knee moment (Myer, Ford, Khoury, Succop, & Hewett, 2010).

In order to quantify pelvic obliquity, the two-dimensional (2D) angle between the z-axis of the laboratory coordinate system and the line connecting the origin of the hip coordinate system and midway between the left ASIS and PSIS was calculated. Because the rotation of the pelvis on the transverse plane was relatively trivial during the SLMS, the 2D angle reasonably
described the rotation of the pelvis on the frontal plane. 3D analysis of the pelvis motion was not possible because the marker setup in this study was not appropriate to create a reference coordinate system of a segment that was proximally linked to the pelvis (i.e., lower trunk segment). An increase of the 2D pelvis angle represented pelvic obliquity (Fig 2.2), whereas a decreased angle represented pelvic drop in both of the limbs.

![Figure 2.2: An example of a pelvic obliquity](image)

To express the data as dynamic angle change relative to the static posture, both 3D and 2D joint angles obtained from the static calibration data were subtracted from the angle values during the movement trials.

### 2.5.2 Kinetics

The kinetic analysis focused on calculating joint moments.

#### 2.5.2.1 Joint moments

Inverse dynamics were used to calculate net internal moments of the hip, knee, and ankle on the supporting limb. Ground reaction force (GRF) data were low-pass filtered with a 4th order Butterworth digital filter at a cutoff frequency of 250 Hz. The joint moment calculated by the
inverse dynamics process was the resultant moment created by the combination of forces from the muscle-tendon units and soft tissues (i.e., ligaments, articulation cartilage) around the joint.

Each body segment was considered to be under the unique influences of muscular, reaction, and gravitational forces that were specific to individual segments. The force and moments acting on a segment are shown in Figure 2.3.

A series of equations (Equation 2.1) derived from Newton’s laws were used to calculate the net moment acting at proximal joint (Mp). The equations were applied on a segment-by-segment basis starting from the most distal segment (i.e. the foot).

\[
\sum M_{COM} = I \alpha \\
\sum M_{COM} = M_p + M_d + M_{Fp} + M_{Fd} \\
M_p = I \alpha - (M_d + M_{Fp} + M_{Fd})
\]

Equation 2.1: Joint moment calculation
The principle moments of inertia (I), the segment mass (m), and the location of the COM of each segment were obtained from the anthropometric tables provided by De Leva (1996). The centre of pressure (COP) under the foot was used to determine the point that the GRF vector was applied. The location of the averaged COP was first calculated using the force platform coordinate system, and then transformed into the kinematic coordinate system in order to calculate the distance between the averaged COP and the COM of each body segment estimated using anthropometric tables (De Leva, 1996).

Applying Newton’s third law of motion, the proximal force and moment from a segment are equal and opposite to the distal force and moment of the next segment; therefore; the Mp of the foot was used to find Md of the shank using the same series of equations used to calculate the Mp of the foot. Then the inverse dynamics process was repeated until finding the net joint moment (Mp) of a target segment. The joint moment values were normalized to each participant’s mass and height.

2.6 Identification of the movement phases

Fig 2.4 illustrates the method used for identifying the movement events for defining functionally distinctive movement phases. Pictures of these events are laid out sequentially in the order they appear in Fig 2.5.

Using the force platform outputs, toe-off timing (TO) and toe-down timing of the non-supporting limb were identified. A mathematical algorithm was used to find the first frame when the vertical (z-axis) forces of the force platform under the non-supporting limb became near zero, and was defined as TO: First, using the kinematic data, the time window in which the foot was
obviously in the air was detected. Then the mean plus two standard deviations (SDs) of the vertical forces was set as an air-time threshold. Finally TO was defined as the point when the vertical forces dropped below the air-time threshold, toe-down timing as the point when the increase of the vertical forces crossed the air-time threshold.

A pelvic centre marker in the centre of the bilateral ASIS and PSIS was used for detecting when a participant initiated descending movement, because its displacement was not substantially affected the anterior-posterior pelvic tilt or pelvic obliquity. After TO, the participant needed time to stabilize herself on one leg before initiating the descent. This is reflected in the Fig 2.4 where the vertical (z-axis in the lab coordinate system) velocity of the pelvic centre marker remains near zero before it becomes negative. Body descent was determined when the velocity became negative. When the vertical displacement of the pelvic centre marker was at the highest position, it was defined as completion of the ascent movement before toe-down.

The lowest position of the SLMS movement was defined when the participant reached maximum knee flexion angle. As shown in the Fig 2.4, the pattern of the knee flexion angle was similar to the vertical displacement of the pelvic centre marker, and the maximum knee flexion coincided with lowest position of the pelvic centre marker.

The movement occurring from double limb standing position to TO was defined as Phase I: Double to single limb transition phase. The movement occurring between TO and the initiation of body descent was defined as Phase II: Single limb stabilization phase. Phase III included the descending movement occurring from the initial descent to maximum knee flexion. The data were analyzed up to phase III, because frontal plane knee movement during the descending
movement of the SLMS is most often observed during the clinical assessment of knee pain (Ageberg et al., 2010; Boling et al., 2006).
2.7 Electromyography (EMG)

EMG data were processed with a customized Matlab routine using a 4th order Butterworth filter. The raw EMG signal was first high-pass filtered at 10Hz to remove the DC offset. The high-pass filtered EMG signal was then full-wave rectified, making the entire signal positive. Finally the full-wave rectified signal was low-pass filtered at 50Hz (Bolga, Malone, Umberger, & Uhl, 2010; Cowan, Bennell, & Hodges, 2000). Since the EMG system was synchronized with the force platforms and the motion capture system, important muscle activity could be associated with specific movement phases.

The onset of muscle activation was detected using a threshold value calculated from the one minute of quiet standing. 1.5 times of the mean amplitude of the processed EMG during the middle 40 seconds of the quiet standing trial was defined as the threshold level. Then the onset timing was found when the movement trial EMG level exceeded the threshold level for at least 65 msec. Offset timing was also found when the EMG level dropped below the threshold level for at least 65 msec. EMG onset timing of gluteus medius (GMED) was expressed relative to TO of the non-supporting limb: Positive timing represented GMED onset prior to TO.

The magnitude of EMG activity was calculated by integrating the area under the processed EMG signal (IEMG). The largest peak burst of EMG that occurred among the entire 16 trials was used for calculating a normalization factor (Liu et al., 2006). All EMG magnitude values were normalized as a percentage of the IEMG obtained over 30 msec before and after the peak burst (total 60 msec window around the peak burst). The normalized IEMGs were calculated over five different time windows. TO was chosen as it represented the time when the forces for inducing a pelvic drop were expected to start taking a substantial effect. Thus, theoretically, the
GMED must be already activated before TO in order to prevent the pelvic drop. It was reported that an electromechanical delay between the GMED onset and the onset of the abduction force is $53 \pm 6$ msec for healthy young female adults (Kim et al., 2011). It was also reported that an onset of a postural control muscle occurred approximately 60 msec prior to an anticipated perturbation of double-to-single leg transition during comfortable-pace walking (Hirschfeld & Forssberg, 1991). For these reasons, a normalized IEMG of the GMED activity over 60 msec prior to TO represented a magnitude of the proactive GMED activation. The normalized IEMG values were also calculated separately over the entire period of the single limb stabilization and descending movement phase. These IEMGs represented the total magnitude of muscle activities during each phase. If an activity pattern of a muscle needs to be changed for serving the different functional demands of each movement phases, the change may have to occur before proceeding to a next movement phase, because there always exists a delay between electrical activity and muscle force production even in already activated muscles (Buchanan et al., 2004). The duration of the electromechanical delay of activated GMED is unknown. Assuming that the electromechanical delay of an activated GMED would not be longer than the electromechanical delay between the onset and the force production, 60 msec prior to the commencement of each phase was defined as preparatory stage for each phase. Therefore the normalized IEMG values over 60 msec prior to each phase were also calculated to represent the total muscular activity over the preparatory stages. An example of the algorithm for EMG events is provided in Figure 2.6.
2.8 Statistical analysis

The Statistical Package for Social Science version 19.0 (SPSS Inc., Chicago, IL) was used for statistical analysis with an α level for significance set at 0.05.

The representative data of GMED activation, kinematics, and kinetics from each participant were used for the statistical analyses. Normal distribution of the data of the total 16 trials from each participant’s leg was tested using Shapiro-Wilk test, skewness, and kurtosis analyses. The mean of the normally distributed data and the median of the non-normal data were used as the representative samples.

Statistical analyses were performed separately for each movement phase. The net change calculated by subtracting a value at the start of a phase from a value at the end of the same phase was used for representing kinematics and kinetics data of the each individual phase.

A difference between the variables in each limb was first tested. Normal distribution of the total 20 samples was tested using Shapiro-Wilk test, skewness, and kurtosis analysis. To find a
significant difference between the limbs, a paired sample t-test was used for normally distributed variables, and a Wilcoxon Signed Rank test was used for non-normal variables. Regardless of the result of the comparison statistics, subsequent correlation analyses were performed separately for each limb due to the possibility that the neuromuscular response to stability demands may be different between limbs (Brophy et al., 2010). As a primary analysis, correlation statistics were used to examine if the GMED activation timing, magnitude, and hip abductor strength were significantly associated with the frontal plane knee kinematics and kinetics. The purpose of the secondary analysis was to confirm the relationships among the frontal plane hip kinematics, kinetics, pelvic obliquity, and frontal plane knee angle and moment. Based on the result of the primary and secondary analyses, a tertiary analysis was performed, when necessary, to identify a possible mechanism affecting the hip control of the frontal plane knee stability. The variables used for the tertiary analyses are explained in the result section. Spearman’s rho was used to determine the correlation coefficients, because it was not our purpose to find a linear relationship between the variables as it is well known that the discharge rate and force for single motor unit have a non-linear relationship (Yao, Fuglevand, & Enoka, 2000).

2.8.1 Analyses during Phase I: Double to single limb transition

The dependent variables for the primary and the secondary analyses were net change of the frontal plane knee angle and moment occurring from the double limb standing start position to toe-off of a non-supporting limb. The independent variables for the primary analyses were GMED onset, total activation magnitude over 60 msec prior to TO, and concentric and eccentric hip abductor strength. The independent variables for the secondary analyses were net change of the frontal plane hip moment, and hip and pelvic angle.
Specific hypotheses for the primary analyses are listed below:

a) Earlier GMED onset and greater activation magnitude are significantly correlated with smaller net increase of the knee abduction moment and knee valgus

b) Greater concentric and eccentric hip abductor strength are significantly correlated with smaller net increase of the knee abduction moment and knee valgus

2.8.2 Analyses during Phase II: Single limb stabilization

The dependent variables for the primary and the secondary analyses were net change of the frontal plane knee angle and moment occurring from the toe-off of a non-supporting limb to the initiation of the descending movement. The independent variables for the primary analyses were GMED onset, total activation magnitude over 60 msec prior to TO, total activation magnitude over the entire phase II, and concentric and eccentric hip abductor strength. The independent variables for the secondary analyses were net change of the frontal plane hip moment, and hip and pelvic angle.

Specific hypotheses for the primary analyses are listed below:

a) Earlier GMED onset, greater activation magnitude over 60 msec prior to TO, and greater activation magnitude over the entire phase II will be significantly correlated with greater net decrease of knee abduction moment and knee valgus

b) Greater concentric and eccentric hip abductor strength are significantly correlated with greater net decrease of knee abduction moment and knee valgus
2.8.3 Analyses during Phase III: Descending movement

As shown in the Fig 2.7, the direction of the knee moment changed from abduction to adduction during this phase in some of the participants. As a variable representing the knee abduction moment, the knee abduction impulse during the first 20% of the knee flexion was included in the analysis. The interval of the first 20% of knee flexion was chosen because all participants showed knee abduction moment during this time (Fig 2.8). The reason that the knee abduction impulse instead of the net knee moment change was used was that the pattern of the knee abduction moment was not unidirectional during the first 20% of knee flexion (Fig 2.8). The net change of the moment would not authentically represent the total amount of change the participants experienced during this period.

The dependent variables for the primary and the secondary analyses were knee abduction impulse during the first 20% of the knee flexion and net change of the frontal plane knee moment occurring from the initiation of the descending movement to the maximum knee flexion of a supporting limb. The independent variables for the primary analyses were total activation magnitude over 60 msec prior to the initiation of the descending movement, total activation magnitude over the first 20% of knee flexion, total activation magnitude over the entire phase III, and concentric and eccentric hip abductor strength. The independent variables for the secondary analyses were net change of the frontal plane hip moment, and hip and pelvic angle during the first 20% of knee flexion and the net change of these variables during the entire phase III.

Specific hypotheses for the primary analyses are listed below:

a) Greater activation magnitude over 60 msec prior to the initiation of the descending movement and greater activation magnitude over the first 20% of knee flexion are
significantly correlated with a smaller knee abdution impulse over the first 20% knee flexion

b) Greater activation magnitude over the entire phase III is significantly correlated with greater net decrease of the knee abdution moment during the entire phase III

c) Greater concentric and eccentric hip abductor strength are significantly correlated with smaller knee abdution impulse over the first 20% knee flexion

d) Greater concentric and eccentric hip abductor strength are significantly correlated with greater net decrease of the knee abdution moment during the entire phase III

Figure 2.7: An example of a frontal plane knee kinetics pattern that changed from knee abdution moment to abduction moment during the phase III

Figure 2.8: The knee abdution moment first increased then decreased during the first 20% of knee flexion in the phase III
3 RESULTS

3.1 Participants
A total of 20 females (22.60 ± 2.46 yrs, 1.67 ± 0.08 m, 65.33 ± 13.31 kg, and 23.24 ± 3.20 kg/m²) completed the entire data collection procedure except one who refused to have her hip abductor and adductor strength measured. Eighteen out of 20 participants were right limb dominant while the rest were left limb dominant (Waterloo Footedness score: 9.5 ± 4.9 for right limb dominant, -2 ± 7.0 for left limb dominant).

3.2 Overview of the gluteus medius activation
Figure 3.1 shows the gluteus medius (GMED) activation pattern plotted with the frontal plane kinematic and kinetic patterns of the supporting limbs. All participants showed onset of the GMED before toe-off (TO) of the non-supporting limbs. GMED remained activated until the completion of each trial.
3.3 Phase I: Double to Single Limb Transition

Fig 3.2 shows the average GMED activation, frontal plane knee, hip, and pelvis kinematics and knee and hip kinetics pattern during Phase I from a representative participant. GMED onset occurred prior to the onset of the pelvic obliquity (represented by an increase in pelvic angle in Fig 3.2) and before the hip and knee abduction moment started to increase (represented by a decrease in the moment in Fig 3.2). The GMED activation magnitude continuously increased throughout Phase I, peaking around TO. Frontal plane knee kinematics during this phase were grouped into two different patterns depending on the direction of the change occurred from double leg standing to TO: Increased knee angle shown in Fig 3.2 represents increased knee varus whereas a decrease in the knee angle represents knee valgus. In the dominant limbs, 12
participants showed increased knee valgus, while eight participants demonstrated increased knee varus. In the non-dominant limbs, eight participants showed increased knee valgus, while all others showed increased knee varus. All participants rapidly increased the knee and hip abduction moments immediately prior to TO in both limbs as shown by a decreased knee moment value in Fig 3.2. All participants showed increase of the pelvic angle, indicating pelvic obliquity. These kinematic and kinetic patterns were identified based on the representative values (mean or median) of the whole trials per each participant. It can be noted that the above mentioned patterns may not have been consistent across the trials.
3.3.1 Paired differences between dominant and non-dominant limbs

Table 3.1 shows the results of the paired t-test and Wilcoxon signed rank tests between the dominant and non-dominant limbs. As shown by the \( p \) values greater than 0.05, there were no significant differences between limbs in any of the variables.

Table 3.1: Paired differences between non-dominant and dominant limbs during Phase I

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean ± Standard deviation</th>
<th>( p )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Net change of the knee angle during Phase I ( (^\circ) )</td>
<td>0.77 ± 1.14</td>
<td>0.209</td>
</tr>
<tr>
<td>Net change of the knee moment during Phase I ( (Nm / Kg\cdot m) )</td>
<td>-0.31 ± 0.15</td>
<td>*0.526</td>
</tr>
<tr>
<td>GMED onset (sec)</td>
<td>0.33 ± 0.15</td>
<td>*0.550</td>
</tr>
<tr>
<td>GMED activation magnitude over 60 msec prior to TO</td>
<td>0.23 ± 0.11</td>
<td>0.465</td>
</tr>
<tr>
<td>Concentric hip abductor strength % body weight</td>
<td>156 ± 41.05</td>
<td>0.094</td>
</tr>
<tr>
<td>Eccentric hip abductor strength % body weight</td>
<td>178 ± 38.92</td>
<td>0.349</td>
</tr>
<tr>
<td>Net change of the pelvic angle during Phase I ( (^\circ) )</td>
<td>2.43 ± 1.78</td>
<td>0.769</td>
</tr>
<tr>
<td>Net change of the hip angle during Phase I ( (^\circ) )</td>
<td>3.35 ± 2.51</td>
<td>0.518</td>
</tr>
<tr>
<td>Net change of the hip moment during Phase I ( (Nm / Kg\cdot m) )</td>
<td>-0.44 ± 0.08</td>
<td>0.1</td>
</tr>
</tbody>
</table>

* Wilcoxon signed rank test (nonparametric statistics)

3.3.2 The relationship between GMED activation parameters, hip abductor strength and frontal plane knee stability

Although no significant differences between the limbs in any of the examined variables were found, the correlation analysis was done separately for non-dominant and dominant limbs to consider a possibility that the neuromuscular response to stability demands may be different.
between the limbs (Brophy et al., 2010). The result of the correlation analysis is shown in table 3. 2.

GMED onset was not significantly correlated with the net change of either frontal plane knee angle or moment during Phase I in the participants’ non-dominant limb: \( r_s (18) = -0.185, p = 0.435 \) for knee angle; \( r_s (18) = -0.131, p = 0.582 \) for knee moment. In the dominant limb, the GMED onset was correlated with the net change of the frontal plane knee angle \( (r_s (18) = -0.626, p = 0.003) \). Since participants showed both knee valgus and varus at TO, a negative relationship between GMED onset and the net change of the frontal plane angle can be interpreted in two different ways: Earlier GMED onset contributed to either decreased knee varus or increased knee valgus. Because relatively more participants showed knee valgus at TO on their dominant limbs (12 participants showed knee valgus), the result was interpreted that earlier GMED onset was related to increased knee valgus. The onset of the dominant limb GMED was not significantly correlated with the net change of the frontal plane knee moment \( (r_s (18) = -0.054, p = 0.821) \).

GMED activation magnitude over 60 msec prior to TO was not significantly associated with the net change of either frontal plane knee angle or moment during Phase I in the participants’ non-dominant limb: \( r_s (18) = -0.319, p = 0.171 \) for knee angle; \( r_s (18) = -0.18, p = 0.446 \) for knee moment. GMED activation magnitude over 60 msec prior to TO was also not significantly associated with the net change of either the frontal plane knee angle or moment in the participants’ dominant limb: \( r_s (18) = -0.183, p = 0.439 \) for knee angle; \( r_s (18) = -0.111, p = 0.64 \) for knee moment.

Concentric hip abduction strength of the non-dominant limb was not correlated with the net change of either the frontal plane knee angle or moment during Phase I in the participants’ non-dominant limb: \( r_s (17) = 0.29, p = 0.229 \) for knee angle; \( r_s (17) = 0.301, p = 0.21 \) for knee
moment. Concentric hip abduction strength of the dominant limb was positively correlated with the net change of the frontal plane knee angle ($r_s (17) = 0.463, p = 0.046$). Since participants showed both knee valgus and varus at TO, a positive relationship between hip abduction strength and the net change of the frontal plane angle can be interpreted in two different ways: Greater hip strength contributed to either increased knee varus or decreased knee valgus. Because relatively more participants showed knee valgus at TO on their dominant limbs (12 participants showed knee valgus), the result was interpreted that the participants with greater hip strength experienced less knee valgus. Concentric hip abduction strength of the dominant limb was not correlated with the net change of the frontal plane knee moment ($r_s (17) = 0.14, p = 0.586$).

Eccentric hip abduction strength of the non-dominant limb was not correlated with the net change of either the frontal plane knee angle or moment in the participants’ non-dominant limb: $r_s (17) = 0.259, p = 0.284$ for knee angle; $r_s (17) = 0.189, p = 0.439$ for knee moment. Eccentric hip abduction strength of the dominant limb was also not correlated with the net change of either the frontal plane knee angle or moment in the participants’ non-dominant limb: $r_s (17) = 0.377, p = 0.112$ for knee angle; $r_s (17) = 0.15, p = 0.539$ for knee moment.

Table 3.2: Relationship between GMED activation, hip strength, and knee kinematics & kinetics during Phase I

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non-dominant limbs</th>
<th>Dominant limbs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation Coefficient</td>
<td>p-value</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase I &amp; GMED onset</td>
<td>-0.185</td>
<td>0.435</td>
</tr>
<tr>
<td>Net change of the knee moment during Phase I &amp; GMED onset</td>
<td>-0.131</td>
<td>0.582</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase I &amp; GMED activation magnitude over 60 msec prior to TO</td>
<td>-0.319</td>
<td>0.171</td>
</tr>
<tr>
<td>Net change of the knee moment during Phase I &amp; GMED activation magnitude over 60 msec prior to TO</td>
<td>-0.18</td>
<td>0.446</td>
</tr>
</tbody>
</table>
### 3.3.3 The relationship between pelvic obliquity, hip kinematics and kinetics, and frontal plane knee stability

The result of the correlation analysis between knee, hip, and pelvis kinematics and kinetics is shown in table 3.3. The net change of the pelvic angle during Phase I was not significantly correlated with the net change of either the frontal plane knee angle or moment in the participants’ non-dominant limb: $r_s (18) = -0.152, p = 0.523$ for knee angle; $r_s (18) = -0.033, p = 0.317$ for knee moment. The net change of the pelvic angle in the dominant limb was also not significantly correlated with the net change of either the frontal plane knee angle or moment: $r_s (18) = -0.253, p = 0.283$ for knee angle; $r_s (18) = 0.251, p = 0.286$ for knee moment.

The net change of the hip angle was not significantly correlated with the net change of either frontal plane knee angle or moment in the participants’ non-dominant limb: $r_s (18) = 0.192, p = 0.418$ for knee angle; $r_s (18) = -0.002, p = 0.995$ for knee moment. The net change of the hip angle in the dominant limb was also not significantly correlated with the net change of either the frontal plane knee angle or moment: $r_s (18) = 0.241, p = 0.307$ for knee angle; $r_s (18) = -0.024, p = 0.92$ for knee moment.

The net change of the hip moment was not significantly correlated with the net change of either the frontal plane knee angle or moment in the participants’ non-dominant limb: $r_s (18) = -
0.206, \( p = 0.385 \) for knee angle; \( r_s (18) = 0.198, p = 0.402 \) for knee moment. The net change of the hip moment in the dominant limb was also not significantly correlated with the net change of either the frontal plane knee angle or moment: \( r_s (18) = 0.308, p = 0.186 \) for knee angle; \( r_s (18) = 0.134, p = 0.574 \) for knee moment.

Table 3.3: The relationship between knee, hip, and pelvis kinematics and kinetics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non-dominant limbs</th>
<th></th>
<th>Dominant limbs</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation Coefficient</td>
<td>( p )-value</td>
<td>Correlation Coefficient</td>
<td>( p )-value</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase I &amp; Net change of the pelvic angle during Phase I</td>
<td>-0.152</td>
<td>0.523</td>
<td>-0.253</td>
<td>0.283</td>
</tr>
<tr>
<td>Net change of the knee moment during Phase I &amp; Net change of the pelvic angle during Phase I</td>
<td>-0.033</td>
<td>0.317</td>
<td>0.251</td>
<td>0.286</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase I &amp; Net change of the hip angle during Phase I</td>
<td>0.192</td>
<td>0.418</td>
<td>0.241</td>
<td>0.307</td>
</tr>
<tr>
<td>Net change of the knee moment during Phase I &amp; Net change of the hip angle during Phase I</td>
<td>-0.002</td>
<td>0.995</td>
<td>-0.024</td>
<td>0.92</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase I &amp; Net change of the hip moment during Phase I</td>
<td>-0.206</td>
<td>0.385</td>
<td>0.308</td>
<td>0.186</td>
</tr>
<tr>
<td>Net change of the knee moment during Phase I &amp; Net change of the hip moment during Phase I</td>
<td>0.198</td>
<td>0.402</td>
<td>0.134</td>
<td>0.574</td>
</tr>
</tbody>
</table>

3.3.4 Relationship among GMED onset, activation magnitude, and hip strength

As a tertiary analysis, the relationship among the GMED activation parameters, hip abductor strength, and pelvic obliquity during Phase I was examined. The results are shown in table 3.3. Both concentric and eccentric hip abduction strength were negatively correlated with the onset of GMED in both non-dominant and dominant limbs: \( r_s (17) = -0.561, p = 0.013 \) for non-dominant limb concentric strength; \( r_s (17) = -0.639, p = 0.003 \) for non-dominant limb eccentric strength; \( r_s (17) = -0.663, p = 0.002 \) for dominant limb concentric strength; \( r_s (17) = -
0.728, \( p < 0.001 \) for dominant limb eccentric strength. These results indicate that the participants with weaker hip abductors tended to activate their GMED earlier.

GMED onset was not significantly correlated with the activation magnitude over 60 msec prior to TO in either limbs: \( r_s (18) = -0.11, p = 0.645 \) for the non-dominant limbs; \( r_s (18) = -0.119, p = 0.618 \) for the dominant limbs. Both concentric and eccentric hip abduction strength were negatively correlated with pelvic obliquity only in the non-dominant limbs: \( r_s (17) = -0.527, p = 0.021 \) for the concentric strength; \( r_s (17) = -0.582, p = 0.009 \) for the eccentric strength. The results indicate that participants with weaker hip abductors tended to show greater pelvic obliquity in their non-dominant limbs. Neither concentric nor eccentric hip abduction strength were significantly correlated with pelvic obliquity in the dominant limbs: \( r_s (17) = -0.347, p = 0.145 \) for the concentric strength; \( r_s (17) = -0.444, p = 0.057 \) for the eccentric strength.

GMED onset was not significantly associated with pelvic obliquity in the non-dominant limbs \( (r_s (18) = 0.377, p = 0.101) \); however, these variables were positively correlated in the dominant limbs \( (r_s (18) = 0.484, p = 0.031) \), indicating that the earlier GMED onset contributed to the increased pelvic obliquity during Phase I.

Figure 3. 3: The relationships among GMED activation parameters, hip strength, and pelvis kinematics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non-dominant limbs</th>
<th>Dominant limbs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation Coefficient</td>
<td>( p )-value</td>
</tr>
<tr>
<td>Concentric hip abductor strength &amp; GMED onset</td>
<td>-0.561</td>
<td>0.013*</td>
</tr>
<tr>
<td>Eccentric hip abductor strength &amp; GMED onset</td>
<td>-0.639</td>
<td>0.003*</td>
</tr>
<tr>
<td>GMED activation magnitude over 60 msec prior to TO &amp; GMED onset</td>
<td>-0.11</td>
<td>0.645</td>
</tr>
<tr>
<td>Concentric hip abductor strength &amp; Net change of the pelvic angle during Phase I</td>
<td>-0.527</td>
<td>0.021*</td>
</tr>
</tbody>
</table>
### 3.4 Phase II: Single Limb Stabilization

Fig 3.3 shows GMED activation, frontal plane knee, hip, and pelvis kinematics and knee and hip kinetics pattern during Phase II from a representative participant. There was a noticeable decrease of the GMED activation magnitude during the late stage of Phase II; however, GMED remained activated. The frontal plane knee and hip moment of all participants remained negative throughout this phase indicating a knee abduction moment. The moments also remained relatively stable throughout this phase; however, most of the participants experienced a slight decrease of the knee abduction moment. The frontal plane knee and hip angles also remained relatively stable throughout this phase. The direction of the net change in the knee and hip angle was not consistent across the participants. On the dominant limbs, 12 participants showed change towards knee valgus, while eight participants demonstrated change towards knee varus. On the non-dominant limbs, 16 participants showed change towards knee valgus, while all others showed changes towards knee varus. On the dominant limbs, 11 participants showed change towards hip adduction, while nine participants demonstrated change towards hip abduction. On the non-dominant limbs, 7 participants showed change towards hip adduction, while all others showed changes towards hip abduction. The frontal plane pelvic angle also remained relatively stable throughout this phase; however, most of the participants showed a slight increase in pelvic obliquity.
3.4.1 Paired differences between dominant and non-dominant limbs

Table 3.4 shows the results of the paired t-tests and Wilcoxon signed rank tests between the dominant and non-dominant limbs. A paired-sample t-test indicated that the participants experienced greater reduction of the knee abduction moment in the non-dominant limbs compared to their dominant limbs ($t(19) = -3.1, p = 0.006, d = 0.6$). A Wilcoxon Signed-ranks test indicated that the participants underwent greater decrease of the hip abduction moment on their dominant limbs (Mdn = 0.08) than on their non-dominant limbs (Mdn = 0.03); $z = -2.8, p =$
0.005, effect size $r = -0.44$. As shown by the $p$ values greater than 0.05, there were no significant differences between limbs in knee angle, GMED activation magnitude, pelvic angle, or hip angle.

Table 3.4: Paired differences between non-dominant and dominant limbs during Phase II

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean ± Standard deviation</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non-dominant limbs</td>
<td>Dominant limbs</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase II (˚)</td>
<td>0.18 ± 0.66</td>
<td>0.36 ± 0.74</td>
</tr>
<tr>
<td>Net change of the knee moment during Phase II (Nm / Kg·m)</td>
<td>0.49 ± 0.06</td>
<td>0.10 ± 0.07</td>
</tr>
<tr>
<td>GMED activation magnitude over Phase II</td>
<td>1.15 ± 0.54</td>
<td>1.20 ± 0.55</td>
</tr>
<tr>
<td>Net change of the pelvic angle during Phase II (˚)</td>
<td>3.35 ± 1.41</td>
<td>3.81 ± 1.24</td>
</tr>
<tr>
<td>Net change of the hip angle during Phase II (˚)</td>
<td>0.38 ± 1.44</td>
<td>-0.47 ± 1.27</td>
</tr>
<tr>
<td>Net change of the hip moment during Phase II (Nm / Kg·m)</td>
<td>0.02 ± 0.06</td>
<td>0.08 ± 0.09</td>
</tr>
</tbody>
</table>

# Wilcoxon signed rank test (nonparametric statistics); * Significant at 0.05 level

3.4.2 The relationship between GMED activation parameters, hip abductor strength and frontal plane knee stability

The results of the correlation analyses between GMED activation onset, magnitude, and hip abductor strength, and knee, hip, and pelvis kinematics and kinetics are shown in table 3.5.

GMED onset was not significantly correlated with the net change of the frontal plane knee angle or moment during Phase II in the participants’ non-dominant limb: $r_s (18) = -0.245, p = 0.307$ for knee angle; $r_s (18) = 0.045, p = 0.85$ for knee abdution moment. The GMED onset of the dominant limb was also not significantly correlated with the net change of either the frontal plane knee angle or abduction moment: $r_s (18) = -0.035, p = 0.885$ for knee angle; $r_s (18) = 0.072, p = 0.762$ for knee moment.
The GMED activation magnitude of non-dominant limbs over the entire period of Phase II was not significantly correlated with the net change of the frontal plane knee angle ($r_s (18) = 0.107, p = 0.654$). On the other hand, it was negatively correlated with the net change of knee abduction moment ($r_s (18) = -0.474, p = 0.035$), indicating that the GMED activation magnitude contributed to the reduction of the knee abduction moment in the non-dominant limb during Phase II. In the dominant limbs, the GMED activation magnitude over the entire period of Phase II was not significantly correlated with the net change of either the frontal plane knee angle or moment: $r_s (18) = -0.161, p = 0.498$ for knee angle; $r_s (18) = 0.071, p = 0.765$ for knee moment.

The GMED activation magnitude of non-dominant limbs over 60 msec prior to Phase II was not significantly associated with the net change of the frontal plane knee angle ($r_s (18) = -0.051, p = 0.83$). On the other hand, it was negatively correlated with the net change of the knee abduction moment ($r_s (18) = -0.463, p = 0.04$), indicating that the GMED activation magnitude before phase II contributed to the decrease of the knee abduction moment during Phase II. The GMED activation magnitude of the dominant limbs over 60 msec prior to Phase II was not significantly correlated with the net change of either the frontal plane knee angle or abduction moment: $r_s (18) = -0.198, p = 0.402$ for knee angle; $r_s (18) = -0.278, p = 0.235$ for knee abduction moment.

Concentric hip abductor strength of the non-dominant limb was not significantly correlated with the net change of either the frontal plane knee angle or abduction moment: $r_s (17) = 0.074, p = 0.756$ for knee angle; $r_s (17) = -0.122, p = 0.619$ for knee moment. Concentric hip abductor strength of the dominant limb was also not significantly correlated with the net change of the frontal plane knee angle or abduction moment: $r_s (17) = 0.077, p = 0.755$ for knee angle; $r_s (17) = -0.098, p = 0.69$ for knee moment.
Eccentric hip abductor strength of the non-dominant limb was not significantly correlated with the net change of either the frontal plane knee angle or abduction moment: \( r_s (17) = 0.01, p = 0.968 \) for knee angle; \( r_s (17) = -0.12, p = 0.626 \) for knee abduction moment. Eccentric hip abductor strength of the dominant limb was also not significantly correlated with the net change of the frontal plane knee angle or abduction moment: \( r_s (17) = 0.132, p = 0.591 \) for knee angle; \( r_s (17) = -0.195, p = 0.423 \) for knee abduction moment.
### Table 3.5: Relationship between GMED activation, hip strength, and knee kinematics & kinetics during Phase II

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non-dominant limbs</th>
<th>Dominant limbs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation Coefficient</td>
<td>p-value</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase II &amp; GMED onset</td>
<td>-0.245</td>
<td>0.307</td>
</tr>
<tr>
<td>Net change of the knee abduction moment during Phase II &amp; GMED onset</td>
<td>0.045</td>
<td>0.85</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase II &amp; GMED activation magnitude over 60msec before Phase II</td>
<td>-0.051</td>
<td>0.83</td>
</tr>
<tr>
<td>Net change of the knee abduction moment during Phase II &amp; GMED activation magnitude over 60msec before Phase II</td>
<td>-0.463</td>
<td><strong>0.04</strong>*</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase II &amp; GMED activation magnitude over the entire phase II</td>
<td>0.107</td>
<td>0.654</td>
</tr>
<tr>
<td>Net change of the knee abduction moment during Phase II &amp; GMED activation magnitude over the entire phase II</td>
<td>-0.474</td>
<td><strong>0.035</strong>*</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase II &amp; Concentric hip abductor strength</td>
<td>0.074</td>
<td>0.756</td>
</tr>
<tr>
<td>Net change of the knee abduction moment during Phase II &amp; Concentric hip abductor strength</td>
<td>-0.122</td>
<td>0.619</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase II &amp; Eccentric hip abductor strength</td>
<td>0.01</td>
<td>0.968</td>
</tr>
<tr>
<td>Net change of the knee abduction moment during Phase II &amp; Eccentric hip abductor strength</td>
<td>-0.12</td>
<td>0.626</td>
</tr>
</tbody>
</table>

* Significant at 0.05 level movement  

#### 3.4.3 The relationship between knee, hip, and pelvis kinematics and kinetics

The results of the correlation analyses between knee, hip, and pelvis kinematics and kinetics are shown in table 3.6. The net change of the pelvic angle during Phase II was not significantly correlated with the net change of the frontal plane knee angle in the participants’ non-dominant limb ($r_s (18) = 0.019, p = 0.935$). It was also not significantly associated with the net change of the knee abduction moment, however, the $p$ value was very close to being statistically significant.
(r_s (18) = -0.442, p = 0.05). The net change of the pelvic angle in the dominant limb was not significantly correlated with the net change of the knee abduction moment (r_s (18) = -0.119, p = 0.618). On the other hand, it was negatively correlated with the net change of knee abduction moment (r_s (18) = -0.48, p = 0.032), indicating that the increased pelvic obliquity of the dominant limb contributed to the reduction of the knee abduction moment during Phase II.

The net change of the hip angle was not significantly correlated with the net change of either the frontal plane knee angle or abduction moment in the participants’ non-dominant limb: r_s (18) = 0.242, p = 0.304 for knee angle; r_s (18) = 0.195, p = 0.409 for knee abduction moment. The net change of the hip angle of the dominant limb was also not significantly correlated with the net change of either the frontal plane knee angle or abduction moment: r_s (18) = -0.303, p = 0.194 for knee angle; r_s (18) = 0.324, p = 0.164 for knee abduction moment.

The net change of the hip abduction moment was not significantly correlated with the net change of the frontal plane knee angle in the participants’ non-dominant limb (r_s (18) = -0.084, p = 0.724 for knee angle). On the other hand, it was positively correlated with the net change of the knee abduction moment (r_s (18) = 0.566, p = 0.009). The net change of the hip abduction moment of the dominant limb was not significantly associated with the net change of the frontal plane knee angle (r_s (18) = 0.075, p = 0.753). However, it was positively correlated with the net change of knee abduction moment (r_s (18) = 0.618, p = 0.004). These results indicate that the increased hip abduction moment was related to the increased knee abduction moment in both of the limbs during Phase II.
Table 3.6: The relationship between knee, hip, and pelvis kinematics and kinetics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non-dominant limbs</th>
<th>Dominant limbs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation</td>
<td>p-value</td>
</tr>
<tr>
<td></td>
<td>Coefficient</td>
<td></td>
</tr>
<tr>
<td>Net change of the knee angle during Phase II &amp; Net change of the pelvic angle during Phase II</td>
<td>0.019</td>
<td>0.935</td>
</tr>
<tr>
<td>Net change of the knee abduction moment during Phase II &amp; Net change of the pelvic angle during Phase II</td>
<td>-0.442</td>
<td>0.05</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase II &amp; Net change of the hip angle during Phase II</td>
<td>0.242</td>
<td>0.304</td>
</tr>
<tr>
<td>Net change of the knee abduction moment during Phase II &amp; Net change of the hip angle during Phase II</td>
<td>0.195</td>
<td>0.409</td>
</tr>
<tr>
<td>Net change of the knee angle during Phase II &amp; Net change of the hip abduction moment during Phase II</td>
<td>0.084</td>
<td>0.724</td>
</tr>
<tr>
<td>Net change of the knee abduction moment during Phase II &amp; Net change of the hip abduction moment during Phase II</td>
<td>0.566</td>
<td>0.009*</td>
</tr>
</tbody>
</table>

* Significant at 0.05 level

3.4.4 Relationship between GMED activation, pelvic obliquity, and hip moment

In order to identify a possible mechanism affecting the relationship between GMED activation magnitude and knee moment, correlations among GMED activation magnitude, pelvic obliquity, and hip abduction moment during Phase II were examined. The results are shown in table 3.7. GMED activation magnitude over 60msec before Phase II was not significantly correlated with the net change of hip abduction moment during Phase II in either limbs: $r_s (18) = -0.343, p = 0.139$ for non-dominant limbs; $r_s (18) = 0.262, p = 0.265$. GMED activation magnitude over the entire phase II was also not significantly correlated with the net change of hip abduction moment during Phase II in either limbs: $r_s (18) = -0.287, p = 0.22$ for non-dominant limbs; $r_s (18) = 0.146, p = 0.539$. 
GMED activation magnitude over 60msec before Phase II was positively correlated with the net change of the pelvic angle during Phase II in the non-dominant limb ($r_s (18) = 0.462, p = 0.04$); however, these variables were not significantly correlated in the dominant limbs ($r_s (18) = -0.099, p = 0.677$). GMED activation magnitude over the entire phase II was also positively correlated with the net change of the pelvic angle during Phase II in the non-dominant limb ($r_s (18) = 0.614, p = 0.004$); however, these variables were not significantly correlated in the dominant limbs ($r_s (18) = 0.179, p = 0.45$). These results imply that an increase of the pelvic obliquity in the non-dominant limb during Phase II was related to not only a greater GMED activation magnitude during Phase II but also a greater GMED activation magnitude over 60 msec before Phase II.

Table 3.7: Correlations among GMED activation, hip kinetics, and pelvis kinematics during Phase II

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non-dominant limbs</th>
<th>Dominant limbs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation Coefficient</td>
<td>p-value</td>
</tr>
<tr>
<td>GMED activation magnitude over 60msec before Phase II &amp; Net change of the hip abduction moment during Phase II</td>
<td>0.343</td>
<td>0.139</td>
</tr>
<tr>
<td>GMED activation magnitude over the entire phase II &amp; Net change of the hip abduction moment during Phase II</td>
<td>-0.287</td>
<td>0.22</td>
</tr>
<tr>
<td>GMED activation magnitude over 60msec before Phase II &amp; Net change of the pelvic angle during Phase II</td>
<td>0.462</td>
<td>0.04*</td>
</tr>
<tr>
<td>GMED activation magnitude over the entire phase II &amp; Net change of the pelvic angle during Phase II</td>
<td>0.614</td>
<td>0.004*</td>
</tr>
</tbody>
</table>

* Significant at 0.05 level

3.5 Phase III: Descending movement

Fig 3.4 shows GMED activation, frontal plane hip and pelvic kinematics, and knee and hip kinetics patterns during Phase III from a representative participant. GMED activation magnitude
remained relatively low during the early stage of this phase with a gradual increase throughout including momentary fluctuations in the activity level. The frontal plane knee abduction moment also fluctuated, but in general, the knee abduction moment decreased throughout this phase. The direction of the knee moment changed from abduction to adduction moment during this phase in many (11 non-dominant limbs and 12 dominant limbs) of the participants (Fig 3.5). All participants showed a knee abduction moment during the first 20% of knee flexion. The hip abduction moment generally increased during Phase III; however, as it is shown in Fig 3.4, there was a noticeable decrease of the hip abduction moment during the early stage of this phase. The hip adduction angle continuously increased throughout this phase, whereas the pelvic obliquity gradually decreased.

Figure 3.5: Gluteus medius activation, frontal plane kinematics and kinematics pattern of supporting limb during Phase III. Data is from a representative participant.

Figure 3.6: An example of a frontal plane knee kinetics pattern that changed from knee abduction moment to adduction moment during Phase III.
3.5.1 *The relationship between maximum knee flexion angle and knee abduction moment*

There was noticeable variability of the maximum knee flexion angle between the participants, with a range of 60 to 70.44 degrees in the non-dominant limbs, and 60.81 to 69.97 degrees in the dominant limbs. There is a possibility that the variability of the maximum knee flexion angle affected the net change of the knee abduction moment during Phase III (Myer et al., 2010). In order to examine if the maximum knee flexion angle between participants was a potential confounding factor affecting the primary analysis, the correlation between maximum knee flexion angle and net change of frontal plane knee angle and abduction moment during Phase III was investigated. No significant correlation was found in either limb: $r_s (18) = -0.255, p = 0.278$ for non-dominant limbs; $r_s (18) = 0.013, p = 0.956$ for dominant limbs. There was also no significant limb difference in the maximum knee flexion angle: $M = -65.39, SD = 4.58$ for non-dominant limbs; $M = -65.1, SD = 5.34$ for dominant limbs; $t (19) = -0.49, p = 0.632$.

3.5.2 *Paired differences between dominant and non-dominant limbs*

Table 3.8 shows the results of the paired t-test and Wilcoxon signed rank tests between the dominant and non-dominant limbs. The direction of the knee moment changed from abduction to adduction during this phase in some of the participants (Fig 3.5). To make sure that only the knee abduction moments were calculated, the knee moment during the first 20% of the knee flexion was also included in the analysis. As shown in the Fig 3.4, the change of knee abduction moment was not unidirectional during the first 20% of knee flexion. The net change of the moment would not authentically represent the total amount of change the participants experienced during this
period; therefore, a knee abduction impulse was calculated representing the total knee abduction moment the participants experienced during the first 20% of knee flexion.

As shown in table 5.8, there were no significant differences between limbs in GMED activation magnitude, pelvic angle, hip angle, and knee moment and impulse. A paired-samples t-test indicated that the participants experienced greater increase of the hip abduction moment in the non-dominant limbs compared to their dominant limbs ($t(19) = -2.23$, $p = 0.038$, $d = -0.47$).

Table 3.8: Paired differences between non-dominant and dominant limbs during Phase III

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean ± Standard deviation</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non-dominant limbs</td>
<td>Dominant limbs</td>
</tr>
<tr>
<td>GMED activation magnitude over the entire phase III</td>
<td>3.85 ± 1.43</td>
<td>3.65 ± 1.35</td>
</tr>
<tr>
<td>GMED activation magnitude over 60msec before Phase III</td>
<td>0.19 ± 0.10</td>
<td>0.21 ± 0.10</td>
</tr>
<tr>
<td>GMED activation magnitude over the first 20% of knee flexion</td>
<td>0.78 ± 0.46</td>
<td>0.73 ± 0.44</td>
</tr>
<tr>
<td>Net change of the knee moment during Phase III (Nm / Kg∙m)</td>
<td>0.25 ± 0.13</td>
<td>0.26 ± 0.19</td>
</tr>
<tr>
<td>Knee abduction impulse over the first 20% of knee flexion (Nm·sec / kg·m)</td>
<td>-0.029 ± 0.013</td>
<td>-0.026 ± 0.01</td>
</tr>
<tr>
<td>Net change of the pelvic angle during Phase III (˚)</td>
<td>-4.59 ± 3.57</td>
<td>-4.01 ± 3.55</td>
</tr>
<tr>
<td>Net change of the pelvic angle during the first 20% of knee flexion (˚)</td>
<td>0.18 ± 0.61</td>
<td>0.28 ± 0.6</td>
</tr>
<tr>
<td>Net change of the hip angle during Phase III (˚)</td>
<td>8.78 ± 4.12</td>
<td>7.72 ± 4.31</td>
</tr>
<tr>
<td>Net change of the hip angle during the first 20% of knee flexion (˚)</td>
<td>0.61 ± 1.04</td>
<td>1.01 ± 1.42</td>
</tr>
<tr>
<td>Net change of the hip moment during Phase III (Nm / Kg∙m)</td>
<td>-0.23 ± 0.08</td>
<td>-0.19 ± 0.09</td>
</tr>
</tbody>
</table>

* Wilcoxon signed rank test (nonparametric statistics); # Significant at 0.05 level
3.5.3 The relationship between GMED activation, hip abductor strength and frontal plane knee stability

The results of the correlation analyses between GMED activation magnitude and hip abductor strength, and knee, hip, and pelvis kinematics and kinetics are shown in table 3.5. In the non-dominant limb, the GMED activation magnitude over 60 msec before Phase III was positively correlated with the knee abduction impulse over the first 20% knee flexion ($r_s (18) = 0.451, p = 0.046$); however, these variables were not significantly correlated in the dominant limbs ($r_s (18) = 0.096, p = 0.686$). These results indicate that a lower GMED activation magnitude immediately before Phase III was related to a decreased knee abduction impulse during the first 20% of the knee flexion during Phase III only in the non-dominant limbs.

The GMED activation magnitude over the entire period of Phase III was not significantly correlated with the net change of the frontal plane knee moment in either limbs: $r_s (18) = 0.087, p = 0.715$ for non-dominant limbs; $r_s (18) = 0.116, p = 0.627$ for dominant limbs.

Concentric hip abduction strength was also not significantly correlated with the net change of the frontal plane knee moment in either limb: $r_s (18) = 0.296, p = 0.219$ for non-dominant limbs; $r_s (18) = 0.048, p = 0.846$ for dominant limbs.

Eccentric hip abduction strength was also not significantly correlated with the net change of the frontal plane knee moment in either limb: $r_s (18) = 0.301, p = 0.211$ for non-dominant limbs; $r_s (18) = -0.007, p = 0.978$ for dominant limbs.
Table 3.9: The relationship between GMED activation, hip abductor strength, and knee kinetics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non-dominant limbs</th>
<th>Dominant limbs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation Coefficient</td>
<td>p-value</td>
</tr>
<tr>
<td>Knee abduction impulse over the first 20% of knee flexion &amp;</td>
<td>0.451</td>
<td>0.046*</td>
</tr>
<tr>
<td>GMED activation magnitude over 60msec before Phase III</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee abduction impulse over the first 20% of knee flexion &amp;</td>
<td>0.639</td>
<td>0.002*</td>
</tr>
<tr>
<td>GMED activation magnitude over the first 20% of knee flexion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Net change of the knee moment during Phase III &amp; GMED activation magnitude</td>
<td>0.087</td>
<td>0.715</td>
</tr>
<tr>
<td>over the entire phase III</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Net change of the knee moment during Phase III &amp; Concentric hip abductor</td>
<td>0.296</td>
<td>0.219</td>
</tr>
<tr>
<td>strength</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Net change of the knee moment during Phase III &amp; Eccentric hip abductor</td>
<td>0.301</td>
<td>0.211</td>
</tr>
<tr>
<td>strength</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee abduction impulse over the first 20% of knee flexion &amp;</td>
<td>-0.175</td>
<td>0.475</td>
</tr>
<tr>
<td>Concentric hip abductor strength</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee abduction impulse over the first 20% of knee flexion &amp;</td>
<td>-0.103</td>
<td>0.675</td>
</tr>
<tr>
<td>Eccentric hip abductor strength</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Significant at 0.05 level

3.5.4 The relationship between pelvic obliquity, hip kinematics and kinetics, and the frontal plane knee stability

The results of the correlation analyses between knee, hip, and pelvis kinematics and kinetics are shown in table 3.10. The net change of the knee moment during Phase III was not significantly correlated with the net change of the pelvic angle during Phase III in either limbs: \( r_s (18) = 0.22, p = 0.352 \) for non-dominant limbs; \( r_s (18) = 0.145, p = 0.541 \) for dominant limbs. The net change of the knee moment during Phase III was also not significantly correlated with the net change of the hip angle during Phase III in either limbs: \( r_s (18) = 0.068, p = 0.775 \) for non-dominant limbs; \( r_s (18) = 0.016, p = 0.947 \) for dominant limbs. It was also not significantly correlated with the net change of the hip moment during Phase III in either limbs: \( r_s (18) = 0.296, p = 0.206 \) for non-dominant limbs; \( r_s (18) = 0.204, p = 0.387 \) for dominant limbs.
The knee abduction impulse over the first 20% knee flexion was not significantly associated with the net change of the pelvic angle during the first 20% knee flexion in either limbs: \( r_s(18) = -0.235, p = 0.319 \) for non-dominant limbs; \( r_s(18) = -0.32, p = 0.169 \) for dominant limbs. It was also not significantly correlated with the net change of the hip angle during the first 20% knee flexion in either limbs: \( r_s(18) = 0.227, p = 0.336 \) for non-dominant limbs; \( r_s(18) = 0.116, p = 0.627 \) for dominant limbs.

Table 3.10: The relationship between knee, hip, and pelvis kinematics and kinetics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non-dominant limbs</th>
<th>Dominant limbs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation Coefficient</td>
<td>( p )-value</td>
</tr>
<tr>
<td>Net change of the knee moment during Phase III &amp; Net change of the pelvic angle during Phase III</td>
<td>0.22</td>
<td>0.352</td>
</tr>
<tr>
<td>Knee abduction impulse over the first 20% knee flexion &amp; Net change of the pelvic angle during the first 20% knee flexion</td>
<td>-0.235</td>
<td>0.319</td>
</tr>
<tr>
<td>Net change of the knee moment during Phase III &amp; Net change of the hip angle during Phase III</td>
<td>0.068</td>
<td>0.775</td>
</tr>
<tr>
<td>Knee abduction impulse over the first 20% knee flexion &amp; Net change of the hip angle during the first 20% knee flexion</td>
<td>0.227</td>
<td>0.336</td>
</tr>
<tr>
<td>Net change of the knee moment during Phase III &amp; Net change of the hip moment during Phase III</td>
<td>0.296</td>
<td>0.206</td>
</tr>
</tbody>
</table>

### 3.6 Summary

Table 3.11 provides summarized interpretations of the results by phases and limbs. GMED activation magnitude contributed to a decrease of the knee abduction moment during Phase II only in the non-dominant limbs. During Phase II, pelvic obliquity was significantly correlated with increased GMED activation magnitude and decreased knee abduction moment in the non-dominant limbs. The non-dominant limbs experienced greater reduction of the knee abduction moment than the dominant limbs during this phase. GMED activation magnitude contributed to
an increase of the knee abduction impulse during Phase III only in the non-dominant limbs. In the dominant limbs, earlier GMED onset and weaker concentric hip abductor strength contributed to the increase of the knee valgus during Phase I.

Table 3.11: Summarized interpretations of the overall results

<table>
<thead>
<tr>
<th>Phase</th>
<th>Non-dominant limbs</th>
<th>Dominant limbs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase I</td>
<td>No significant relationship</td>
<td>Earlier GMED onset</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Increased knee valgus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Weaker concentric hip abductor strength</td>
</tr>
<tr>
<td>Phase II</td>
<td>More GMED activity</td>
<td>No significant relationship</td>
</tr>
<tr>
<td></td>
<td>More pelvic obliquity</td>
<td></td>
</tr>
<tr>
<td></td>
<td>More decrease of the knee abduction moment</td>
<td></td>
</tr>
<tr>
<td></td>
<td>The dominant limbs experienced less decrease of the knee abduction moment than the non-dominant limbs</td>
<td></td>
</tr>
<tr>
<td>Phase III</td>
<td>More GMED activity</td>
<td>No significant relationship</td>
</tr>
<tr>
<td></td>
<td>Greater knee abduction impulse</td>
<td></td>
</tr>
</tbody>
</table>

→: Significantly correlated
4 DISCUSSION

The primary purpose of this thesis was to examine how GMED activation parameters and hip abductor strength were related to the control of knee abduction moment and knee valgus during a SLMS. The secondary purpose was to clarify if hip abductor strength is related to knee abduction moment and dynamic knee valgus. The data were analyzed with consideration to the functionally unique movement phases. The relationship between GMED activation parameters and knee kinematics and kinetics was investigated within and between the SLMS movement phases. The results showed that hip strength was related to knee valgus, while GMED activation magnitude was related to the knee abduction moment. A possible mechanism affecting the relationship between GMED activity and the knee abduction moment was the frontal plane pelvic motion. It was also observed that the GMED activity during the late stage of one phase was related to the control of knee moment in the next movement phase. In addition, the significant relationship between GMED activity and the knee abduction moment was found in the non-dominant limb only, indicating a possible effect of limb dominance on the hip’s control of knee stability. These results provide useful knowledge regarding biomechanical mechanisms in which hip control influences knee stability. This information may be important for developing rehabilitation and prevention strategies for knee pain.

4.1 GMED activation parameters and knee abduction moment

4.1.1 Phase I: Double to single limb transition

Our hypothesis was that the GMED activity before TO would mitigate an increase of the knee abduction moment by proactively controlling contralateral pelvic drop. Participants began to rapidly increase the knee abduction moment immediately before TO; however, contrary to the
hypothesis, an earlier GMED onset was not protective against an increased knee abduction moment at TO. Total GMED activation magnitude over 60 msec prior to TO was also not related to the increased knee abduction moment at TO. While the GMED onset was not correlated with the knee abduction moment, earlier GMED onset was associated with increased pelvic obliquity at TO, though such relationship was observed only in the dominant limbs. While it has been consistently theorized that frontal plane pelvic motion would affect the knee abduction moment in a stance limb (Chang et al., 2005; Perry & Burnfield, 2010; Powers, 2010), no significant relationship between increased pelvic obliquity at TO and increased knee abduction moment at TO was found in our study. Overall, these results suggest that while GMED activation timing may be important for controlling frontal plane pelvic motion, it did not have an influence on the knee abduction moment during the double to single limb transition.

4.1.2 Phase II: Single limb stabilization

Increased pelvic obliquity in Phase II was significantly related to the decreased knee abduction moment in the dominant limb within the same phase. Non-dominant limbs also showed a similar relationship, though the $p$ value ($p = 0.051$) did not achieve a statistical significance. Greater total GMED activation magnitude during Phase II was related to both increased pelvic obliquity and decreased knee abduction moment in the same phase, though such relationships were observed only in the non-dominant limbs. These results imply that during single limb stabilization, the GMED activity was protective against the increase of a knee abduction moment with the help of pelvic obliquity.

The total GMED activation magnitude over 60 msec prior to TO was also related to both increased pelvic obliquity and decreased knee abduction moment during Phase II in the non-
dominant limbs. As illustrated in Fig 3.2, the pelvic obliquity started rapidly increasing immediately after GMED onset. The knee abduction moment stopped increasing directly following the onset of the pelvic obliquity (Fig 3.2 and 3.3). It is also notable that there was an increase of the GMED activity just before the knee abduction moment stopped increasing (Fig 3.2). Increased GMED activity before an increase of the pelvic obliquity and the subsequent decrease of the knee abduction moment seems reasonable because an electrical activity of a muscle must substantially increase beforehand to overcome an electromechanical delay (Buchanan et al., 2004) and successfully generate a sufficient force at a targeted time. Therefore the above mentioned sequential EMG, kinematic, and kinetic patterns indicate a possibility that an increased GMED activity before TO was part of proactive neuromuscular strategy to increase pelvic obliquity and subsequently decrease the knee abduction moment.

Preparatory modulation of lower extremity muscles for facilitating movement has been frequently reported in walking, stair descending and jump landing (Hirschfeld & Forssberg, 1991; Perry & Burnfield, 2010; Santello & McDonagh, 1998). A unique finding of our study is that the magnitude of a preparatory GMED activation at the end of the double to single limb transition of a SLMS was significantly associated with the knee abduction moment in the upcoming single limb stabilization phase. This result could help improve the quality of an EMG-based biofeedback training applied to SLMS. It was reported that a real time EMG-based feedback was an effective supplementation to physiotherapy exercises for improving PFP including SLMS and single limb step down (Yip & Ng, 2006). Currently, the major focus of the EMG-based biofeedback training for knee pain is on improving neuromuscular control of the of the quadriceps muscles (Ng, Zhang, & Li, 2008; Wise, Fiebert, & Kates, 1984). The results from
this study suggest that more attention needs to be given to improving the neuromuscular control of GMED when using biofeedback training. In particular, the relationship between GMED activation before TO and the knee abduction moment during the single limb stabilization phase could guide a therapist to focus on increasing the GMED activity before single limb stance.

4.1.3 **Phase III: Descending movement**

In the non-dominant limbs, a decreased GMED activation magnitude during the first 20% of knee flexion for Phase III was related to decreased knee abduction impulse over the same period. Similarly, decreased GMED activity 60 msec before the descending phase was also related to decreased knee abduction impulse over the first 20% knee flexion. These results seem to be contradicting what was observed in the previous movement phase. A rationale for this relationship can be found in our data of EMG, kinematic and kinetic patterns. As shown in Fig 3.4 and 3.5, the participants showed a noticeable decrease of GMED activity immediately before the descending phases and the EMG activity level remained low during the early stage of the descending phase. Hip adduction started to increase and pelvic obliquity began to decrease from the beginning to the end of the descending phase. Furthermore, the hip abduction moment decreased during the early stage of the descending phase while the GMED activity remained low. It can therefore be interpreted that the decreased GMED activity may have facilitated the increased hip adduction and the decreased pelvic obliquity by reducing the hip abduction moment. It is also shown in Fig 3.5 that despite the gradual decrease of the pelvic obliquity, the knee abduction moment decreased. This pattern may indicate that the frontal plane motion of the pelvis no longer played an important role for the control of the knee abduction moment during the descending phase. Supporting this explanation, no significant relationship between the pelvic
obliquity and the knee abduction moment was found in the descending phase. Instead, a
decreased knee abduction moment seems to be influenced by the hip adduction angle during this
phase: As hip adduction increased, the knee abduction moment generally decreased (Fig 3.5). It
is known that hip adduction in a weight bearing limb can be used as a strategy to reduce any
knee abduction moment by moving the knee joint centre closer to the GRF line of action
(Henriksen, Aaboe, Simonsen, Alkjaer, & Bliddal, 2009). Therefore the relationship between
decreased GMED activity and decreased knee abduction impulse in the descending phase may
reflect the effect of GMED moderation for assisting hip adduction necessary to control the knee
abduction moment.

Considering that a substantial increase of hip adduction and decrease of the pelvic obliquity
in the descending phase occurred after the GMED activity started dropping (Fig 3.4 and 3.5), the
decrease of the GMED activity immediately before the descending phase can be interpreted as
neuromuscular preparation for facilitating the increase of the hip adduction and the decrease of
pelvic obliquity. If the decreased GMED activity was also a necessary preparation for controlling
knee abduction moment during the descending phase, there should be a relationship between
these two variables. The significant relationship between lower GMED activation magnitude
over 60 msec before the descending phase and less knee abduction impulse during the first 20%
knee flexion in the non-dominant limbs may provide some support for this proposed mechanism.

It is necessary to further investigate whether or not the relationship between the GMED
activity and the knee abduction impulse was a coincidental finding, because the GMED activity
was not significantly related to the hip adduction angle. Data of hip adductor EMG is required to
clarify if the hip adduction was involved in the control of knee abduction moment during the descending phase.

4.2 Relative contribution of GMED activation and hip abductor strength to the knee valgus

GMED activation magnitude was not related to the frontal plane knee angle. Weaker hip abductor strength and earlier GMED onset was related to either greater knee valgus or less knee varus in the dominant limbs. Because the majority of participants showed knee valgus in their dominant limbs at TO, the result was interpreted as the correlation between earlier GMED onset and increased knee valgus. Considering that the importance of the GMED has been emphasized for its role for preventing excessive knee valgus (Heiderscheit, 2010; Powers, 2010), the relationship between earlier GMED onset and increased knee valgus seems counter intuitive. The hip abductor strength showed a more intuitive relationship with the knee kinematics: Concentric hip abductor strength of the dominant limb was associated with decreased knee valgus. The results suggest that having stronger hip abductors rather than activating the GMED earlier may be more effective for preventing dynamic knee valgus and that this relationship may be dependent on the type of movement being performed.

The results of the present study seems to contradict previous findings of a significant association between hip abductor strength and frontal plane knee alignment (Bolga et al., 2008; Hollman et al., 2009; Rutherford & Hubley-Kozey, 2009; Thijs et al., 2011; Thijs et al., 2007). Hollman et al. (2009) reported that hip abductor strength was positively related to increased knee valgus during a single limb step down movement which is functionally similar to a SLMS. The authors (Hollman et al., 2009) of the study speculated that the reason for this result may be that
the activity of the hip abductors, especially GMED, might have contributed more to internal rotation rather than abduction of the hip as the hip flexion angle increased. Partially supporting this speculation, Bolgla et al. (2008) reported that the hip abductor strength was not related to the hip abductor moment during stair descent in which hip flexion is also inevitably involved. The effect of hip flexion on the functional changes of the hip abductors have been well explained in anatomical studies (Delp et al., 1999; Neumann, 2010); however, it is not well established whether or not the functional changes of the hip abductors play an important role in the relationship between hip strength and knee valgus because this concept is contradicted by previous evidence of the association between weaker hip abductor strength and the increased knee valgus in single leg squatting (Claiborne et al., 2006; Willson, Ireland, & Davis, 2006). In our study, the relationship between hip weakness and increased knee valgus was observed only in the double to single limb transition phase where no noticeable hip flexion occurred. In order to understand if hip strength is also important during the descending phase of the SLMS, a further analysis of the relationship between hip abductor strength and knee valgus is ongoing.

4.2.1 How can earlier GMED onset contribute to the knee valgus?

A question should be asked as to how the earlier GMED onset contributed to increase of the knee valgus. An explanation for this counterintuitive relationship could be the reciprocal action of hip abductor and adductors required to facilitate a double to single limb transition. In order to accomplish the unloading of the non-supporting limb and loading of the supporting limb, the stance limb hip adductors should first generate a propulsive impulse to initiate a lateral movement of the COM (Rogers & Pai, 1993). The linear momentum of the COM is then
substantially reduced by the activity of the hip abductors to maintain postural stability (Rogers & Pai, 1993). Given this functional sequence of the hip adductor and abductor action, GMED activation that occurs too early would impose greater mechanical demands on the hip adductors because the hip adductors must then overcome a greater amount of force to initiate the COM movement. Hip adduction in a weight bearing limb creates a force that pulls the knee into valgus (Claiborne et al., 2006). This valgus force at the knee should be opposed by the hip abductors to stabilize the knee alignment. It is critical that the valgus force is opposed before TO, because the valgus alignment of the knee would impose greater stress within the knee joint in a single limb weight bearing posture compared to a double limb standing posture. Considering that the electromechanical delay of GMED for generating a hip abduction moment among female young adults has been reported to be 53 ± 6 msec (Kim et al., 2011), GMED activity over 60 msec before TO would have been critical for our participants to create the required opposing force. The results of the present study, however suggest that earlier GMED onset did not lead into an increased GMED activation over this critical time window. Taken all together, it is possible that an earlier GMED activation led to an increased knee valgus by inducing an intensified hip adductor activity that was not sufficiently balanced by the GMED activation. To examine whether or not this proposed mechanism is feasible, further analysis including the timing and magnitude of the adductor longus activation is ongoing.
4.3 **Relative contribution of GMED activation and hip abductor strength on the knee moment**

While hip abductor strength showed relatively more influence on the control of knee alignment, GMED activation magnitude showed greater influence on the knee abduction moment. Increased GMED activity contributed to decreased knee abduction moment only when there was a significant relationship between GMED activation magnitude and pelvic obliquity, and between pelvic obliquity and knee abduction moment. Considering that the hip abductors directly control pelvic obliquity, it is odd that the hip abductor strength was not related to the knee abduction moment. It can be speculated that the movement position and velocity used for measuring the hip strength was not similar enough to appropriately measure the functional capability of the hip abductors for controlling pelvic motion relative to femur: Hip abductor strength was measured while participants ab ducted their femur relative to a fixed pelvis. Further research for finding more appropriate methods for measuring functional strength of pelvic on femur motion would be necessary to improve the quality of future investigations on the relationship between hip strength and knee abduction moment.

4.4 **The relationship between pelvic obliquity and knee abduction moment**

It has been consistently theorized that a contralateral pelvic drop would increase the knee abduction moment in a stance limb (Chang et al., 2005; Perry & Burnfield, 2010; Powers, 2010). The present study attempted to test this theory but could not provide directly related evidence, because none of our participants demonstrated a pelvic drop; they showed pelvic obliquity instead. We speculate that pelvic drop may occur in specific population and during a specific
movement. According to Trendelenburg (1895), pelvic drop is a manifestation of inefficient coordination or weakness of hip abductor muscles; in a healthy population pelvic obliquity rather than pelvic drop is expected (Connolly, 2011). The fact that none of our participants had clinically diagnosed hip dysfunction may explain why no pelvic drop had occurred in our study. Occurrence of pelvic drop may also depend on specific mechanical demands induced by continuous locomotion. Researchers have suggested possible adverse effects of pelvic drop on the frontal plane knee stability in regard to continuous locomotion (e.g., walking or running) not to SLMS (Chang et al., 2005; Perry & Burnfield, 2010; Powers, 2010). The COM remains outside (medially) of the BOS during the single limb stance phase of continuous locomotion which may cause gravity-induced pelvic drop (Patla, 2003). We cannot rule out the possibility that our participants shifted their COM towards an expected single leg BOS even before TO, thereby reducing the possibility of the gravity-induced pelvic drop.

The pelvic obliquity was correlated with a decreased knee abduction moment only in the single limb stabilization phase. This result suggests an important message that the association between the frontal plane pelvic motion and the knee abduction moment of the stance limb should not be considered as an absolute relationship. Specifically, whether or not the frontal plane pelvic motion influences the knee abduction moment may depend on how the pelvic motion is involved in achieving the functional goals of each movement phase. It is likely that pelvic obliquity before TO of the SLMS was actively involved in the lateral transfer of body weight by facilitating the lifting of an unloading limb (Assaiante, Woollacott, & Amblard, 2000). In the single limb stabilization phase, the main purpose of frontal plane pelvic motion is likely to be assisting postural stability (Chang et al., 2005). Decreased pelvic obliquity during the
descending phase may indicate that the frontal plane pelvic motion served a purpose of
descending the body weight as well as maintaining the postural stability. The fact that the
relationship between pelvic obliquity and knee abduction moment was found only in the single
limb stabilization phase may imply that the frontal pelvic motion has an influence on the knee
abduction moment only when its primary purpose is to control postural stability. In order to
understand how a postural control strategy affects the knee abduction moment, further study is
recommended including the data of body COM and the location of centre of pressure.

Physical therapists or exercise therapists often instruct clients to maintain a level pelvic
posture while performing a single limb knee rehabilitation exercises such as SLMS or single
limb step-down (Bahr & Engebretsen, 2009). Considering the relationship between pelvic
obliquity and decreased knee abduction moment found in the present study, our study supports a
clinicians’ emphasis on controlling frontal plane pelvic motion for enhancing the efficacy of
knee rehabilitation exercises. In addition, these results add insight into when the control of pelvic
motion should be emphasized: The fact that the pelvic obliquity was correlated with reduced
knee abduction moment only during the single limb stabilization phase suggests that the control
of frontal plane pelvic motion before proceeding to the descending phase should be emphasized
for appropriate management of the knee abduction moment during rehabilitation.

While pelvic obliquity appears to be a potential factor involved in stabilizing knee abduction
moment in the present study, caution is required not to conclude that pelvic obliquity is
absolutely desirable for maintaining frontal plane knee stability. Our study did not investigate
possible confounding factors for the relationship between pelvic obliquity and knee abduction
moment. Trunk leaning could also affect the frontal plane motion of the pelvis. According to
Neumann (2010), a common maneuver to compensate for poor function of the hip abductors and shift the COM during continuous locomotion is to increase trunk leaning towards the stance limb which is usually accompanied by increased pelvic obliquity. It was suggested that excessive leaning of the trunk towards the supporting limb may cause an excessive knee adduction moment especially when the body is supported by a single limb (Powers, 2010). Therefore, further studies investigating how trunk leaning is involved in establishing the relationship between pelvic obliquity and knee abduction moment are required for confirming the clinical applicability of our study.

4.5 Limb dominance and GMED control of knee biomechanics

A positive influence of GMED activation on the reduction of a knee abduction moment was found only in the non-dominant limbs. In the dominant limbs, a rather adverse impact of earlier GMED onset on increased knee valgus was seen. Taken these results together, it seems that the non-dominant limb may more effectively control frontal plane knee kinematics and kinetics than the dominant limb. Supporting this speculation, the non-dominant limbs experienced significantly greater reduction of a knee abduction moment compared to the dominant limbs in the single limb stabilization phase.

To our knowledge, this is the first study observing the effect of limb dominance on the relationship between GMED and knee kinetics and kinematics. A study by Matsusaka, Fujitta, Hamamina, Norimatsu, and Suzuki (1985) may provide a clue about this effect of limb dominance on the GMED control of knee mechanics: In their study, the medial-lateral thrust and braking ground reaction forces on each foot during walking were examined as variables representing lateral balance control. While the forces on the dominant foot were significantly
affected by the forces generated by the non-dominant limb, the non-dominant limb independently controlled its medial-lateral ground reaction force (Matsusaka et al., 1985). The authors concluded that medial-lateral balance during walking is predominantly controlled by non-dominant limbs. This implication can be related to our study, because the lateral ground reaction forces discussed in the study by Matsusaka et al. (1985) are known to be substantially controlled by the GMED activity (Rogers & Pai, 1993). Our participants may also have preferred their non-dominant limb to their dominant limb for balance control, because the legs that provide support during daily activities (Elias et al., 1998) were defined as non-dominant limbs in the present study. Considering the possibility that the non-dominant limb is under stability demands more frequently than the dominant limbs, we can speculate that the non-dominant limb GMED was trained to have higher adaptability for coordinating postural and joint stability during a single limb balance control activity. A study by Ford et al. (2003) may also support the aforementioned idea: In their study, female high school athletes showed a greater increase of knee valgus in their dominant limb than non-dominant limbs during a single leg landing from a jump. The authors argued that the result may indicate poorer neuromuscular coordination of the dominant limbs for stabilizing frontal plane knee alignment (Ford et al., 2003). The neuromuscular coordination discussed in that study can be reasonably related to the GMED activation examined in our study. Our result that an earlier GMED onset only in the dominant limb was related to an increased knee valgus may support the speculation by Ford et al. (2003) that the dominant limbs have a less efficient neuromuscular control of the frontal plane knee motion compared to the non-dominant limbs.
Brophy et al. (2010) argued that dominant limbs may be more prone to sports related noncontact leg injuries, possibly because it is less trained for various stability demands than non-dominant limbs. Limb dominance has been frequently listed as a risk factor of noncontact knee injuries (L.Y. Griffin et al., 2006; Murphy, Connolly, & Beynnon, 2003), but there is controversy regarding which limb is more prone to the injury (Murphy et al., 2003). Our study suggests a possibility that the capability of GMED for controlling frontal plane knee kinetics and kinematics is higher in non-dominant limbs than in dominant limbs. This may imply that a dominant limb might be more predisposed to the knee injuries that are related to a poor frontal plane knee alignment in a weight bearing situation.

Despite the results showing non-dominant limb-biased GMED control of the knee mechanics, we cannot assume that the dominant limb GMED does not have any influence on the frontal plane knee motion and moment. The lack of relationship in the dominant limbs may have been caused by an incompetent adaptability of the dominant limb GMED for controlling medial lateral stability of the lower extremity during a novel task. Participants in our study were asked to coordinate the timing of the movement according to a consistent metronome beats. Even though 80 BPM is a comfortable pace to match each phase of SLMS, and the participants were given enough time to practice before the actual data collection, there may still be a possibility that even after the participants learned to adapt their movement pattern to the metronome beat, coordinating intra-limb behavior (i.e., GMED control of frontal plane knee stability) could still be challenging. It was suggested that a neuromuscular mechanism for single limb balance control may be more adaptable to a specific task if the responsible musculoskeletal system has previously been functionally challenged by similar tasks (Gioftsidou et al., 2006). Considering
that a limb that is mainly responsible for stability during daily activities was defined as non-dominant limb in our study, it is possible that the participants had been previously exposed to functional challenges similar to that of SLMS in their non-dominant limbs more than their dominant limbs.

4.6 Comparisons with clinical studies

Our study indicated a potential association between the GMED activation magnitude and the knee abduction moment. Given that excessive knee abduction moment is a well known biomechanical risk factor of knee osteoarthritis (OA) and PFP, our study justifies the need for the further investigations on the association between the GMED activation magnitude and the knee pain. Currently, there is lack of literature on how GMED activation magnitude is altered among the patients with above mentioned knee pain syndromes. Brindle et al. (2003) compared GMED activation magnitude between symptomatic and asymptomatic legs of participants with PFP walking down stairs, and found no significant differences. Considering that the SLMS and stair descent have many functional similarities (Ageberg et al., 2010), the result of our study is somewhat contradictory to the study by Brindle et al. (2003). One major limitation of the study by Brindle et al. (2003) is that the GMED activation parameter was compared within the participants who already developed PFP. Asymptomatic legs of the patients with PFP cannot necessarily be assumed to have different EMG patterns from the symptomatic legs, because unilateral pain perception may induce a bilateral response of the central nervous system (Kakigi, Inui, & Tamura, 2005). In fact, the effect of unilateral leg pain on the alteration of EMG pattern of a contralateral leg has been reported (Berger, Regueme, & Forestier, 2010). In addition, while the GMED activation magnitude in our study was calculated separately by functionally unique
movement phases, Brindle et al. (2003) calculated it by integrating the EMG over the entire duration of GMED activation. Moreover, the authors themselves (Brindle et al., 2003) stated that having only six participants may have contributed to the statistical non-significance. Our study analyzed data of 20 participants which was an enough sample size to detect correlation coefficients of 0.50 or greater at $\alpha = 0.05$ at a statistical power of 0.70.

Aminaka et al. (2011) reported that patients with PFP had both slower GMED onset and greater knee abduction impulse than healthy participants during a stair descent. Their study warranted a need for investigating a possible association between the GMED onset and knee abduction moment; however, our study could not support the possibility that these two variables are correlated. It is worth noting that our study and the study by Aminaka et al. (2011) used different reference movement events when calculating GMED onset. In the study by Aminaka et al. (2011) GMED onset time was calculated relative to toe-contact of the stance limb, while, in the present study, the onset was calculated relative to toe-off of the unloading limb. It was expected that the knee abduction moment would rapidly increase at toe-off of the SLMS because of the substantial influence of the forces inducing pelvic drop; however, the knee abduction moment started increasing rapidly even before TO, and all participants showed pelvic obliquity instead of pelvic drop at TO. Moreover, GMED onset occurred on average 330 msec before TO, which is much longer than $53 \pm 6$ msec, a previously reported electromechanical delay of this muscle (Kim et al., 2011). Therefore it is possible that some other movement event occurring even before TO might have been a critical reference event for the central nervous system to preplan the GMED activation timing. On the other hand, Aminaka et al. (2011) found a significant delay of GMED onset among the PFP group when the onset time was calculated
relative to the toe-contact of the stance limb. The functional demands on the GMED for preventing excessive knee abduction moment at toe-contact can be fundamentally different from that at TO, because the ground reaction forces (GRF) induced by the combination of contact forces and the muscular forces for decelerating the forward projection of the body COM play an important role for generating a knee abduction moment at toe-contact. GRF data will provide further insight regarding neuro-mechanical demands on GMED for facilitating weight acceptance of the supporting limb; these insights are required for more meticulous investigation on the relationship between GMED onset and knee abduction moment.

4.7 Limitations

Understanding several methodological limitations described below will provide guidance for practical interpretation of the results of the present study.

Some participants showed knee varus at toe-off of the non-supporting limb. Because relatively more participants showed knee valgus than varus at TO on their dominant limbs, our result was interpreted that an earlier GMED onset contributed to increased knee valgus. However, we cannot rule out the possibility that earlier GMED onset actually contributed to decreased knee varus. Considering that varus alignment of the knee has been associated with the progression of medial knee osteoarthritis (Sharma et al., 2001), further investigation is required to confirm the relationship between GMED onset and knee alignment.

Caution is also required to avoid concluding that increased pelvic obliquity is absolutely a good thing for maintaining the frontal plane knee stability. Trunk leaning can affect the frontal plane motion of the pelvis. It was suggested that excessive leaning of the trunk towards the supporting limb may cause excessive knee adduction moment especially when the body is
supported by a single limb (Powers, 2010). Because we did not measure trunk leaning, we cannot rule out the possibility that the trunk-leaning-mediated excessive pelvic obliquity might cause excessive knee adduction moment.

The frontal plane knee angle was calculated using a 2D method. A 3D method was not possible due to cross-talk between signals of the different planes. The major limitation of the 2D method is that the knee valgus angle can be affected by hip internal rotation and the toe-out angle of the stance limb. Future studies can be benefitted by controlling toe-out angles between the participants as well as between the trials.

Due to time limitations, the 2D knee angle was analyzed only up to single limb stabilization phase. Further analysis on the 2D knee angle during the descending phase is ongoing.

Skin movement may have influenced the location of the 3D motion analysis markers during data collection. We used cluster market set to minimize this artifact, but it is difficult to assume that our data was completely unaffected by the skin movement.

It should be noted that estimating the joint centre in 3D model can be affected by the joint ROM participants reached during functional calibration session. According to Camomilla et al. (2006), wider ROM leads to more accurate estimation of the hip joint centre. Given the probability that the hip ROM is dependent on the flexibility of the participants, there exists a possibility that 3D kinematics and kinetics for each participant were calculated at different accuracy.

The variability of the maximum knee flexion angle between the participants was high. Spearman’s Rank Order correlation analyses indicated that the knee flexion angle did not
significantly affect the knee abduction moment. However, because we have not yet analyzed the 2D knee angle, we cannot rule out the possibility that the knee angle was affected by the maximum knee flexion angle.

There were many occasions when the EMG system detected heart beats during the data collection which could not be eliminated through the filtering method. This led to a problem of false detection of EMG onset when using a 25 msec onset threshold window which was known to be reliable (Bolgla et al., 2010). We solved this problem by increasing the threshold window to 65 msec. The 65 msec window was also applied as an offset threshold. The GMED activity was very low during the late stage of the single limb stabilization phase and the early stage of the descending movement phase. There is a possibility that the offset was not detected during this period due to the relatively long offset threshold window. While this may be a methodological limitation, it did not seriously affect the interpretation of our data, because offset timing was not an outcome variable of our study.

The participants were instructed to coordinate their movement with the consistent metronome beats. Even though 80BPM was determined as a comfortable pace during the pilot testing, we cannot rule out the possibility that the demands for following the metronome beat might have disrupted participants’ natural movement rhythms.

Multiple hypotheses were tested by repeating correlation statistics. Because α level of significance was not adjusted (due to small sample size: N = 20) in the present study, we cannot rule out the possibility of type I family-wise error.
4.8 Summary and implications

The primary purpose of the present study was to examine how neuromuscular function of GMED is related to knee valgus and knee abduction moment during the Single Limb Mini Squat (SLMS). Contrary to our hypothesis, earlier GMED onset was not protective against increased knee valgus and abduction moment. Greater abductor strength of the dominant limb was related to less knee valgus in the same movement phase. Considered together, hip strengthening exercises should continue to be emphasized for designing prevention strategies of knee pain.

The GMED activation magnitude, not the activation timing or hip strength, was significantly related to the knee abduction moment. It is likely that this relationship was influenced by the control of pelvic obliquity. The relationship between pelvic obliquity and the knee abduction moment was noticeable only during the single limb stabilization phase. This provides important information as to when the pelvis control should be emphasized during rehabilitation exercises for knee pain such as SLMS.

Another notable finding is that a greater magnitude of the GMED activation over 60 msec prior to TO was related to both decreased knee abduction moment and increased pelvic obliquity during the single limb stabilization phase. This may indicate that the preparatory GMED activation before the single limb stabilization phase is critical for protecting against an excessive knee abduction moment. This can improve EMG based biofeedback rehabilitation training for treating PFP or knee OA.

There seems to be an effect of limb dominance on GMED function for coordinating knee joint stability. The result showing significantly greater knee abduction moment in the dominant limb may indicate relatively less coordinated neuromuscular control in the dominant limbs. Our
results suggest that studies examining neuromuscular control of knee biomechanics include both the dominant and non-dominant limb. A possible mechanism for the relatively poorer neuromuscular control of the dominant limbs is that the dominant limbs have less exposure to daily stability demands than the non-dominant limbs. An exercise program should therefore equally challenge each limb with the balance control, or if necessary, more on the dominant limbs.
5 CONCLUSIONS

The primary purpose of the present study was to examine how GMED activation parameters were related to the control of knee abduction moment and knee valgus during the SLMS. The secondary purpose was to clarify if hip abductor strength is related to knee abduction moment and dynamic knee valgus. Data were analyzed according to functionally unique movement phases to appropriately relate GMED response and knee joint behavior occurring under similar neuro-mechanical demands. The SLMS movement consisted of double to single limb transition, single limb stabilization, and a descending movement. Based on the major findings listed below, it can be concluded that hip abduction strength is important for controlling knee alignment during the double to single limb transition. In addition, a high activity level of GMED is important for decreasing knee abduction moment during the single limb stabilization phase. Preparatory activation of GMED before toe-off is also important for mitigating the knee abduction moment during the single limb stabilization phase. Moreover, there seems to be an effect of limb dominance on neuromuscular coordination of GMED for controlling knee abduction moment.

- Greater concentric hip abductor strength was significantly correlated with less knee valgus at toe-off only in the dominant limb. On the other hand, earlier GMED onset was significantly correlated with more knee valgus at toe-off only in the dominant limb. This provides partial evidence that having greater hip abductor strength may be more effective than activating the GMED earlier for preventing knee valgus.
- Greater total GMED activation magnitude over the entire single limb stabilization phase was correlated with a decreased knee abduction moment during the same phase in the non-
dominant limb only. This result provides partial evidence for the association between GMED activation magnitude and knee abduction moment.

- A significant relationship between GMED activation magnitude and knee abduction moment was found only when the following conditions were present: 1) Greater GMED activation magnitude was significantly correlated with increased pelvic obliquity; 2) Increased pelvic obliquity was significantly correlated with decreased knee abduction moment. This strongly suggests a possibility that GMED activation affects knee abduction moment by controlling frontal plane pelvic motion.

- The association between greater GMED activation magnitude and reduced knee abduction moment was observed in the non-dominant limb only, and the non-dominant limbs experienced greater mitigation of knee abduction moment compared to the dominant limbs. This indicates a potential effect of limb dominance on the ability of GMED to control knee abduction moment.

- Greater total activation magnitude over 60 msec before the single limb stabilization phase was significantly correlated with decreased knee abduction moment during the single limb stabilization phase in the non-dominant limb only. Reduced total activation magnitude over 60 msec before the initiation of descending movement was significantly correlated with decreased knee abduction impulse during the descending movement. It will be necessary to further investigate whether a preparatory modulation of GMED activity affects knee abduction moment during an upcoming movement.
6 REFERENCES


Chumanov, E. S., Wall-Scheffler, C., & Heiderscheit, B. C. (2008). Gender differences in walking and running on level and inclined surfaces. [Comparative Study]


A. Questionnaire for participant eligibility

Please answer YES or NO to the following questions:

- Do you have current injury and/or pain that changes how you move in your daily life including stepping down the stairs?
- Have you had a knee sprain of grade 2 (partial tears of ligaments) and/or above?
- Have you had a hip fracture, dislocation, or sprain of grade 2 (partial tears of ligaments) and/or above?
- Have you had ankle sprain that required immobilization for at least 3 days?
  a. If Yes, did you go through any formal rehabilitation (i.e., physical therapy)?
  b. If Yes, have you had pain or feeling of looseness at the previously sprained ankle during daily activities within the past 3 months?
- Have you had any low back pain in the last six months?
  a. If Yes, please describe whether the back pain has been consistent or periodic and the associated timelines with your low back pain
- Have you ever been instructed on proper technique for performing a squat or jump landing?
- Are you currently enrolled KIN 322?
B. Questionnaire about current injuries

1. If any additional pain/injury occurred after the phone/e-mail interview, please answer following questions:

   (1) Does your pain/injury change how you walk up and down the stairs?      Yes       no
   (2) Does your pain/injury change how you stand on one leg?                        Yes       no
   (3) Does your pain/injury change how you run?      Yes       no

* For each statement, please indicate with an ‘X’ how much knee pain you have experienced over the last week.
### C. Questionnaire about leg dominance

**Modified Waterloo Footedness Questionnaire (Elias et al., 1998)**

**Instructions:** Answer each of the following questions as best you can. If you always use one foot to perform the described activity, circle **Ra** or **La** (for **right always** or **left always**). If you **usually** use one foot circle **Ru** or **Lu**, as appropriate. If you use **both** feet equally often, circle **Eq**.

Please do not simply circle one answer for all questions, but imagine yourself performing each activity in turn, and then mark the appropriate answer. If necessary, stop and pantomime the activity.

<table>
<thead>
<tr>
<th>Question</th>
<th>Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Which foot would you use to kick a stationary ball at a target straight in front of you?</td>
<td>La Lu Eq Ru Ra</td>
</tr>
<tr>
<td>2. If you had to stand on one foot, which foot would it be?</td>
<td>La Lu Eq Ru Ra</td>
</tr>
<tr>
<td>3. Which foot would you use to smooth sand at the beach?</td>
<td>La Lu Eq Ru Ra</td>
</tr>
<tr>
<td>4. If you had to step up onto a chair, which foot would you place on the chair first?</td>
<td>La Lu Eq Ru Ra</td>
</tr>
<tr>
<td>5. Which foot would you use to stomp on a fast-moving bug?</td>
<td>La Lu Eq Ru Ra</td>
</tr>
<tr>
<td>6. If you were to balance on one foot on a railway track, which foot would you use?</td>
<td>La Lu Eq Ru Ra</td>
</tr>
<tr>
<td>7. If you wanted to pick up a marble with your toes, which foot would you use?</td>
<td>La Lu Eq Ru Ra</td>
</tr>
<tr>
<td>8. If you had to hop on one foot, which foot would you use?</td>
<td>La Lu Eq Ru Ra</td>
</tr>
<tr>
<td>9. Which foot would you use to help push a shovel into the ground?</td>
<td>La Lu Eq Ru Ra</td>
</tr>
<tr>
<td>10. During relaxed standing, people initially put most of their weight on one foot, leaving the other leg slightly bent. Which foot do you put most of your weight on first?</td>
<td>La Lu Eq Ru Ra</td>
</tr>
<tr>
<td>11. Is there any reason (i.e. injury) why you have changed your foot preference for any of the above activities?</td>
<td>YES NO (circle one)</td>
</tr>
<tr>
<td>12. Have you ever been given special training or encouragement to use a particular foot for certain activities?</td>
<td>YES NO (circle one)</td>
</tr>
<tr>
<td>13. If you have answered YES for either question 11 or 12, please explain:</td>
<td></td>
</tr>
</tbody>
</table>
INTRODUCTION
You are invited to take part in this research study because you are a healthy young female adult and have qualified via prescreening to consider full participation.

It is up to you to decide whether or not you wish to take part. If you decide to participate, you will be asked to sign this form. If you decide to take part in this study, you are still free to withdraw at any time without giving any reasons for your decision.

If you do not wish to participate, you will not lose any benefit to which you are entitled or are presently receiving. It will not affect your relationship with any of the researchers associated with this study nor will withdrawal affect your academic standing.

Please take time to read the following information carefully. You can ask the study investigator to explain any words or information that you do not clearly understand. You may ask as many questions as you need. Please feel free to discuss this with your family, friends or family physician before you decide.

WHY IS THIS STUDY BEING DONE?
Poor knee joint alignment is a risk factor for knee injury and chronic knee pain (Andriacchi et al., 2004; Hewett et al., 2005). Recent research suggests that hip abductor function is related to the control of knee joint position; however, it is not clear how the hip abductors affect knee joint alignment. This study will investigate the relationship between hip abductor activation and knee joint position. Approximately 20 healthy young female adults will be recruited from the University of Saskatchewan. The results of this study will provide useful knowledge to clinicians for developing evidenced-based methods for injury prevention.

WHO CAN PARTICIPATE IN THE STUDY?
You are eligible to participate in this study if you are female between the ages of 18-45 years. If you have any of the following conditions, you are not eligible to participate in this study:

1. Current injury and/or pain causing modification to activities of daily living, in particular, stepping down the stairs
2. A history of knee sprain with grade 2 (partial tears of ligaments) and above
3. A history of hip fracture, dislocation, or sprain with grade 2 (partial tears of ligaments) and above
4. A history of at least 1 ankle sprain that required immobilization for at least 3 days with any of the following conditions: (1) no formal rehabilitation of the involved ankle, (2) pain or feeling of looseness at the previously sprained ankle during daily activities within the past 3 months
5. Currently pregnant
6. A history of periodic back pain episodes within the past 6 months

In addition, if you have had any previous instruction on proper technique for performing a single-leg mini-squat, you are not eligible to participate. In order to prevent potential conflict of interest, if you are currently enrolled in KIN 322 with Dr. Alison Oates as an instructor and/or Daehan Kim as a teaching assistant, you are not eligible to participate.

**WHAT DOES THE STUDY INVOLVE?**

Before we ask you to come to the Biomechanics lab for the main data collection, the student researcher (Daehan Kim) will ask you via phone or e-mail about any history of injuries, knowledge of a squat technique, and whether you are enrolled in KIN 322. These prescreening questionnaires will help us to determine your eligibility for participating in this study. We will ask you to come to the Biomechanics lab on a single occasion for approximately 2 hours. There is a possibility; however, that we may ask you to come for a second visit to perform the strength testing on a separate day. This second visit will only be requested if there is a scheduling conflict between the two labs where data collection will take place. Upon arrival to the Biomechanics lab, you will be asked a series of questions about current injuries, pains, and your dominant leg. You may refuse to answer questions that you are not comfortable with.

After completing the questionnaires, your hip and knee strength will be measured. Before performing the strength tests, you will be required to do a five-minute warm-up including dynamic stretching and biking on a stationary bike lead by a student researcher (Daehan Kim). An isokinetic dynamometer will be used to measure your strength. Your legs will be strapped in a special device to stabilize the joints and assure your safety during testing. You will be asked to exert as much force as you can when performing movements centered around your hip and knee. You will have the opportunity to practice the movements to become familiar with the task before the actual testing. Each strength test will be repeated three times per leg. Hip strength will be measured in a side-lying position, and knee strength will be measured in seated position. You will be provided with a minimum of two minutes rest between each strength test.
After strength testing, reflective spheres will be placed on specific parts of your body using double-sided tape. Special cameras in the room will record the movement of the markers. The data from the markers will be used to provide information about how you moved during the data collection. To measure the activity of your muscles, electrodes similar to those used to measure your heart beat will be placed over specific muscles on the side of your buttocks, the inside, front and back of your thighs and, possibly the front and back of your lower legs. You will be asked to wear shorts (preferably spandex). You can use a private change room in front of the laboratory. The area where the electrodes are to be placed will be prepared in advance by shaving your skin to remove any hair where the electrodes will be placed (approximately 10x10cm) and cleaning that area with a standard alcohol solution. Shaving and cleaning is mandatory to improve the quality of the electrical signal from your muscles, but they will be limited to the necessary areas in order to minimize potential discomfort. Someone of the same gender as you will do the shaving and electrode placement. The electrode preparation and application will be done in a closed laboratory for your privacy. The data from the electrodes is sent to the main collection computer wirelessly through a pack that you will wear on a belt. The system is CSA approved and has had extensive safety testing to ensure that you are safe. No electrical activity is sent from the computer or the pack on your belt to your muscles. To measure the forces under your feet, you will stand on a force platform embedded in the standing surface. There is no extra preparation required for you to stand or move on the force platform.

After all the markers and electrodes are placed on your body, you will be asked to stand quietly on a force platform for two minutes. After the quiet stance trial, you will be asked to perform single-leg mini-squats. You will be asked to perform the movement at a predetermined pace following metronome beats at 90 beats per minute. You will receive brief instructions and a demonstration before beginning data collection. You will be asked to perform each movement 10 times for each leg in a random order. You are free to rest between trials as much as you want.

**WHAT ARE THE BENEFITS OF PARTICIPATING IN THIS STUDY?**
This study does not provide direct benefit to participants. It is hoped that the information gained from this study can be used in the future for developing useful injury prevention methods.

**ARE THERE POSSIBLE RISKS AND DISCOMFORTS?**
The preparation and adhesive used for the electrodes and markers may cause a mild, temporary skin irritation similar to a band-aid that should disappear within a few days. Additionally, you may experience mild fatigue and soreness as a result of the strength testing and movements performed during data collection. This fatigue and soreness is temporary and should disappear in a few days. If fatigue and soreness does not go away within four days, please contact Daehan Kim (306) 716-6498, or Alison Oates, PhD (306) 966-1080.

**WHAT HAPPENS IF I DECIDE TO WITHDRAW?**
Your participation in this research is voluntary. You may withdraw from this study at any time. You do not have to provide a reason. Your decision of withdrawal will not affect your academic status nor your
relationship with any of the researchers. If you choose to enter the study and then decide to withdraw later, all data collected about you during your enrolment will be retained for analysis.

**WILL I BE INFORMED OF THE RESULTS OF THE STUDY?**
It is the intention of the researchers to publish the results of this research and to present the findings at related conferences and workshops. You may request the results of this study as well as your individual data. It is important to note that none of the information or data will be provided to you for any diagnostic or prescription purposes.

**WHAT WILL THE STUDY COST ME?**
You will not be charged for any research-related procedures. You will not be paid for participating in this study. Reimbursement for study-related expenses (e.g. travel, parking, meals) is not available.

**WHAT HAPPENS IF SOMETHING GOES WRONG?**
By signing this document, you do not waive any of your legal rights. In the case of a medical emergency during your visit, necessary medical treatment will be made available at no cost to you.

**WILL MY TAKING PART IN THIS STUDY BE KEPT CONFIDENTIAL?**
In Saskatchewan, the Health Information Protection Act (HIPA) protects the privacy of your personal health information. Your privacy will be respected. Your name will not be attached to any information, nor mentioned in any study report, nor be made available to anyone except the research team. It is the intention of the research team to publish results of this research in scientific journals and to present the findings at related conferences and workshops, but your identity will not be revealed. In case the high-speed digital video camera data is presented at a conference or a workshop, any indentifying parts of the image will be completely masked to protect your confidentiality.

**WHO DO I CONTACT IF I HAVE QUESTIONS ABOUT THE STUDY?**
If you have any questions or desire further information about this study before or during participation, contact Daehan Kim (306) 716-6498, or Alison Oates, PhD (306) 966-1080. If you have any concerns about your rights as a research participant and/or your experiences while participating in this study, contact the Chair of the University of Saskatchewan Research Ethics Board, at 306-966-4053. The Research Ethics Board is a group of individuals (scientists, physicians, ethicists, lawyers and members of the community) that provide an independent review of human research studies. This study has been reviewed and approved on ethical grounds by the University of Saskatchewan Research Ethics Board.

**REFERENCES**


CONSENT TO PARTICIPATE

Study Title: Relationship between gluteus medius activation and dynamic knee position

- I have read (or someone has read to me) the information in this consent form.
- I understand the purpose and procedures and the possible risks and benefits of the study.
- I was given sufficient time to think about it.
- I had the opportunity to ask questions and have received satisfactory answers.
- I understand that I am free to withdraw from this study at any time for any reason and the decision to stop taking part will not affect my future relationships.
- I give permission to the use and disclosure of my de-identified information collected for the research purposes described in this form.
- I understand that by signing this document I do not waive any of my legal rights.
- I will be given a signed copy of this consent form.

I agree to participate in this study:

Printed name of participant:

__________________________________________________________________________

Signature                                        Date

Printed name of person obtaining consent:

__________________________________________________________________________

Signature                                        Date
E. Electrodes location and orientation

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Electrodes location</th>
<th>Electrodes orientation</th>
</tr>
</thead>
<tbody>
<tr>
<td>GMED</td>
<td>50% on the line from the iliac crest to the greater trochanter</td>
<td>In the direction of the line from the iliac crest to the greater trochanter</td>
</tr>
<tr>
<td>AL</td>
<td>1/3 of the distance between medial epicondyle of the femur and the pubic symphysis</td>
<td>In the direction of the line from pubic symphysis to the middle third of the medial lip of the linea aspera</td>
</tr>
<tr>
<td>BF</td>
<td>50% on the line between the ischial tuberosity and the lateral epicondyle of the tibia</td>
<td>In the direction of the line between the ischial tuberosity and the lateral epicondyle of the tibia</td>
</tr>
<tr>
<td>VM</td>
<td>80% on the line between the ASIS and the joint space in front of the anterior border of the medial ligament.</td>
<td>Almost perpendicular to the line between the ASIS and the joint space in front of the anterior border of the medial ligament.</td>
</tr>
</tbody>
</table>

* GMED: gluteus medius, AL: adductor longus, BF: biceps femoris, VM: vastus medialis
F. Electrode set up

G. Marker set up
G. Isokinetic dynamometry set up

Hip testing

Knee testing