A Biomechanical and Neuromuscular Investigation of Femoroacetabular Impingement: Understanding Pathomechanics using Inclined and Declined Walking

by

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Submitted in partial fulfilment of the requirements
for the degree of Master of Science

at

Dalhousie University
Halifax, Nova Scotia
July 2020

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Abstract

Femoroacetabular impingement (FAI) is a proliferative musculoskeletal diagnosis in young adults. The purpose of the current study was to investigate the effect of challenged walking on gait mechanics in individuals with FAI compared to matched asymptomatic individuals. Two groups of seven underwent gait analysis during level, incline and decline walking. Kinematic data and surface electromyography were recorded using standardized procedures. Sagittal plane kinematics and activation of the gluteus maximus and medius were calculated. A 2-way mixed methods ANOVA identified differences in kinematics and neuromuscular activations. Significant main effects for condition were found for all biomechanical and neuromuscular variables ($p \leq 0.05$). No significant between group differences were found; however, moderate to large effect sizes were identified for sagittal plane hip range of motion, peak hip extension, gluteus maximus and gluteus medius. Results suggest that challenged walking may elicit novel biomechanical and neuromuscular alterations between individuals with FAI and asymptomatic controls.

List of Abbreviations Used

ANOVA – Analysis of variance

ASIS – Anterior superior iliac spine

CI – Confidence interval

CoM – Center of mass

EMG – Electromyography

FABER – Flexion abduction external rotation

FADIR – Flexion adduction internal rotation

FAI – Femoroacetabular Impingement

FFT – Fast Fourier Transformation

Gmax – Gluteus maximus

Gmed – Gluteus medius

GRF – Ground reaction force

HOOS – Hip Outcome Osteoarthritis Score

ICC – Intraclass correlation coefficient

ICF – International Classification of Functioning, Disability and Health

iHOTT-33 – International Hip Outcome Tool

LH – Lateral hamstrings

MG – Medial gastrocnemius

MH – Medial hamstrings

MVIC – Maximum voluntary isometric contraction

NAHS – Non-Arthritis Hip Score

NSHA – Nova Scotia Health Authority

NSHRF – Nova Scotia Health Research Foundation

OA – Osteoarthritis

OR – Odds ratio

QTM – Qualisys Track Manager

REB – Research Ethics Board

RF – Rectus femoris

ROM – Range of motion

SD – Standard deviation

SENIAM – Surface Electromyography for the Non-Invasive Assessment of Muscles

SOL – Soleus

SPSS – Statistical Package for the Social Sciences

VL – Vastus lateralis

VM – Vastus medialis

VR – Veterans Rand

WHO – World Health Organization

Acknowledgements

There are a number of people I would like to thank who directly or indirectly had a positive impact on my experience as a master's student at Dalhousie University. Firstly, I would like to thank my supervisor, Dr. Rebecca Moyer for her continued support and patience throughout my MSc studies over the past two years. Dr. Moyer's organization, enthusiasm and dedication to higher learning has left a lasting impact on my appreciation for the research process. Through these unprecedented times, this work would not have been possible without her mentoring and continued encouragement.

I would also like to thank my supervisory committee, Dr. Janice Moreside and Dr. Derek Rutherford for not only their guidance and support but for providing their time and feedback throughout my MSc work. Also, I would like to thank Dr. Scott Landry for agreeing to act as my external reviewer and his continued mentorship, without whom I would not be where I am now.

To all the members and fellow graduate students of the Dynamics of Human Motion and Joint Action Research Laboratories, I thank you for your assistance with data collections and for making this process as easy as possible. A special thank you to Matthew Baker for taking me under his wing throughout this process and always lending a helping hand.

Finally, this would not have been possible without my family and partner, Taylor Johnston. Her love and support over these years has helped me though many tough times. I cannot thank her enough for her patience, encouragement and tolerance of late nights and occasional bad moods. Her constant positive attitude has always inspired me to keep working, and I could not have asked for a better person to have spent these past years with.

Chapter 1 - Introduction

1.1 Introduction

Femoroacetabular impingement (FAI) is defined as a "motion-related clinical disorder of the hip, with a triad of symptoms, clinical signs and imaging findings" and represents abnormal, symptomatic contact between the proximal femur and the acetabulum[1]. Currently, the prevalence of FAI in the general adolescent population is approximately 25%[2], but in an older, athletic population the prevalence has been reported up to 55%[3]. The characteristic bony contact associated with FAI is classified as either cam- or pincer type, and often presents as a combination of both. Cam type FAI is caused by a bony outgrowth on the femoral shaft, leading to an aspherical femoral head and abnormal articulation with the acetabulum[6,7,8,9]. Pincer type FAI is caused by an overgrowth of the acetabular rim over the femoral head, leading to the abutment of the femoral neck on the acetabulum during end ranges of motion (ROM)[10]. This symptomatic contact between the femoral head and acetabulum is a proposed risk factor for abnormal joint mechanics thus accelerating cartilage degradation, labral damage and increases in pain[4,5].

FAI is a proliferative musculoskeletal diagnosis among young-to-middle aged adults and proposed to be a significant risk factor for hip osteoarthritis (OA). In 2010, the prevalence of arthritis in Canada was approximately 15.3%[11], which increased to 17% by 2017[12] and is estimated to escalate beyond 25% by 2035[13]. OA etiology is complex and continues to evolve. Recently, individuals with FAI have been thought to be an early, at-risk population for the development of hip OA[14,15,16]. The link between these pathologies was not proposed until 2003; however, since then, FAI research has increased to improve our understanding of this potential pathway[17]. The proposed mechanism suggests that the

bony outgrowth on the femoral neck or acetabulum mechanically disrupts the distribution of joint stresses, typically over the anterior superior region of the acetabulum, leading to subsequent cartilage and labrum damage[4]. Recent studies suggest that cam type FAI (defined as an alpha angle greater than 50^{o}) was associated with hip OA[18,15,19]. Specifically, individuals with severe cam type pathology (alpha angle >83^{o}) had a 9-fold increase in their risk for hip OA development[20].

Individuals with FAI commonly present with hip and/or groin pain, which can be aggravated by prolonged sitting, high levels of physical activity, or movement patterns that occur at the end ROM[1,21,22]. Restricted movement associated with these symptoms include limited passive hip flexion and internal rotation due to the structural restrictions associated with contact of the femur on the acetabulum[23,24,25,26], while active ROM restrictions include reduced abduction, internal and external rotation[27]. A physical assessment tool for detecting FAI challenges is the hip flexion, adduction and internal rotation (FADIR) test, forcing the hip into an impinged position[23]. Additionally, diagnostic imaging includes radiographic assessment of the hip. Although a number of different imaging assessments exist, cam type morphology is commonly identified using the alpha angle to measure the size of the bony femoral outgrowth[28,29,30,31], and pincer type morphology is commonly identified using the center-edge angle to measure the distance of acetabular overgrowth on the femur[7]. As recommended by the FAI Warwick Agreement, no one sign or symptom should be used solely to diagnose FAI, but rather a combination of symptoms, clinical signs and imaging findings should be used[1].

Symptomatic, functional limitations are often observed in individuals with FAI during activities such as walking, running or squatting[32,33]. During level ground walking,

research suggests that individuals with FAI walk with less dynamic sagittal, frontal and transverse plane hip ROM[32], with consistent patterns also observed during more challenging ambulatory tasks such as stair ascent and descent. These functional limitations have proposed associations with individual self-reports of decreased physical abilities and diminished quality of life[21,34,35,36].

The anatomical adaptations of the hip and its corresponding abnormal mechanical joint loading associated with both types of FAI, have driven human movement research using gait analysis to examine if differences exist between the hip movement patterns of individuals with FAI versus asymptomatic individuals. During level ground walking, inconsistent findings have been reported between these groups. In the sagittal plane, differences in total hip ROM have been reported[37,38,39]; however, there are discrepancies on whether flexion or extension is the limiting factor. Hunt and colleagues (2013)[38] suggested hip extension ROM as the limiting factor in the sagittal plane, which approximates the closed packed position of the hip resulting in an anteriorly directed force displacing the femur further into the damaged portion of the acetabulum. On the contrary, Rylander and colleagues (2013)[39] proposed hip flexion as the limiting factor in the sagittal plane primarily due to its association as the common impinged position of the hip, and therefore individuals may be reluctant to adopt this movement. Contradictory results also exist when describing altered frontal plane kinematics in this population with previous findings suggesting both reductions in peak hip adduction[38] and abduction[39,27] during stance. Similar to limited hip flexion, hip adduction may also reflect a position of hip impingement and possibly result in avoidance of this movement during gait[38]. Alternatively, decreased strength may be associated with reduced peak hip abduction[39,27].

Reduced hip abductor strength has previously been reported in individuals with FAI[40,41], which may result in a reduced ability to maintain proper hip abduction angles during stance. Lastly, few studies have investigated altered kinematic patterns in the transverse plane. Researchers observed overall decreases in transverse plane ROM, which was primarily driven by a reduced peak internal rotation angle[38,39]; however, multiple reports exist describing no difference in transverse plane ROM between individuals with FAI and asymptomatic individuals[35,37,42] suggesting further research is needed.

Understanding the neuromuscular contributions relative to hip joint kinematics during gait is also a key component for understanding potential human movement differences in this population; however, very few electromyography (EMG)-driven studies have evaluated muscular activation patterns during walking in individuals with FAI. To date, only two studies have reported muscle activation patterns during gait. Most recently Rutherford and colleagues (2018)[35] examined activation patterns of the primary lower limb muscle groups during level ground walking using principal component analysis. Results suggested that individuals with FAI had increased gluteus maximus (Gmax) activation during stance compared to asymptomatic controls and increased medial (MH) versus lateral hamstring (LH) activation[35]. Additionally, work using fine-wire EMG examined deep hip external rotator muscles during gait and reported increased activation during pre-swing, which was proposed as a compensation to limit hip internal rotation during the early swing phase of gait[43]. The variability in findings and methods between these studies currently limits the understanding of neuromuscular activation in this population.

Numerous authors have proposed that the previous inconsistencies, or lack of group differences, may reflect the minimal challenges of level walking that may not elicit

detectable, biomechanical changes and as a result, more challenging movements may be warranted[35,37]. Inclined and declined walking are suggested approaches for gait assessment that impose more demanding increases in hip ROM. We are unaware of previous research that has investigated the effects of inclined and declined walking in individuals with FAI; however, results stemming from asymptomatic individuals provides a thorough framework for beginning to understand how challenged ambulation might impact hip movement patterns in individuals with FAI. Previous reports of inclined walking over increasing gradient challenges suggest a progressive increase in sagittal plane hip ROM[44,45,46], increased hip adduction at initial contact[44,47] and increased hip internal rotation at initial contact[47]. Importantly, these sagittal, frontal and transverse plane hip movements are concurrent with the limiting movement patterns previously reported during level ground walking in individuals with FAI[38,39,27]. Therefore, these potential challenges imposed by inclined walking in asymptomatic individuals may be sufficient for eliciting biomechanical adaptations in the affected hip of individuals with FAI.

Neuromuscular responses to inclined versus level ground walking have also been observed in asymptomatic individuals. Previous research has shown increased activation of the Gmax[48,50] and the hamstrings[48,49,50] suggesting that this increased demand on the hip extensors may be required for translating the body up an incline. For example, a 9^{o} incline was associated with a 345% increase in mean Gmax activation and a 635% increase in mean biceps femoris activation during stance[48]. Notably, Gmax and hamstrings were previously shown to have altered activation patterns between individuals with FAI and asymptomatic individuals during level walking, which suggests that the added challenge

of inclined walking may amplify these potential neuromuscular differences and may elicit novel patterns un-identified during level walking.

Previous investigations of human movement during declined walking are substantially more limited. To date, no studies have investigated either frontal or transverse plane hip motions during decline walking in asymptomatic individuals. In the sagittal plane, decreases in hip ROM with increasing decline magnitudes have been reported such that a 29^{o} decrease in hip ROM was observed when transitioning the angle of walking from an 8.5^{o} incline grade to an 8.5^{o} decline grade[45]. Further, only one study has been identified that investigated the effects of declined walking on neuromuscular patterns of the major hip musculature[50]; therefore, more research quantifying potential movement differences during declined walking may better inform the joint-specific challenges associated with these walking patterns. These finding suggests that there is a significant increase in the demand on the hip in the sagittal plane, and further research focused on the effects of this demand on hip joint movement is needed.

Currently, there is a narrow understanding of human movement and hip-joint specific mechanics associated with FAI pathology, which is highlighted by contradictory sagittal, frontal and transverse plane hip kinematic differences as well as the minimal evidence related to neuromuscular activation patterns during gait. These inconsistencies suggest that for individuals with FAI, specifically prior to the development of hip OA, level ground walking may lack the necessary challenges to elicit significant biomechanical changes[35,37]. For this reason, examining movements that place the hip into a more impinged position, or challenge the joint beyond level ground walking, is a critical next step to better understand the symptomatic-response and functional limitations experienced by

individuals with FAI. Inclined walking is the proposed strategy to challenge the joint by potentially increasing sagittal plane hip ROM, peak flexion, adduction and internal rotation during stance, recreating a position of impingement as well as increase the demand on the neuromuscular system. Alternatively, declined walking may be less likely to recreate an impinged hip position; however, its effect on the biomechanical and neuromuscular systems in the sagittal, frontal and transverse planes is currently unknown. Understanding hip function throughout these movement patterns may provide valuable information on hip biomechanics associated with FAI, not evident during level ground walking.

Interventions for individuals with FAI, particularly surgical treatment decisions, are made using a two-dimensional image of a three-dimensional joint, combined with clinical tests and self-report measures. Individuals report difficulty with functional activities such as walking, sport and recreation and stair ambulation[21,34,35,36]; however, current level ground gait analyses may not be challenging enough to elicit significant biomechanical and neuromuscular responses within this population. Therefore, to help understand the effects of surgical or rehabilitation interventions on the pathomechanics of FAI, a need exists to further evaluate objective outcome measures that can be used to assess the effectiveness of these treatment strategies.

1.2 Overall Objective

For individuals with FAI, biomechanical and neuromuscular alterations are suggested outcomes associated with boney impingement during hip flexion, adduction and internal rotation, and increased risk for hip articular cartilage degradation. To date, there are a limited number of studies comparing biomechanical and neuromuscular patterns between individuals with FAI and asymptomatic individuals during level ground walking;

however, inconsistent findings support recommendations for investigating human movement and joint-specific mechanics during activities that require a more impinged position, or challenge the joint to a greater extent than level ground walking. Inclined walking in asymptomatic individuals increases sagittal, frontal, and transverse hip plane ranges of motion, as well as increase hip muscular activity to propel the individual up the incline. However, a thorough understanding of the effects of decline walking is lacking but may be less likely to challenge the joint by recreating an impinged position. We are unaware of previous research using inclined and declined walking to challenge movement in individuals with FAI. Challenged walking may be an effective way to elicit significant biomechanical and neuromuscular responses, which may clarify potential mechanical adaptations that link FAI and increased risk for hip OA.

The main purpose of this study is to determine whether hip joint kinematics and neuromuscular activation patterns differ during level ground walking and more challenged walking activities, including inclined and declined walking, between individuals with FAI and asymptomatic individuals.

1.3 Specific Objectives

The primary objectives of this study are to:

1. Determine if sagittal plane hip kinematics differ between individuals with FAI and asymptomatic individuals during level ground walking and more challenged walking. Total sagittal plane hip ROM as well as peak extension and flexion will be compared between five walking conditions: level ground walking, 5^{o} and 10^{o} inclined walking, and 5^{o} and 10^{o} declined walking. A secondary objective will determine if sagittal plane

hip kinematics differ during level ground walking before and after challenged walking in either group.

2. Determine if lower limb muscle activation amplitudes will differ between individuals with FAI and asymptomatic individuals during level ground walking and more challenged walking. Individual muscle amplitudes normalized to a percentage of maximum voluntary isometric contraction (MVIC) for the gluteus maximus (Gmax) and gluteus medius (Gmed) will be compared between five walking conditions: level ground walking, 5^{o} and 10^{o} inclined walking, and 5^{o} and 10^{o} declined walking. A secondary objective will determine if lower limb muscle activation amplitudes differ during level ground walking before and after challenged walking in either group.

1.4 Hypotheses

For the first objective, it is hypothesized that:

1. (a) Level ground walking will not result in any differences in peak flexion or extension; however, will result in decreased sagittal plane hip ROM in the FAI group compared to asymptomatic individuals, and (b) no differences will be observed for sagittal plane ROM, peak hip extension or flexion for either group during level ground walking when compared before and after the challenged walking protocol.
2. The 5^{o} and 10^{o} inclined walking trials will result in significantly decreased peak hip flexion, as well as decreased sagittal plane hip ROM in the FAI group compared to asymptomatic individuals.
3. The 5^{o} and 10^{o} declined walking trials will result in significantly decreased sagittal plane hip ROM in the FAI group compared to asymptomatic individuals.

For the second objective, it is hypothesized that:

4. (a) Level ground walking will not result in any differences in mean or peak MVIC normalized activation of the Gmed, however, will result in increased mean and peak MVIC normalized Gmax activation in the FAI group compared to asymptomatic individuals, and (b) no differences will be observed for mean or peak Gmax or Gmed activation for either group during level ground walking when compared before and after the challenged walking protocol.
5. The 5^{o} and 10^{o} inclined walking trials will result in increased mean and peak MVIC normalized activation of the Gmax, and reduced mean and peak MVIC normalized activation of the Gmed in the FAI group compared to asymptomatic individuals.
6. The 5^{o} and 10^{o} declined walking trials will result in decreased mean and peak MVIC normalized activation of the Gmed in the FAI group compared to asymptomatic individuals.

Chapter 2 - Review of Relevant Literature

2.1 What is FAI?

FAI is a mechanically driven clinical disorder resulting from premature contact between the proximal femur and acetabulum[1]. Although the concept of hip impingement was first documented in 1936[51], a recent surge in diagnostic rates has resulted in a growing prevalence of disease and greater recognition of its role as a potential risk factor for hip OA[17].

2.1.1 Economic Burden

While the direct economic impact of FAI from a societal perspective has yet to be quantified, its escalating prevalence has clinical implications on the development of hip OA, one of the most pervasive musculoskeletal disorders world-wide. In 2010, OA alone was associated with 10% of disability adjusted life years of all musculoskeletal conditions[52]. The estimated direct annual cost of arthritis in Canada has been stated as ~10 billion dollars, and indirect costs totalling ~17 billion[13]. Further, the Arthritis Alliance of Canada has stated that a new diagnosis of OA is made every 60 seconds, leading to 30% of our current labour-force reporting difficulties working due to OA[13].

Since the proposed link between FAI and OA was published in 2003, surgical interventions such as hip arthroscopy have been steadily increasing[53,54,55]. From 2004 to 2009, the rates of hip arthroscopies being performed in the United States of America has risen 365%[54]. The two most common treatment strategies for FAI include: (1) arthroscopic surgery, a less invasive surgical procedure proposing fast return to work rates, and (2) physiotherapy-led conservative care pathways. These two treatment strategies have been gaining notoriety as to which is not only better from a patient standpoint, but also

examining the economic impact of each treatment. Due to the relatively young age of individuals being treated for FAI and its associated functional limitations there is a high potential for treatment success, which is demonstrated by two thirds of individuals returning to work within five-weeks post-surgery[56]. Recent research has found that hip arthroscopy for the treatment of FAI outperformed conservative treatments from an economic standpoint, leading to an estimated 10-year savings of greater than $60,000 per surgery performed[56]. While the research demonstrating that arthroscopic surgery for FAI is economically superior, Canada's current healthcare system cannot keep up with the growing demand for orthopedic surgical procedures, resulting in less than 75% of individuals within Canada having a hip or knee replacement within the benchmark 182 days[57].

Improving our understanding of the mechanisms associated with FAI and its impact on the hip musculoskeletal system, more informed surgical interventions and targeted rehabilitation strategies may be developed to slow or alter the disease trajectory. Further, more evidence to better identify individuals most likely to benefit from surgical or non-surgical interventions to disrupt the disease process may have long-term implications on OA-related personal and societal burdens as well as support long-term alleviations for the current strain on the healthcare system.

2.1.2 Physical Burden

The World Health Organization's (WHO) International Classification of Functioning Disability and Health (ICF) provides a standardized health status framework that describes the functional status of an individual with some form of disability[58]. When examining FAI, impairments to the body include bone[1,4], cartilage[59,60] connective tissue[61]

and the neuromuscular system[32], with progression of these impairments resulting in self-reported activity limitations and reduced quality of life[21,34,35,36]. The impairments and physical limitations are also strongly linked to the development of hip OA, resulting in progressive worsening of impairments and further decline in functional limitations experienced by these individuals. Studying the interplay of these impairments may help guide the development and implementation of targeted strategies and interventions for improving the quality of life for individuals with FAI.

FAI is "described as a motion related clinical disorder caused by either a bony outgrowth on the femoral neck or an over-coverage of the acetabulum on the femur, leading to an abnormal articulation between the femoral head and acetabular rim"[1]. A study examining the clinical presentation of individuals with symptomatic FAI found that 37% of individuals reported limitations in walking distance, 71% reported the main aggravating factor was activity related, and 73% reported a mild limp when symptoms presented themselves[21]. However, the authors did not specify what was characterized as walking distance. The criteria may not have been strenuous enough to initiate walking limitations, which could explain why only 37% reported walking distance difficulties and may reflect an underrepresentation in this population.

2.1.3 Cam Type Morphology

Cam type FAI is caused by an aspherical femoral head abutting into the acetabulum[6,7,8,9]. Typically, this aspheric femoral head is caused by an osseous bump, which may begin to manifest itself on individuals during early maturation, mainly from high impact sports and activities[62,63,64]. This osseous bump results in a smaller femoral head-neck offset, defined as the widest diameter of the femoral head and the most

prominent part of the femoral neck[65]. Authors describe that this decreased femoral head-neck offset leads to an impingement between the boney prominence and the acetabular rim during movements requiring hip flexion, adduction, and internal rotation[7,28,65,66], leading to concentrated compression forces against the acetabular labrum and may result in tearing of the cartilaginous tissue[5,65]. Researchers have reported that hips with cam impingement most commonly have labral damage on the anterior superior area of the acetabulum and is accompanied by separation of the acetabular cartilage from the labrum[4]. This separation is caused by excessive compression and shear stresses, which cause the labrum to stretch outward while the cartilage is compressed into the joint resulting in a separation between the two structures. This type of morphology is more common in males[6,67,68], this finding stays consistent in athletic and non-athletic populations with 17% and 4% of males and females, respectively[67], and in elite athletes with 58% and 34% of males and females, respectively[73].

2.1.4 Pincer Type Morphology

Pincer type FAI is common in middle-aged females and caused by acetabular retroversion, anterior/lateral over-coverage of the acetabulum on the femur, and coxa profunda, all of which either deepen the acetabulum or increase the over-coverage on the femur[10]. These abnormal acetabular morphologies lead to a linear contact between the acetabular rim and femoral head-neck junction[70], wherein the femoral neck abuts against the labrum, leading to the transmission of forces from the labrum to the cartilage[4].

The damage caused by a Pincer morphology is typically more circumferential compared to cam morphology, where the damage tends to be restricted to a narrow strip of the acetabular cartilage, with consistent damage to the labrum in co-locations[4,71]. This

repeated microtrauma caused by pincer morphology can lead to a boney growth appearing at the base of the labrum, followed by ossification and subsequent increases in the acetabular over-coverage on the femur. In extreme cases, the abutment of the femur and acetabulum during extreme flexion can cause the femoral head to protrude posteriorly, causing damage to the posterior aspect of the joint[4]. Similar to cam type morphology, ROM restrictions in pincer morphology are described to mirror what has been previously reported, with restrictions in hip flexion, adduction and internal rotation[72].

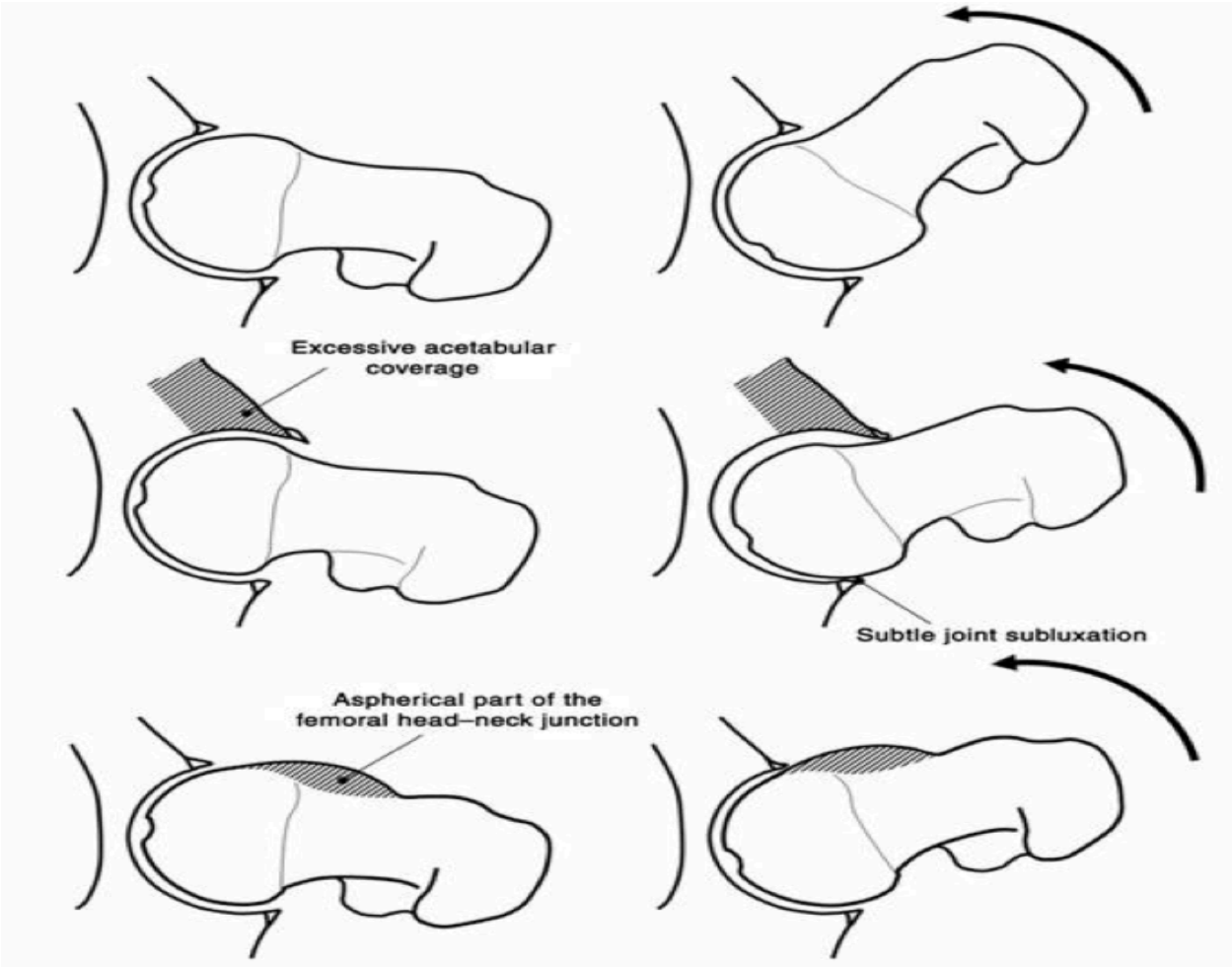

Figure 2-1: Illustration demonstrating hip flexion in a healthy hip (top), pincer impingement (centre), and cam impingement (bottom) (image modified from Tannast et al., 2006).

2.1.5 Signs and Symptoms

A consensus statement for signs and symptoms of FAI was established in 2016 during the Warwick agreement on FAI[1]. The primary FAI symptom is movement- or position-related pain, which typically manifests itself in the hip or groin, but has also been reported in the lower back, buttock or thigh of individuals[1]. This pain is characterized as intermittent motion related pain; however, in some cases of prolonged periods of sitting, pain can be transformed into a constant hip or groin ache[1,21,22]. Secondary symptoms to pain commonly reported by patients treated for FAI include clicking, catching, locking, giving way, or stiffness[1].

Diagnostic recommendations suggest that no one sign be used exclusively to diagnose the condition, but rather that a combination of several physical tests, as well as radiographic imaging be used to consolidate the diagnosis[1]. As symptoms begin to worsen, individuals tend to lose terminal ROM, typically in extreme hip flexion and internal rotation[23,24,25,26]. Therefore, this movement pattern is one of the most common assessments used by clinicians as an initial protocol for diagnosis and is referred to as the impingement test or FADIR test[23]. This test is performed with the patient supine, hip flexed to 90^{o}, then applying internal rotation and adduction to the joint. This joint position compresses the bony femoral outgrowth into the acetabulum thus increasing compressive and shearing stress. A second common test is the flexion, abduction, external rotation (FABER) test, which is performed with the patient supine and the leg put into a figure four position with flexion, abduction and external rotation, then a gentle force is applied downward onto the leg, while stabilizing the contralateral hip[23].

2.1.6 Cam Type Radiographic Signs and Symptoms

Hip radiographs are acquired in an anterior-posterior orientation, used to aid in the diagnosis of FAI and inform clinicians on the severity of the morphology. One of the most common measurements for detecting the severity of an asymmetrical femoral head and cam morphology is the alpha angle[28,29,30,31]. Notzli and colleagues (2002)[28] defined the alpha angle as the angle formed between a line from the midpoint of the femoral neck to the center of the femoral head, and a line from the center of the femoral head to the section where the bone first begins to deviate from the normal shape of the head. It has been described previously that alpha angles greater than 50^{o} are indicative of cam type morphology[70], however a study performed by Laborie and colleagues (2014)[69] stated that this cut-off value may be too low and may lead to false positives. As a result, researchers recommended a more conservative approach, and acknowledged recommendations from Sutter and colleagues (2012)[9], who suggested increasing the cut-off to 55-60^{o} to reduce false positives, while still maintaining sufficient sensitivity.

The femoral head-neck offset, and offset ratio are two other commonly used measurements to aid in an accurate diagnosis of cam type FAI[8,74,75]. The head-neck offset has been defined as the difference between the radius of the anterior femoral head, and the radius of the femoral neck[74]. Eijer and colleagues (2001)[74] described that the mean head-neck offset for asymptomatic individuals was 11.6 ± 0.7mm, and 7.2 ± 0.7mm for symptomatic individuals. The offset ratio is calculated by subtracting the femoral neck radius from the radius of the femoral head, divided by the radius of the femoral head[8]. Typical offset ratios for the asymptomatic population have been described as 0.21 ± 0.03, and 0.13 ± 0.05 for the symptomatic population[8,74].

2.1.7 Pincer Type Radiographic Signs and Symptoms

The center-edge angle is a common method for quantifying the severity of pincer type morphology and acetabular over-coverage. The center-edge angle is defined as the angle formed between a vertical line and a line connecting the femoral head center with the lateral edge of the acetabulum[7]. Reference values for these angles within a healthy control population have been reported between 23-33^{o}, whereas an individual with pincer type morphology and acetabular over-coverage may exceed angles of >39^{o}[7,76]. The center-edge angle has been found previously to have significant negative correlations with reduced stride length and hip flexion moments during gait[77].

Three other main radiographic assessments for diagnosing pincer type morphology are the crossover sign, ischial spine sign and posterior wall sign[78]. The crossover sign is considered positive if the anterior and posterior acetabular boarders crossed before meeting at the lateral border of the weight bearing zone[79], the ischial spine sign is considered positive if the ischial spine projects inside the pelvic cavity[80], and the posterior wall sign is classified as positive if the centre of the femoral head is lateral to the posterior acetabular border[81]. While these three radiographic signs are the most commonly reported diagnostic tools for pincer morphology in the literature, they have demonstrated moderate intra-observer reliability with correlation coefficients of 0.514, 0.543, and 0.633, respectively[81]. Diagnosing pincer type FAI morphology is more challenging than cam morphology[78], however, either diagnosis presents challenges due to the 2-dimensional nature of the radiographs being used for the diagnosis of the 3-dimensional orientation of the hip joint[78].

2.1.8 Etiology of Femoroacetabular Impingement

Currently the etiology of FAI is not well understood, however there have been a few proposed factors that may put an individual at an increased risk of development. The most commonly hypothesized cause for the development of FAI is physical activity levels, especially during maturation[62,63,82]. Siebenrock and colleagues (2011)[63] investigated a population of basketball players against age matched non-athletes to assess the prevalence of cam type morphology in the athletic population. Results showed that the average alpha angle within basketball players was significantly greater than the age matched nonathletes, 60.5^{o} compared to 47.4^{o} respectfully[63]. Further, the athletic population was found to have a 10-fold increase in hip alpha angles >55^{o}, compared to the age matched controls[63]. These cam type changes generally are initiated during skeletal maturation, preceding the fusion of the femoral epiphysis. Van Kilj and colleagues (2019)[82] found a high association between cam type development and open epiphysial plates in young athletes. Researchers found that open epiphysial plates led to a significantly increased risk of FAI development (OR: 10.02, 95%CI; 3.49-28.8) compared to the development of FAI once the epiphysis is closed. Similarly, Tak and colleagues (2015)[83] found that adolescent soccer players who practiced greater than four times per week prior to the age of twelve, had a significantly increased risk for the development of cam type morphology.

A possible reason for this morphology presenting itself during maturation may be due to a combination of over work and wolff's law, which states that bone tissues respond to the forces and loads placed upon them. A bone may remodel itself based on the external strains and stresses it encounters[84], and during maturation the skeleton is especially susceptible to these loads. This is evidenced by the femoral angle of inclination dropping

from 165-170° at birth to 125-130° at full maturation[85,86,87]. During this time of maturation and prior to the closing of the epiphysial plates, the femoral neck is very susceptible to over use, and the combination of over use and stresses during maturation when the skeleton is highly susceptible to mechanical loading[64], could lead to development of new bone, and a subsequent osseous bump as found in cam impingement.

Genetics, although less commonly described has been reported as a possible intrinsic risk factor for the development of FAI. Pollard and colleagues (2010)[88] investigated the risk of developing FAI for the siblings of individuals already treated surgically for the condition. Authors found that siblings had a relative risk ratio of 2.8 (95%CI; 1.8-4.2), which led them to conclude that there must be an underlying genetic predisposition[88]. The authors also noted that physical activity levels between siblings and controls was not significantly different, strengthening the argument that genetic factors may influence FAI development.

Figure 2-2: Pathway for the diagnosis and management of femoroacetabular impingement as outlined by the Warwick Agreement on femoroacetabular impingement syndrome (image modified from Griffin et al., 2016).

2.1.9 Femoroacetabular Impingement and Hip Osteoarthritis

OA is a highly prevalent musculoskeletal disease in Canada affecting ~17% of the population with estimates of affecting ~25% within the next 30 years[13]. Historically, OA has been considered a mechanical disease of the cartilage, yet recent research has shown that OA is a whole joint disease, affecting multiple structures including muscles, bone and cartilage[89]. OA is currently one of the leading causes of pain, and decreased function in older adults around the world[90,91,92]. A cure for OA remains elusive, therefore treatment strategies tend to focus on symptom reduction until eventual joint replacement at end stage

of disease. While the etiology of OA is incredibly vast, one commonly reported mechanical cause of hip OA is abnormal morphologies at the hip, and therefore recently linked with FAI pathology[14,15,16].

The hypothesis that mild femoral deformities could lead to the development of hip OA was originally proposed over four decades ago[93]. More recently, researchers have thoroughly described the underlying mechanical cause of the development of hip OA, which in the case of more severe hip morphologies, is excessive stress history on the cartilage following the reduction in contact area caused by the acetabular or femoral morphologies[5,94]. These increased and centralized joint stresses could lead to a progressive shearing and eventual avulsion of the cartilage. Continued abnormal articulations may progressively lead to accelerated deterioration of the joint's cartilage, and eventual onset of OA[5,94]. In pincer type, the damage tends to be a slower progression, but more symptomatic, and often leads to OA development in the anterior superior regions of the acetabulum. In the later stages of FAI disease, pincer type morphology damage begins in the anterior superior region, however, may also lead to degradation in the posterior inferior hip due to subluxation of the femoral head posteriorly. Conversely damage caused by cam type hips tend to be limited to damage only in the anterior superior region of the joint[4].

Cam type morphology has more recently been shown to be associated with increased risk of MRI assessed labral lesions (OR: 2.77, 95%CI: 1.31,5.87), and labrum deformity (OR: 2.45, 95%CI: 1.06,5.66) compared to those without a cam morphology[95]. Further, researchers found that the mean difference between the combined thickness of the anterior superior femoral and acetabular cartilage to be -0.19mm less in those with cam morphology compared to those without[95]. These anatomical changes may begin to explain

results from Hosnijeh and colleagues (2017)[15] who found individuals displaying a cam morphology to be twice as likely to develop OA (OR:2.11, 95%CI:1.55,2.87), while the same results were not found for pincer morphology (OR:1.24, 95%CI: 0.93,1.66). Similarly, when examining a sample of >1400 hips, it was found that a severe cam type morphology (alpha angle >83^{o}) at baseline was associated with a 9-fold increase in the chance of developing end-stage hip OA[20]. Second, a lack of internal rotation ability (<20^{o}), a common movement restriction associated with FAI pathology, was also strongly associated with a 7-fold increase in the chance of end stage hip OA development[20].

2.2 Biomechanics

2.2.1 Gait Analysis

Despite consistent patient-reported functional impairments and walking difficulty, our understanding of the potential underlying biomechanical mechanisms responsible for these movement related impairments is limited. Rutherford and colleagues (2018)[35] indicated that individuals with unilateral symptomatic FAI report moderate difficulties in walking long distances (44/100) with 100 indicating no difficulty and 0 indicating extreme difficulty. Despite individuals reporting difficulties, no kinematic or kinetic differences were found between individuals with FAI and an asymptomatic group[35]. Most studies focusing on differences in gait kinematics between individuals with FAI and asymptomatic individuals report minimal, if any differences between groups with regards to joint motions. Symptomatic individuals with FAI typically only report discomfort at extreme hip ROM, and typical level ground gait analysis may not be challenging enough to elicit any movement related disorders causing the reported physical limitations. Gait analysis has been defined as "the instrumented measurement of the movement patterns that make up

walking and the associated interpretation of these"[96]. The anatomical adaptations of the hip and the corresponding abnormal mechanical joint loading associated with FAI, have driven human movement research using gait analysis to identify potential biomechanical adaptations that may accompany the pathology.

2.2.2 Spatiotemporal Parameters

Differences in spatiotemporal parameters between individuals with FAI and asymptomatic individuals are inconsistent. Previous studies have reported that individuals with FAI walk significantly slower during gait[35,38], and with a reduced cadence[38]. Other researchers have stated no differences in gait speed, cadence, stride or step length between individuals with FAI and asymptomatic individuals[37]. Walking speed may be affected in individuals with more symptomatic FAI as a compensatory mechanism to reduce stress and torque on the joint, as increasing gait speed has been shown to be positively correlated with increased joint loading at the hip[97]. A possible reason for discrepant findings and varied differences in gait speed between symptomatic and asymptomatic individuals may be that gait speed has been highly correlated to center-edge angle and not the alpha angle[77], which is a more common diagnostic tool for pincer type impingement[70]; yet, the previously described studies primarily consist of individuals with cam impingement.

2.2.3 Sagittal Plane Kinematics

Gait analysis in individuals with FAI is a relatively new area of research, however findings to date suggest minimal kinematic differences between individuals with FAI and asymptomatic individuals during level ground walking[35,37]. Asymptomatic individuals age matched with an FAI population have previously demonstrated sagittal plane hip ROM values during walking between 36º during stance and 48º during the entire gait cycle[35,37].

Peak hip flexion and extension values have been previously reported around 31°[37,38] and 11° to 12°[38,37], respectively.

In a FAI cohort comprised of 11 cam and 4 mixed type morphologies, Diamond and colleagues (2016)[37] found a reduction of 4° sagittal plane hip ROM during gait, however, authors question the clinical significance of this finding. Reductions in sagittal plane hip kinematics were also found in a sample of 22 cam, 2 pincer and 6 mixed type morphologies by Hunt and colleagues (2013)[38] who reported sagittal plane limitations; however, they were caused by a reduction in peak hip extension during pre swing. These results are conflicted by Rylander et al (2013)[39], who in a sample of 3 pincer and 14 mixed type morphologies also found a reduction in sagittal plane hip ROM; however, hip flexion was limiting, and demonstrated by a reduced peak hip flexion angle during stance. Hunt and colleagues (2013)[38] hypothesized that the limited hip extension may be due to tension within the anterior hip's soft tissue, leading to increased compression within the anterior portion of the acetabulum, with the resulting discomfort leading to the extension reduction. Conversely, researchers did not speculate as to why a reduction in peak hip flexion was present, although a possible hypothesis may be pattern avoidance to minimize hip impinged positions (i.e., increased hip flexion and adduction)[39]. This theory; however, should be considered with caution, as peak flexion angles during level ground walking are less than flexion angles recorded during passive ROM[38].

Musculoskeletal models suggest that increasing hip extension angles increases anterior hip compression forces, and limited hip extension has been reported for individuals with anterior hip pain[98]. Hip extension is the primary close packed position of the hip, during which the capsular ligaments of the hip become taut and the hip flexor musculature

act to limit this motion. The femoral head is pulled anteriorly into the acetabulum, where joint damage is often reported[4], possibly supporting findings from Hunt and colleagues (2013)[38], and consistent with the reduction of hip extension during gait within this population.

Sagittal plane hip kinematics commonly reported in the literature for individuals with FAI appear to parallel findings reported for individuals with hip OA. Individuals with mild to moderate hip OA walk with reduced peak hip extension and reduced total sagittal plane hip ROM during stance[99,100,101]. The reductions in sagittal plane hip motion, specifically during hip extension, may be a learned response to limit loading in the anterior portion of the joint.

In summary, individuals with FAI typically walk with altered sagittal plane hip kinematics compared to asymptomatic individuals, however, results are inconclusive. Both reduced peak hip extension and peak hip flexion have been reported during walking in individuals with FAI. While hip flexion is a position where individuals typically exhibit impingement, it is unlikely that the hip moves through enough flexion during gait to cause pain from impingement. Peak hip extension however has also been reported as the limiting factor to sagittal plane hip ROM[38]. This limitation may be a compensatory mechanism to limit the closed packed position of the joint. As estimated using musculoskeletal modeling, hip extension results in higher compression forces in the anterior portion of the acetabulum, being the most common position for cartilage damage[98]. A deeper investigation into tasks putting the joint through more ROM, or closer to an impinged position may give researchers a better understanding and elucidate more biomechanical differences between individuals with FAI and asymptomatic individuals.

2.2.4 Frontal Plane Kinematics

Frontal plane kinematics during walking in an asymptomatic population who are age matched with individuals with FAI have demonstrated frontal plane ROM values of 9° to 10° [35,38] during stance and upwards of 17° during the entire gait cycle[37]. Peak adduction values have been previously reported to range from of 6° to 10° [37,38] during stance, with peak abduction values of around 2° during stance[39,38].

Hip adduction is another impinging position for individuals with FAI[21]. The hip generally allows for approximately 25° of adduction, however, with FAI this range may be limited to 15-20°[21]. During walking, individuals with FAI have shown 9-11° of peak adduction during gait[37,38,39], which is much closer to an end range where the articulating femur and acetabulum would abut against one another. Therefore, kinematic compensations in the frontal plane would be hypothesized for individuals with FAI suggesting that symptomatic individuals may develop an alternative gait pattern to avoid excessive adduction during walking. This hypothesis is supported by Hunt and colleagues (2013)[38] who reported reduced peak hip adduction during stance. Importantly, these results are not conclusive as other authors have reported contradictory findings during gait such as decreased hip abduction during stance[38,39,27,42]. These differences included a reduced peak hip abduction angle and reduced total frontal hip ROM[36,39,27]. Authors speculated the reduced hip abduction was not associated with bony hip impingement and perhaps a more complicated mechanism such as reduced hip abductor strength may exist in individuals with FAI[37,40]. Further, several authors have reported no differences in frontal plane kinematics between individuals with and without FAI[35,60,102].

In summary, frontal plane hip kinematic differences between individuals with FAI and asymptomatic individuals are inconclusive and results are contradictory. Reductions in frontal plane hip abduction may be a result of reduced peak hip abduction during stance or a reduced hip abduction angle at initial contact, which may be associated with less hip abductor strength previously reported in the literature[40,41]. Putting the joint in a more challenged position, and more difficult tasks that test the strength of the hip abductors, along with investigations that quantify neuromuscular control may provide additional information to clarify the role of muscles during this movement pattern or highlight if a more complicated mechanism is responsible.

2.2.5 Transverse Plane Kinematics

Hip adaptations in the transverse plane are also reported in the FAI literature. Comparable to inconsistent messaging in the sagittal and frontal planes, conflicting results suggest possible reductions in total transverse plane ROM[39] or a reduced peak internal rotation angle during gait[38,38], while other authors report no differences in the transverse plane between individuals with and without FAI[35,37,42]. Overall transverse plane ROM values for asymptomatic age matched individuals may range between 10^{o} to 14^{o} [35,37,39], with 8^{o} to 11^{o} observed for peak hip internal rotation[38,39], and 3^{o} to 7^{o} observed for peak hip external rotation during stance[38,39].

Similar to hip adduction, internal rotation ROM during gait approaches maximal passive end ROM for individuals with FAI (15-25^{o})[21]. Associated symptoms may elicit a learned gait response to modify walking patterns and limit impingement-like positions. This functional loss of transverse plane hip ROM has also been noted within hip OA populations. Rutherford and colleagues (2015)[103] reported a reduced peak internal rotation

in a severe hip OA group compared to moderate hip OA and asymptomatic individuals, as well as a reduced total transverse plane ROM throughout gait compared to the asymptomatic group. These movement similarities suggest a possible link between the underlying biomechanical mechanisms of FAI and hip OA. Interestingly, however, the transverse plane changes were not observed for individuals with moderate hip OA compared to asymptomatic individuals, highlighting the potential spectrum that individuals with FAI may be display gait changes in the transverse plane.

2.2.6 Sex Differences

Gait differences during level ground walking have previously been observed between asymptomatic males and females[104,105]. Asymptomatic females are suggested to walk with increased hip flexion[104], adduction[104,105] and internal rotation compared to asymptomatic males[104]. Several authors[104,105] proposed that these differences were likely associated with increased anterior pelvic tilt and excessive pelvic motion in the frontal plane.

Studies examining sex-related biomechanical differences in individuals with FAI are limited; however, few potential sex differences have been observed[106,107]. It has been found that males with FAI walked with an average of 4° less peak hip extension and less then 1° difference in peak hip flexion then the females with FAI[106]. In the frontal plane males with FAI walked with 6° peak hip adduction and 1° more hip abduction then females with FAI[106]. Although not directly compared, these hip kinematic changes may be primarily influenced by alterations in pelvic motion, as the differences seen in peak thigh extension differed by only 1° between males and females with FAI[106]. Biomechanical, sex-related investigations in individuals with FAI have also been reported during a single leg

stepdown task[107]. At 60° of knee flexion during the task, females with FAI had approximately 6° more hip flexion and 5° more hip adduction than males with FAI, suggesting that movement differences exist between males and females during walking as well as other dynamic activities and may contribute to disease related structural impairments[107].

Males with FAI had increased anterior pelvic tilt during static standing, which was also observed during squatting and stair climbing[108,39]. Males and females with versus without FAI walked with decreased peak hip extension but was notably associated with the symptomatic limb in females only. Additionally, no differences were observed in anterior pelvic tilt between females with and without FAI, which is consistent with observations that asymptomatic females walk with increased anterior pelvic tilt compared to males[104]. Increases in anterior pelvic tilt could increase the likelihood for impingement between the femur and acetabulum resulting in reduced hip flexion ROM and earlier impingement. Due to this earlier impingement during hip ROM it would be thought that increases in anterior pelvic tilt may increase damage of the joint and possibly accelerate the damage process leading to hip OA. It has been thought that this increase in anterior pelvic tilt may be due to reduced hip extensor activity, specifically the gluteus maximus or hamstrings during these movements[108].

2.3 Strength

Reductions in size and strength of the hip musculature have been found in individuals with hip pathology such as hip OA[109]. While current research indicates that FAI is a precursor to hip OA, it is currently unknown whether these muscular deficits are present in individuals with FAI prior to the development of OA, as research examining hip

strength deficits within the FAI population are also inconsistent. Castillari and colleagues (2011)[40] showed that individuals with FAI were significantly weaker in isometric hip adduction, abduction, external rotation, and isokinetic hip flexion, whereas Diamond and colleagues (2016B)[41] found that only isometric hip abduction strength was reduced in individuals with FAI compared to asymptomatic individuals. More recently, Kierkegaard and colleagues (2017)[110] showed that individuals with FAI had reduced isometric, concentric and eccentric hip flexion strength, as well as reduced isometric and concentric hip extension strength in their affected compared to their contralateral limb, and asymptomatic individuals. There has also been research indicating that these muscular weaknesses are bilateral in individuals with FAI. Rutherford and colleagues (2018)[35] found reduced isometric hip flexion/extension and adduction strength bilaterally compared to asymptomatic individuals. The participants in the study had unilateral symptomatic FAI, however the alpha angle found within the asymptomatic limb was found to be 71°, which would radiographically diagnose them with FAI. Interestingly, this finding suggests that without symptoms, the structural changes associated with cam type morphology may lead to reduced strength of the hip.

Reductions in muscular strength can have detrimental effects to joint movement and stability, specifically reduced hip abduction strength in this population. During single leg stance, and activities requiring single leg support, the weight of the trunk, arms and head are displaced laterally from the body's base of support, causing the pelvis to rotate toward the contralateral side, thereby adducting the hip and bringing the weight of the upper body more in line with the base of support[87,111]. To counteract this external hip adduction moment, the hip abductors must generate a substantial internal hip abduction

moment to stabilize the hip joint[87,111]. Reductions in hip abduction strength can therefore lead to excessive frontal plane movement of the pelvis and femur, leading to impingement of the two structures and subsequent accelerated joint damage.

2.4 Electromyography

EMG quantifies the electrical activity of the muscles and provides an indirect measure of neuromuscular activity. Neuromuscular activity is proposed as a key component during joint loading, therefore better understanding neuromuscular control during walking can give insight into joint-specific mechanics and response to activity.

2.4.1 Muscular Activations During Gait

During gait, muscles surrounding the hip actively stabilize the joint and support its movement through ROM; however, they also alter loading patterns within the hip, specifically by increasing compressive forces[112]. For example, in a young asymptomatic population, peak and mean MVIC normalized activation of vastus medialis was reported to be approximately 19% and 5% MVIC, 21% and 6% MVIC for vastus lateralis, 12% and 4% MVIC for rectus femoris, 10% and 4% MVIC for medial hamstrings, 11% and 3% MVIC for lateral hamstrings, and 47% and 16% MVIC for Gmed, respectively[113]. Consistent with FAI pathological processes, increases in compressive forces in the hip may lead to accelerated degradation of the joint's tissues, reductions in strength, less stability and further compression[112]. Reduced muscle forces in individuals with FAI have been highlighted by Ng and colleagues (2018)[114] who reported reduced psoas major and iliacus forces during contralateral initial contact. Contrary to previous work, these authors speculated that this force reduction was a compensatory mechanism to reduce loading in the anterior femoroacetabular compartment during terminal hip extension[114].

Limited research has utilized EMG analyses to quantify neuromuscular control in individuals with FAI; however, few studies exist and suggest different EMG findings between individuals with and without FAI[35,43]. Rutherford and colleagues (2018)[35] reported in an exclusively cam morphology sample, differences in muscular MVIC normalized amplitudes during gait, with Gmax having an overall larger normalized amplitude within the FAI population. This increase in gluteal activation may be a compensatory mechanism to reduce joint loading in the anterior portion of the hip joint, as it has been reported that decreasing gluteal muscle activity during hip extension acts to increase anterior joint forces in the hip[98]. Although limited studies exist for comparing this finding across different FAI populations, similar results have been reported in individuals with hip OA. Rutherford et al (2015)[103] reported increased Gmax activation throughout stance within a severe hip OA population compared to controls. Comparing results between these two studies should be cautioned due to differences in EMG normalization techniques; however, similar findings do suggest a possible consistent compensatory technique between the two populations.

The hamstrings also function as a hip extensor during walking. Evidence of altered hamstring activation during gait within the FAI population is limited, yet few notable differences between activation patterns have been reported. Rutherford and colleagues (2018)[35] observed an increased overall MVIC normalized amplitude in the MH compared to LH in individuals with FAI, a finding that was not observed for asymptomatic controls. No differences were found in amplitude between individuals with FAI and controls, or symptomatic and asymptomatic legs of the FAI population, for the Gmed or RF[35]. Additionally, a solitary study performed by Diamond and colleagues (2017)[43] examined

muscle synergies of the deep external rotators[43]. Results showed that deep external rotators (e.g., obturator internus and quadratus femoris) of individuals with FAI were more highly activated during the early swing phase of gait compared to asymptomatic individuals. Authors speculated that this observation may be a compensatory mechanism that individuals with FAI perform prior to moving the hip into a flexed position[43].

In summary, EMG research regarding neuromuscular control of the lower limb in the FAI population is lacking, but the evidence that does exist suggests that altered muscular coordination and activation is varied in individuals with FAI and warrants future work. The literature suggests greater activation amplitude of the Gmax, greater MH – LH ratio during stance and altered hip external rotator coordination during early swing. Continued research into neuromuscular activations during walking and more challenging tasks may better elucidate muscular deficiencies, or coordination techniques employed by individuals with FAI to avoid pain, or impingement.

2.5 Inclined Walking

Previous investigations of individuals with FAI suggest that level ground walking may not provide enough challenge during movement to elicit biomechanical differences in this younger, and often more active population[35,37]. The restrictions and symptoms that exist at end ROM when performing passive and active ROM testing may contribute to the lack of, or consistency in, significant findings previously reported[21]. Typically, the hip moves through an arc of 40-50^{o} of hip sagittal ROM within an asymptomatic population, with peak hip flexion at ~35^{o}, and peak hip extension at ~10^{o}[65]. Therefore, common level ground walking may not elicit enough ROM to stress individuals with FAI.

2.5.1 Sagittal Plane Kinematics

Inclined walking has emerged as an approach to challenge an individual's walking by increasing stress on the cardiovascular system, as well as increasing the tri-planar ROM patterns at the hip[44], and potentially increasing neuromuscular demands[48]. Sagittal plane hip differences are the most commonly reported outcome during inclined walking. Previous research has documented that hip flexion at initial contact progressively increases with greater gradients (Figure 2-3)[44,45,46], and a similar trend exists at the knee having a progressively larger knee flexion angle at initial contact during increasing inclinations[44,45,46]. Using inclined walking, individuals with FAI may be more challenged, enabling pathological-driven, biomechanical adaptations to be observed that may not be detected during level ground walking.

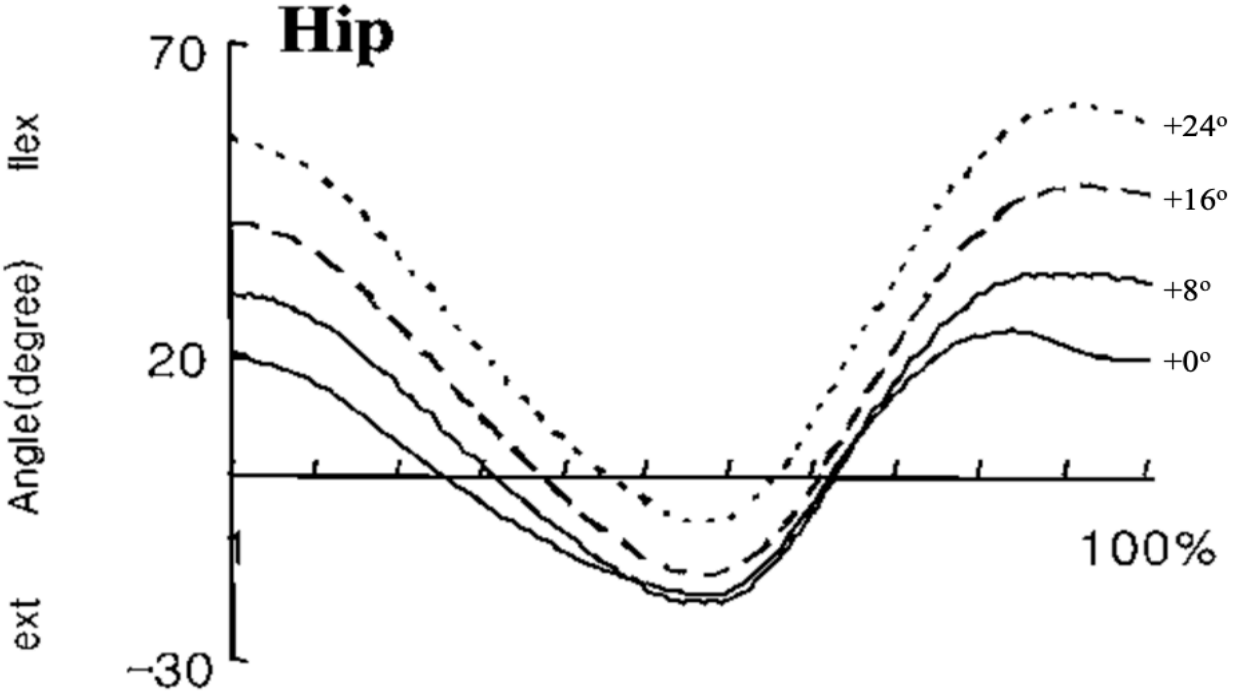

Figure 2-3: Sagittal plane hip motion during a gait cycle during four inclination levels (image modified from Han et al., 2009).

2.5.2 Frontal and Transverse Plane Kinematics

Inclined walking has also been shown to elicit changes in the frontal plane of asymptomatic individuals during gait[44,47] including increased hip adduction angles at initial contact. Haggerty and colleagues (2014)[44] found differences at 5% and 10% grades, while Han and colleagues (2009)[47] reported differences at 8° and 16°. Haggerty et al (2014)[44] speculated that this increased hip adduction at initial contact may be attributed to the center of mass (CoM) being shifted more toward the ipsilateral side during initial contact, to allow for unloading of the contralateral limb as it prepares for toe clearance during swing. Similarly, research examining data in the transverse plane is limited as the vast majority of research on inclined walking focuses on sagittal plane mechanics. Review of the literature found only one study which reported transverse plane kinematics during inclined walking[47]. Authors reported increased internal rotation at initial contact inclinations of 8° and 16° compared to level ground; however, did not comment on the significance of this finding[47].

Although understanding possible differences in the frontal and transverse planes during inclined walking is limited, the results reported do support the validity of observing these movements in individuals with FAI. The finding that inclined walking will influence an asymptomatic individual's frontal and transverse plane kinematics, specifically toward that of adduction and internal rotation does give support to the challenges individuals with FAI would face while walking on an inclined surface. As has been stated earlier, individuals with FAI tend to walk with less peak hip internal rotation than asymptomatic individuals, therefore, inclined walking may be a way to elicit a greater biomechanical response or alteration to an FAI gait pattern.

2.5.3 Neuromuscular Activations

During the first half of inclined stance, the hip extensors experience more demand to extend the leg and propel the body up the incline due to the increase in hip flexion, which has been observed through increased activation of the Gmax[48,50], as well as increased hamstring activation during pre swing[48,49,50]. Franz and Kram (2012)[48] examined mean muscular activation amplitude, normalized to percent of mean activation during level ground walking for the RF, VM, medial gastrocnemius (MG) and soleus (SOL) muscles. A 9^{o} incline resulted in a mean Gmax activation increase of 345% compared to activity during level ground walking, and a 635% increase in bicep femoris (BF) activation compared to level ground walking[48]. Additionally, minimal focus has been directed to muscles controlling frontal plane movement of the hip. Wall-Scheffler and colleagues (2010)[115] examined mean activation of Gmed and the hip adductors, normalized to peak activation during level ground walking, demonstrating increased activation of both muscles[115]. Similar to joint angles, the activation of these muscles tends to increase as the grade of the walking surface increases[48].

To date, no research exists that quantifies human movement during inclined walking in individuals with FAI, therefore improving our understanding of the biomechanical mechanisms during challenging tasks is warranted. Diamond and colleagues (2016)[37] suggested that level ground walking is unlikely to elicit any meaningful biomechanical changes compared to asymptomatic individuals and recommended that future research activities involve tasks that put the hip in a more impinged position. Inclined walking has been shown to increase hip flexion and adduction[44,45,46]. Currently, it is unknown how individuals with FAI will adapt to inclined walking; however, the

combination of increased hip flexion and adduction recreates a possible position of discomfort for individuals with FAI. This in combination with increased hip extensors activity, may generate a scenario that challenges the joint, and elicit meaningful changes in joint kinematics and EMG activity.

2.6 Declined Walking

2.6.1 Sagittal Plane Kinematics

To our knowledge, the few studies examining gait analysis during declined walking have only reported kinematic changes in the sagittal plane, with no investigations in either the frontal or transverse planes at the hip. Research into hip joint kinematics during declined walking is lacking; however, the limited research does suggest an opposite effect on sagittal plane hip kinematics compared to inclined walking. These hip flexion angles at initial contact during declined walking are significantly reduced, with a difference of 29^{o} being found between declined walking at -8.5^{o} and inclined walking at +8.5^{o}[45]. The hip also goes through a limited sagittal plane hip ROM during stance when walking on a decline. When walking at -8.5^{o} Lay and colleagues (2006)[45] found that the hip moved through 26^{o} ROM during stance, compared to +8.5^{o} where the hip moved through nearly 55^{o} sagittal plane hip ROM. This reduced hip flexion during declined walking has not been reported as significantly different compared to level ground walking[116]. These kinematic hip differences were most pronounced during swing and early stance phases. Interestingly, authors noted a hip flexion adjustment during early stance, after which similar motions at the hip were seen compared to level walking.

The main kinematic differences during downhill walking are found at the knee, primarily in the sagittal plane, characterized with increased knee flexion during weight

acceptance and midstance. The differences in sagittal knee motions during declined walking at approximately 11°, led to a 20° increase in knee flexion during midstance compared to level walking[116].

2.6.2 Muscular Activations

While inclined walking puts pressure on the hip extensors to propel the individual up an incline, declined walking has been reported as having the opposite effect, and required the hip flexors and knee extensors to slow down the individual's forward momentum resulting in increased activity for VM[48,50] and RF[48,50]. The contribution of the knee extensors during declined walking is significantly elevated, with findings that activation of the RF and VM muscles amplitude normalized to mean activation during fast (1.75m/s) level ground walking displayed increases of 310% and 243% respectfully.

From the hip extensor muscles, there is little to no difference in mean magnitude from the extensors (Gmax, MH, and LH) during declined walking compared to level ground[50]. With the increase in breaking force required to safely ambulate down a slope, authors hypothesized that the multi-joint RF may be preferentially activated during downslope walking; however, this was not the case, as VM and RF magnitude increased nearly identically throughout the stance phase of gait[50]. The authors speculated that increased knee shock absorption during declined walking may alter hip joint muscle recruitment during this challenging task. However, the single joint hip flexor muscle iliopsoas was not collected in the study and may have given a more holistic view of hip muscular activity during declined gait.

Altered patterns in neuromuscular activity may provide new information associated with increased hip joint compression during both inclined and declined walking[117].

Although the primary adaptations during declined walking appear to occur at the knee with increased knee extensor activation, there is limited research on hip neuromuscular control during declined walking and to our knowledge no investigations in individuals with FAI. Yang and colleagues (2019)[51] have found that during declined walking the hip had significantly increased negative joint work (i.e., energy absorption), compared to level ground walking in the frontal and sagittal planes. During a 12^{o} declined trial, the hip had a 193% increase in frontal plane negative joint work, and a 164% increase in sagittal plane negative work[51]. These results indicate the significant contribution of the hip during declined walking and illustrates that EMG-driven research focused on neuromuscular activity at the hip requires further investigation.

Chapter 3 - General Methodology

This study is funded by the Nova Scotia Health Research Foundation (NSHRF). Recruitment, data collection protocols and analysis procedures were approved by the Nova Scotia Health Authority (NSHA) Research Ethics Board (REB file number: 1024401). To date, this study has been suspended due to COVID-19 restrictions and is expected to return when research at Dalhousie University resumes.

3.1. Participant Recruitment

3.1.1 Participants with Femoroacetabular Impingement

Both male and female patients diagnosed with FAI and candidates for arthroscopic hip surgery were recruited from Dr. Ivan Wong's Orthopaedic and Sports Medicine Clinic at the QEII Health Science Center. A clinical diagnosis was based on a triad of signs and symptoms outlined in the FAI Warwick Agreement[1]. Signs and symptoms included pain induced with hip flexion/abduction/external rotation, groin pain with hip flexion, and sharp pain with flexion and internal rotation. Radiographic evidence included an alpha angle >55^{o} for cam type, or acetabular retroversion for pincer type morphologies. Patients were not eligible to participate if they were less than 18 years of age, had radiographically defined hip OA classified as a Kellgren and Lawrence grade of ≥ 2[118], inflammatory arthritis in either limb, prior surgery in either limb or bilateral FAI symptoms. Eligible participants were approached by Dr. Wong using a standardized introduction to the research study and were given a letter with information about the study objectives. Interested participants provided written consent for a transfer of contact information. This information was used by the research team to contact the potential participants by telephone using a standardized script to determine eligibility for the study. The standardized script determined the presence

of cardiovascular, neuromuscular, inflammatory or musculoskeletal conditions that would affect their gait, ability to climb stairs or complete the study as well as confirm the diagnosis of unilateral FAI. If eligible, the participant was given details of the visit and a data collection was scheduled.

3.1.2 Sample Size

To our knowledge, inclined and declined gait analyses have not been performed in individuals with FAI. Due to the novel, pilot design of the current study, no power calculation was performed, and a predetermined sample size of ten individuals with FAI and ten asymptomatic individuals was estimated. Data from the current study will be used in a post hoc power analysis to determine sufficient sample size required for future investigations.

3.2 Procedures

3.2.1 Participant Preparation

All testing related to the current study took place in the Joint Action Research Laboratory, School of Physiotherapy at Dalhousie University. A brief description of the lab and its equipment was provided to all participants when they arrived. Participants were asked if they had any questions related to the letter of information prior to providing written informed consent. A series of questionnaires related to health-related quality of life, physical function and symptoms were completed by all participants prior to data collection. The Questionnaires included the International Hip Outcome Tool (iHOT-33)[119], containing subdomains on symptoms and functional limitations, sports and recreational activities, job related concerns, and social, emotional and lifestyle concerns. The iHOT-33 has shown good reliability with an intraclass correlation coefficient (ICC) of 0.78[119]. The Hip

Outcome Osteoarthritis Score (HOOS) measures hip pain, other symptoms, activities of daily living, sports and recreation, and quality of life. The HOOS has good validity, demonstrated by correlations of 0.44-0.66 with other health-related questionnaires such as the Short-Form-36[120]. The EQ5D[121], Veterans Rand (VR-12) for health-related quality of life, Cronbach's Alpha (0.95)[122], and the Non-Arthritis Hip Score (NAHS) have good-to-excellent test retest reliability of 0.87-0.95[123]. After completing the questionnaires, participants were asked to change into tight fitting clothing and to remove their footwear. Anthropometric measurements were taken for each participant including height and weight, and waist, hip, thigh and shank circumferences.

3.2.1.1 Surface Electromyography

Participant set-up and data acquisition for surface EMG followed a standardized protocol as described by the Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM) guidelines. Participant preparation included light shaving and cleaning using 70% alcohol wipes in the measurement area at each muscle site (Table 3-1). Consistent with guidelines from the International Society of Electromyography and Kinesiology[124], Ag/AgCL surface electrodes (10mm diameter, 30mm interelectrode distance) (Red Dot; 3M Health Care) were placed bilaterally in a bipolar configuration over the muscle sites[124] (Table 3-1). EMG signals were recorded at 2000Hz using an AMT-8 (Bortec, Inc., Calgary, Alberta, Canada) EMG system with gains of 100-5000x (Input impedance of ~10 GΩ, CMRR:115 dB at 60Hz, Band-pass 10-1000Hz) in order to maximize the signal without reaching signal saturation. After approximating each muscle site, a series of isometric muscle contractions were used to verify that electrodes were placed over the corresponding muscle belly. Once locations were verified, electrodes were

placed parallel to the underlying muscle fibers. Lead wires with 500x pre-amplification were connected to the electrodes over each muscle, with a single ground electrode being placed on the tibial shaft. Electrodes were tested for signal quality, and the proper gain adjustment following manual muscle testing. The quadriceps signals were tested by a standing maximal contraction of the quadriceps, hamstrings were tested using resisted knee flexion, Gmed signal was checked using resisted hip abduction, and Gmax signal was checked via a standing maximal glute contraction. Gain amplification for the EMG signal was set between 100-5000x for each muscle to maximize amplitude.

Table 3-1: SENIAM guidelines for the standardized electrode placement of the lower limb.

Muscle	**Muscle Site**
Vastus Medialis (VM)	80% of the distance between the ASIS and the joint space in front of the anterior MCL
Vastus Lateralis (VL)	2/3 of the distance from the ASIS to the lateral side of the patella
Rectus Femoris (RF)	50% of the distance between the patellar base and ASIS
Medial Hamstrings (MH)	50% of the distance between the ischial tuberosity and the medial epicondyle of the tibia
Lateral Hamstrings (LH)	50% of the distance between the ischial tuberosity and the lateral epicondyle of the tibia
Glutes Maximus (Gmax)	50% of the distance between the 2nd sacral vertebrae and the greater trochanter
Glutes Medius (Gmed)	50% of the distance from the iliac crest to the greater trochanter

Note: ASIS – anterior superior iliac spine; MCL – medial collateral ligament

Following EMG electrode placement, participants were asked to walk barefoot along the GAITRite™ portable pressure sensitive walkway at their self-selected speed at least 15-20 times. Five trials were randomly selected, and their walking speed averaged to determine the participant's self-selected walking speed. The treadmill speed was set to match the self-selected walking speed for the duration of the walking protocol. The GAITRite™ system has previously been shown to be a valid[125] and reliable[126] tool for calculating walking speed (ICC > 0.9).

3.2.1.2 Motion Capture

An eight-camera Qualysis Oqus 500 (Qualysis, Gothenburg, Sweden) motion analysis system was used to track three-dimensional joint motion data. Ridged plastic plates containing clusters of four retroreflective markers were placed bilaterally over the trunk, pelvis, lateral femur, lateral shank and feet, and secured using Velcro straps. The marker clusters placed over the lateral thigh were positioned in the middle of the thigh on each participant to try and limit differences in skin movements found at the proximal and distal ends of the thigh. Single retroreflective markers were placed on the spinous process of the seventh cervical vertebrae and bilaterally over the shoulders just below the acromion, greater trochanters, medial and lateral tibial and femoral epicondyles, lateral and medial malleoli, first, second, and fifth metatarsal heads and posterior heels. Marker placement is illustrated in Figure 3-1.

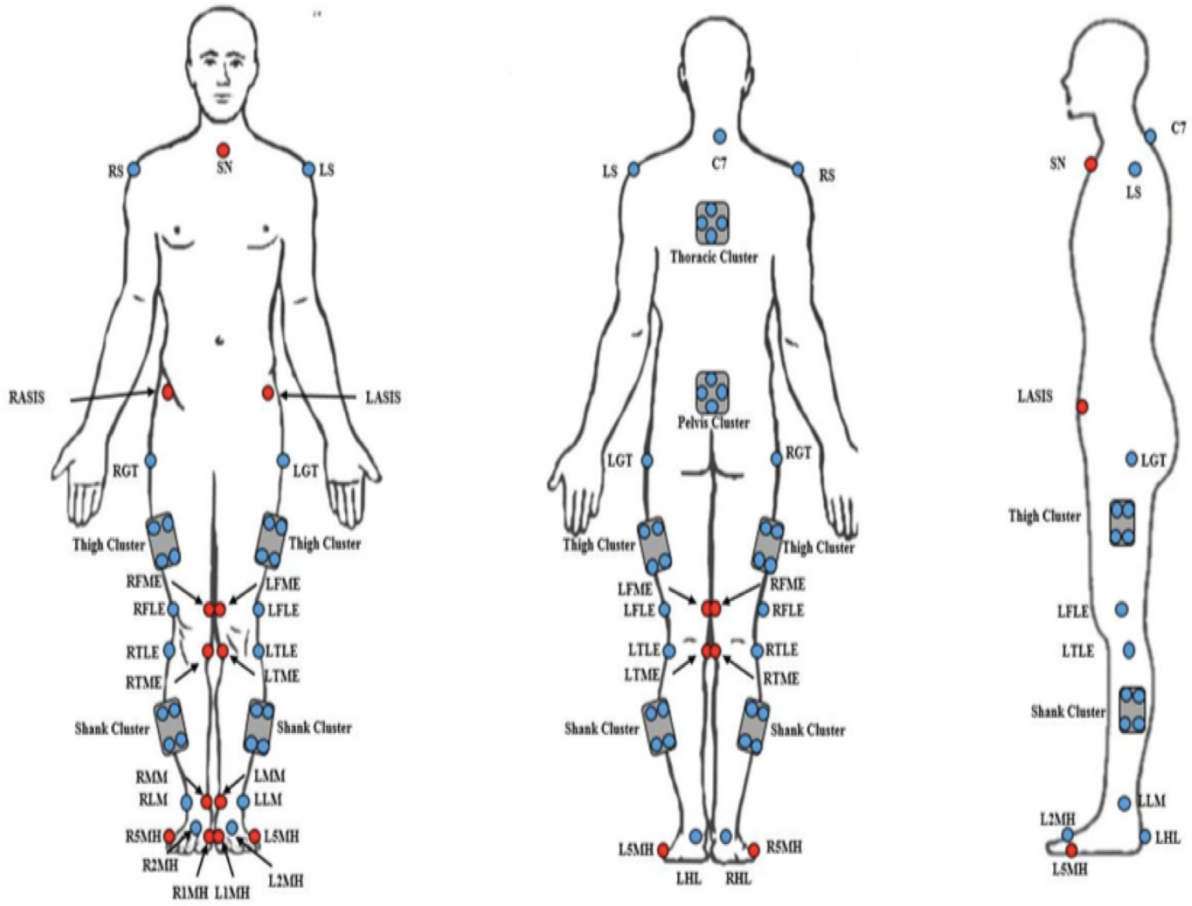

Figure 3-1: A figurative representation of the three-dimensional marker set used for the current study. Individual circles represent each marker. Single blue circles represent individual markers placed over specific bony landmarks including: right (RS) and left (LS) shoulders, 7th cervical vertebrae (C7), right (RFLE) and left (LFLE) femoral lateral epicondyles, right (RTLE) and left (LTLE) tibial lateral epicondyles, right (RLM) and left (LLM) lateral malleoli, right (R2MH) and left (L2MH) 2nd metatarsal heads, and right (RHL) and left (LHL) heels. Grey squares combined with four blue circles represent the rigid marker clusters placed bilaterally on the mid-thigh, mid-shank and foot. The red circles represent virtual markers including: right (RASIS) and left (LASIS) anterior superior iliac spines, right (RGT) and left (LGT) greater trochanters, right (RFME) and left (LFME) femoral medial epicondyles, right (RTME) and left (LTME) tibial medial epicondyles, right (RMM) and left (LMM) medial malleoli, right (R5MH) and left (L5MH) 5th metatarsal heads, and right (R1MH) and left (L1MH) 1st metatarsal heads.

3.2.2 Walking Protocol

3.2.2.1 Calibration

Prior to the walking protocol, participants were asked to remove their shoes for walking, if they were comfortable walking on a treadmill and to determine whether the use of a harness was necessary. A two second standing calibration was then taken on the R-Mill dual-belt instrumented treadmill (Motekforce Link, Culembord, the Netherlands), with the participant standing forward with their feet shoulder width apart and their knees as straight as possible. After the standing calibration, markers located at the medial femoral and tibial epicondyles, medial malleoli, the first and fifth metatarsal heads, lateral tibial epicondyles and greater trochanters were removed. Virtually digitized points were collected for two seconds to define both anterior superior iliac spines (ASISs) and sternal notch using a pre-calibrated digitizer wand.

3.2.2.2 Warm up

Prior to walking, a standardized explanation of the protocol was provided for each participant, including instructions to focus on a point straight ahead of the treadmill to keep their gaze from shifting. Once the participant felt comfortable, the researcher told the participant when the treadmill was going to begin to move at their self-selected walking speed previously determined using the GAITRite™ system. A six-minute level ground walking trial was completed for the participant to acclimatize to the treadmill and lab environment and help with minimizing variability between strides[127] and EMG measures[128]. Participants were reminded to walk with one foot on each belt and to minimize crossover as able. Using the treadmill handrails was not permitted during testing, but in the event that the participant felt unstable or unsafe handrail use was encouraged for the

participant to regain their comfort. During the sixth minute of the walking warm-up, a 20-second baseline walking trial was collected as the level ground condition prior to initiating the inclination protocol.

3.2.2.3 Inclination Protocol

The inclination protocol consisted of four randomized inclined and declined walking conditions including: 5° and 10° of inclined walking, and 5° and 10° of declined walking. A balanced Latin square design was used to randomize the walking conditions[129]. This design was intended to remove the possible effect of condition order on outcomes by allowing each walking condition to be followed by another condition only once. The four possible walking condition combinations are listed in Table 3-3. Using a random number generator, participants were randomly assigned to one of four walking condition combinations prior to their arrival and were blinded to the order of the protocol. Participants walked for three minutes in each condition and during the third minute, a 20-second collection was recorded. After the condition was complete, participants were notified that the treadmill was going to be lowered to level ground for one-minute of walking before adjusting the treadmill to the next condition. The treadmill inclination rate was set at 1m/s per manufacture setting. After all conditions were completed, the treadmill was set to level ground walking for a three-minute cool down. The entire walking protocol took approximately 24-minutes. During each walking condition, three-dimensional kinematic data was collected at 100Hz, surface EMG was collected at 2000Hz, and ground reaction forces (GRF) were collected at 2000Hz using Qualisys Track Manager V2.10 (QTM). All EMG and GRF data were analog to digital converted (16bit, ±5V) and synchronized with motion capture data through QTM.

Table 3-2: Four walking protocols (including each condition) determined using a balanced Latin square design.

	Protocol Order			
Protocol Number	Condition 1	Condition 2	Condition 3	Condition 4
One	-10º	+10º	-5º	+5º
Two	+10º	+5º	-10º	-5º
Three	-5º	-10º	+5º	+10º
Four	+5º	-5º	+10º	-10º

3.2.3 Maximum Voluntary Isometric Contraction (MVIC) Strength Testing

Retroreflective markers and marker clusters were removed after the walking protocols were completed, with EMG electrodes remaining untouched and secured for strength testing. Participants were asked to lay supine on a therapy bed and instructed to relax as much as possible while a one second resting muscle bias trial was collected. This resting muscle bias was used during processing, and all raw EMG recordings were corrected for subject bias.

To prevent the possibility of fatigue during challenged walking trials, maximal voluntary isometric strength was collected after the walking protocol bilaterally using a Humac Norm (Computer Sports Medicine, Inc., Stoughton, MA) isokinetic dynamometer for knee and hip flexion and extension, hip abduction and adduction. Trunk extension was collected manually with the participant laying prone on a therapy bed. Velcro straps were used to secure the participants shoulders and hips to the bed and the researcher manually stabilized the participants shanks to the bed. Knee flexion and extension MVICs were collected with the participant seated and their hips flexed to 90º, and the knee flexed to 45º

with the lever positioned on the anterior portion of the shank just proximal to the ankle joint[35]. Hip flexion and extension MVICs were collected with the participant supine and their hip flexed to 45° with the lever positioned on the thigh proximal to the knee joint[35]. Hip abduction and adduction MVICs were collected with the participant laying on their side and their hip abducted to 15° with the lever positioned on the lateral aspect of the thigh just proximal to the knee joint[35]. Velcro straps were used to stabilize the participant for each strength test. A gravity correction value was recorded by weighting the participants limb prior to each strength trial to adjust for the effect of gravity on limb mass. This correction was not required for trunk extension because this movement was only used to acquire an MVIC and not strength outcomes. The correction value was either added (extension/abduction) or subtracted (flexion/adduction) from the values for each trial. A warm-up, practice contraction was performed to familiarize the participant with the test protocol. Two, three-second MVICs with a 40-second rest period between each contraction were collected for each strength trial. The participant was given a ten-second break prior to testing a new muscle group. During each contraction, EMG was recorded at 2000Hz to acquire maximal muscle activation during the MVIC, which was used to normalize EMG collected during gait[130]. Verbal encouragement was provided to each participant to maximize effort and contraction consistency[131].

3.3 Data Processing

3.3.1 Kinematic Data

Three-dimensional motion data was filtered using a fourth order lowpass Butterworth 6Hz recursive filter custom written using MatLab R2018b (The Mathworks Inc., Massachusetts, USA). Local and technical anatomical bone embedded coordinate

systems for the pelvis, thigh, shank and foot were derived from virtual points, marker clusters and retroreflective skin markers. Joint angles were calculated using a six-degree of freedom model and a Cardan/Eular rotational sequence[132] including flexion/extension, adduction/abduction, internal rotation/external rotation. Flexion, adduction and internal rotation represent positive angles. All kinematic data was time normalized to 100% of the gait cycle (initial contact to ipsilateral initial contact) using a kinetic initial contact and pre swing detection method[133].

3.3.2 Electromyographic Data

All EMG signals were visually checked for any movement artifacts, dynamic range saturation or 60Hz noise and fast Fourier transformed (FFT) for each participant to represent the signal frequency spectrum and visually verify the power density spectra. Each raw EMG signal was bandpass filtered using a fourth order recursive Butterworth filter at cut-off frequencies of 10-500Hz, then corrected for subject bias and gain, and lastly converted to microvolts. The corrected signals were then full wave rectified and low pass filtered using a 6Hz fourth order recursive Butterworth filter.

All gait EMG waveforms were amplitude normalized to percent MVIC, using the maximum amplitude calculated from the corresponding MVIC trial and a 100ms moving average window[134]. Maximal strength was calculated using a 500ms moving average window to determine maximal torque generated during the three second MVIC trial[35]. The highest torque value calculated from either of the two MVIC trials was recorded as maximum and amplitude normalized to body mass (Nm/kg).

3.4 Reliability of outcomes

Gait analyses are considered the gold standard for assessing outcomes of lower extremity joint function. The reliability of gait analysis is integral for both researcher and clinician, as it gives the ability to interpret whether the difference between repeated assessments represents a true change or a change within boundaries of standard error[135]. These associated errors may prevent detection of significant between group or condition differences or lead to an underestimation of results. Studies have shown good to excellent day to day reliability in sagittal plane hip kinematics during level ground walking on a dual belt instrumented treadmill[113,135]. Authors have found excellent ICC values for peak sagittal plane hip flexion (ICC>0.80), peak sagittal plane hip extension (ICC>0.80), and total hip ROM (ICC>0.80) during the stance phase[113,135]. Similarly, overall frontal plane ROM during stance also had excellent reliability (ICC>0.80); however, frontal plane peak hip adduction during stance was high, but not excellent (ICC>0.60)[113,135]. In the transverse plane total ROM was found to have high test retest reliability (ICC>0.60)[135]. These results are further supported by a systematic review of 15 manuscripts and 8 abstracts stating that the majority of papers found good to excellent test retest reliability for sagittal and frontal plane hip ROM, with the majority of authors findings ICC values less than 0.7 for transverse plane ROM[136].

Electromyographic outcomes have also been shown to have good to high test-retest reliability during dual belt treadmill walking in asymptomatic individuals[113], with good to excellent test-retest reliability for both mean and peak MVIC normalized activation of the VM, VL, RF, MH, LH (ICC=0.74-0.95). However, Gmed showed fair test-retest reliability for both mean (ICC=0.42) and peak (ICC=0.53) MVIC normalized activation during

stance. To our knowledge, reliability data for Gmax activation during treadmill walking has not previously been reported.

3.5 Data Analysis

All kinematic and EMG data were time normalized to 101 data points representing a single gait cycle. Initial contact was used to detect the start and end of each cycle and identified using a 30N threshold for the vertical component of the GRF. Pre-swing was identified when the vertical component of the GRF fell below the 30N threshold. If a force plate crossover occurred during any walking condition (i.e., the left foot stepped on the right force plate or vice versa), initial contact instance was cross referenced with a kinematic technique that used the maximal positive sagittal displacement between the heel marker and the first pelvis marker as initial contact, and maximal negative sagittal displacement between the pelvis marker and second metatarsal marker as pre-swing[133].

For Objective 1, motion data was extracted from the symptomatic limb of individuals with FAI and a randomly selected limb from the asymptomatic individuals and was ensemble averaged across the 20-second trial for each of the five walking conditions. Kinematic data for the sagittal, frontal and transverse planes were collected; however, to address objective 1 only discrete sagittal plane biomechanical variables that are commonly reported in the FAI literature were further investigated. For sagittal plane hip kinematics, ROM (i.e., the difference between peak hip flexion and extension), peak hip extension and flexion were calculated (Table 3-3). Similarly, surface EMG data for the VM, VL, RF, MH and LH muscles were also collected; however, to address Objective 2, only mean and peak MVIC normalized Gmax and Gmed activation amplitudes during the stance phase were compared between individuals with FAI and asymptomatic individuals.

Table 3-3: Equations used for calculation of discrete hip kinematics

Discrete Metric	Equation
PkHF	Peak hip flexion during early stance
PkHE	Peak hip extension during mid stance
(S)ROM	Difference between peak hip flexion and extension

3.5 Statistical analysis

Participant demographics and clinical characteristics were reported as means with standard deviations for continuous variables and counts with percentages for categorical variables. For Objectives 1 and 2, group, condition or interactions for significant kinematic or EMG amplitude differences between the symptomatic limb for individuals with FAI and a randomly selected limb from the asymptomatic individuals were determined using a 2-way mixed measures ANOVA. Assumptions for the mixed measures ANOVA include: [1] normally distributed data within each group, and [2] homogeneity of variance, and sphericity. For each outcome measure, assumptions of equal variance and normality were tested using Levene's test and Kolmogorov-Smirnov[137], respectively. If assumption one was violated the data was transformed using a square root transformation or Johnson transformation, and if assumption two was violated the Brown-Forsythe corrected F value was used to examine significance[137]. Bonferroni corrected pair-wise comparisons were employed for significant main effects. An alpha (α) level of 0.05 was used to determine statistical significance. Effect sizes were calculated using the mean difference between groups for each walking condition divided by the pooled standard deviation and interpreted using previously determined cut points for Cohen's d including 0.2 (small), 0.5 (moderate),

and 0.8 (large)[138]. All statistical analyses were completed using SPSS (Version 25) and Minitab (Version 19.2).

Chapter 4 - A Comparison of Hip Joint Biomechanics and Neuromuscular Activations Between Individuals with Femoral Acetabular Impingement and Asymptomatic Individuals During Inclined and Declined Walking

4.1 Abstract

Femoroacetabular impingement (FAI) is a proliferative musculoskeletal diagnosis in young adults. Inconsistent findings during level ground walking between FAI and asymptomatic populations have led authors to speculate whether traditional level ground walking can elicit significant biomechanical alterations. The purpose of the current study was to investigate the effect of challenged walking on gait mechanics in individuals with FAI compared to asymptomatic, age matched individuals.

Seven patients with a clinical diagnosis of FAI and seven asymptomatic individuals were recruited to participate. All participants underwent gait analysis during level, inclined and declined walking. Surface electromyography was normalized to percent maximum voluntary isometric contraction. A 2-way mixed methods ANOVA assessed for within and between group differences for sagittal plane hip range of motion (SROM), peak hip extension and flexion angles, and peak and mean activation of the gluteus maximus (Gmax) and gluteus medius (Gmed). Effect sizes (ES) were calculated using the mean change between level ground and each incline/decline condition between groups.

No significant interactions or main effects for between group differences were observed. Significant main effects for condition were found for all biomechanical and neuromuscular variables ($p \leq 0.05$), with larger differences observed during 10° versus 5° incline or decline walking. Between group exploratory analyses revealed large ES for SROM and peak hip extension during all walking conditions ($d=0.81$-1.37). Varied ES

were observed for the remaining outcomes; however, for peak hip flexion, ES ranged from small to moderate (d=0.32-0.50). For peak Gmax, ES ranged from small to moderate (d=0.26-0.73). For mean Gmax, ES ranged from no difference to moderate (d=0.0-0.64). For peak Gmed, ES ranged from moderate to large (d=0.60-0.87) across all walking conditions. For mean Gmed, ES ranged from no difference to small (d=0.0-0.24).

Moderate to large ES suggest that challenged walking may elicit significant biomechanical and neuromuscular alterations between individuals with FAI and asymptomatic controls and support further investigation with larger samples and increased statistical power.

4.2 Introduction

Femoroacetabular impingement (FAI) is one of the most proliferative musculoskeletal diagnoses among young to middle aged adults, characterized by increased symptoms and premature contact between the femoral head and acetabular rim[1]. Aberrant hip joint structures result in multiple subclassifications of FAI including cam type (i.e. femoral head structural changes), pincer type (i.e. acetabular rim structural changes) and mixed type morphologies[6,10]. The contact between the femoral head and acetabulum is a proposed risk factor for abnormal joint mechanics thus accelerating cartilage degradation, labral damage and increased pain[4,5], and proposed to be a significant contributor to hip osteoarthritis (OA) development[15,18].

Previous studies have investigated the effects of FAI on biomechanical and neuromuscular outcomes to better understand the mechanical relationship to hip OA development; however, study findings are inconsistent or limited. Several studies have shown that individuals with FAI walk with reduced sagittal plane hip range of motion (ROM) compared to asymptomatic individuals[37,38]; however, there have been inconsistent results as to whether peak extension[38] or flexion[39] is the limiting factor. For example, peak flexion may be associated with a more impinged position of the joint, possibly resulting in premature contact between the femur and acetabulum[38]. Alternatively, peak hip extension may be associated with increased stretch of the anterior muscles and connective tissues, possibly resulting in anterior displacement of the femoral head and therefore increased pain[38]. Importantly, few studies have investigated neuromuscular activation to quantify the neuromuscular contributions to overall hip joint function in this population. To our knowledge, only two studies have previously investigated neuromuscular activation during

level ground walking in individuals with FAI[35,43]. Rutherford and colleagues (2018)[35] reported that individuals with FAI had increased overall gluteus maximus activation compared to asymptomatic individuals during the stance phase of gait. Conversely, Diamond and colleagues (2017)[43] examined muscle synergies of the deep hip external rotators. Obturator internus and quadratus femoris were more highly activated during the early swing phase of gait[43], potentially limiting internal rotation before the hip is moved into a flexed position. These limited findings, combined with their varied methodological approaches and structural outcomes of interest, highlight a need for further neuromuscular research to better understand its role in the disease process.

Although level ground walking is considered the gold standard for quantifying lower extremity joint mechanics, findings are inconsistent and limited in this population, thus highlighting whether more challenged walking tasks are necessary to understand hip joint mechanics in individuals with FAI[35,37]. Inclined and declined walking tasks are proposed to challenge the hip joint beyond level ground walking and may elicit larger differences between individuals with FAI and asymptomatic individuals. Although limited, previous research in asymptomatic individuals provides a framework for understanding how challenged walking tasks such as inclined and declined walking may alter hip joint movement throughout the gait cycle. Previous studies investigating challenged walking have shown that progressive gradient increases and decreases lead to corresponding increases and decreases in both sagittal plane hip ROM[44,46] and peak hip flexion[45], respectively. To our knowledge, there are no studies examining the effects of inclined and declined challenged walking on hip joint function in individuals with FAI; however, the increases in sagittal plane hip ROM and peak hip flexion observed during inclined and

declined challenged walking in asymptomatic individuals are consistent with limiting movement patterns previously reported during level ground walking in individuals with FAI[37,38]. Similarly, limited evidence exists quantifying neuromuscular activation patterns during challenged walking tasks in asymptomatic individuals. Previous findings suggest that increased gluteus maximus activation was observed during progressive gradient increases and may be associated with increased sagittal plane hip ROM and neuromuscular demands when ambulating on an incline[48,50]. Franz and Kram (2012)[48] reported increases up to 345% for mean gluteus maximus activation when walking at a 9^{o} incline compared to level ground walking. Research examining neuromuscular activation during declined walking has primarily focused on knee extensors with minimal insight into neuromuscular activity at the hip (e.g., rectus femoris)[50]. The observed differences in hip joint kinematics in asymptomatic individuals during declined walking suggest that potential neuromuscular differences may also be expected and elicit novel patterns un-identified during level walking in individuals with FAI.

We are unaware of previous research investigating the effects of challenged walking tasks on hip joint function in individuals with FAI. Inclined and declined walking may provide a more challenged walking environment to improve our understanding of biomechanical and neuromuscular effects on hip joint function and potential increased risk for hip OA development in this population. Therefore, the purpose of this study was to determine whether hip joint kinematics and neuromuscular activation patterns differ between individuals with FAI and asymptomatic individuals during level ground and inclined and declined walking tasks.

4.3 Methodology

4.3.1 Participant Recruitment

Fourteen individuals were recruited to participate including seven individuals with a clinical diagnosis of FAI and seven asymptomatic individuals. Individuals with FAI were recruited from an orthopaedic surgeon's clinic at the QEII Health Science Center. A clinical diagnosis for FAI was based on a triad of signs and symptoms outlined in the FAI Warwick Agreement[1]. Signs and symptoms included pain induced with hip flexion/abduction/external rotation, groin pain with hip flexion, or sharp pain with flexion and internal rotation. Radiographic evidence included an alpha angle >55^{o} for cam type, and acetabular retroversion for pincer type morphology. Patients were not eligible to participate if they were less than 18 years of age, had radiographically defined hip OA classified as a Kellgren and Lawrence grade of ≥ 2[118], inflammatory arthritis in either limb, prior surgery in either limb or bilateral FAI symptoms. Asymptomatic individuals were recruited through convenience sampling from the local Dalhousie and Halifax Regional Municipality communities and were given the same inclusion criteria excluding FAI requirements. All participants were required to complete a standardized telephone health screen to determine the presence of cardiovascular, neuromuscular, inflammatory or musculoskeletal conditions that would affect their gait, ability to climb stairs or complete the study. All participants provided written consent for the study procedures in accordance with the Nova Scotia Health Authority's Research Ethics Board (REB file number: 1024401).

4.3.2 Preparation

Participants completed the International Hip Outcome Tool (iHOTT33)[119], the Hip Osteoarthritis Outcome Survey (HOOS)[120], the Non-Arthritis Hip Score (NAHS)[123] and the Hip and Groin Outcome Score (HAGOS). All participants then changed into tight-fitting shorts and a T-shirt and removed their footwear. Anthropometric measures (i.e. height, weight, limb segment circumference) were obtained. All participants were then asked to walk barefoot along the GaitRITE instrumented walkway for a minimum of ten trials. Five trials were randomly selected to calculate their average walking speed[125,126].

Participants were prepared for surface electromyography (EMG) using a standardized protocol as described by the Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM) guidelines. All muscle sites were lightly shaved and cleaned with 70% alcohol wipes. Consistent with guidelines from the International Society of Electromyography and Kinesiology[124], Ag/AgCl surface electrodes (10mm diameter, 30mm interelectrode distance) (Red Dot; 3M Health Care) were placed bilaterally in a bipolar configuration over the gluteus maximus and medius. Surface EMG was recorded at 2000Hz using two AMT-8 (Bortec, Inc., Calgary, Alberta, Canada) EMG systems with gains of 100-5000x (Input impedance of ~10 GΩ, CMRR:115 dB at 60Hz, Band-pass 10-1000Hz) using Qualisys Track Manager (QTM). Quality of the gluteus maximus and medius EMG signals were visually checked using a standing maximal glute contraction and resisted hip abduction, respectively.

An eight-camera Qualysis Oqus 500 (Qualysis, Gothenburg, Sweden) motion analysis system was used to track three-dimensional kinematic data. Ridged plastic plates containing clusters of four retroreflective markers were placed over the trunk, pelvis, and

bilaterally over the lateral femur, lateral shank and feet, and secured using Velcro straps. Retroreflective markers were placed over the seventh cervical vertebrae and bilaterally over the shoulders, greater trochanters, medial and lateral tibial and femoral epicondyles, lateral and medial malleoli, first, second and fifth metatarsal heads and posterior heels. Both anterior superior iliac spines and sternal notch were determined using a pre-calibrated digitizer wand.

4.3.3 Warmup and Inclination Protocol

No participants used a harness during walking for this study. Participants acclimatized to walking barefoot on the dual-belt instrument treadmill (R-Mill; Motekforce Link) using a six-minute walking warmup protocol[127,128]. During the sixth minute of walking, a 20-second level ground baseline walking trial was collected prior to initiating the inclination protocol. The protocol consisted of four randomized inclined and declined walking conditions including: 5^{o} and 10^{o} inclines, and 5^{o} and 10^{o} declines. A balanced Latin square design was used to randomize the walking conditions[129]. A random number generator was used to assign participants to one of the four walking conditions and participants were blinded to the protocol order. Participants walked for a total of three minutes at each condition, during the third minute a 20-second collection was recorded. Between each challenged walking condition participants walked for one minute at level ground before transitioning to the next challenged walking condition. After all level ground and challenged walking conditions were completed, the treadmill was set to level ground where the participants walked for an additional three-minute cooldown. The entire walking protocol lasted approximately 24-minutes. During each walking condition, three-dimensional kinematic data was collected at 100Hz, surface EMG was collected at 2000Hz,

and ground reaction forces (GRF) were collected at 2000Hz using QTM V2.10. All EMG and GRF data were analog to digital converted (16bit, ±5V) and synchronized with motion capture data using QTM.

After participants completed the inclination protocol, all retroreflective markers were removed, and a resting muscle bias trial was recorded with participants supine on a therapy bed. Maximal voluntary isometric contractions (MVIC) were then collected via a Humac Norm (Computer Sports Medicine, Inc., Stoughton, MA) isokinetic dynamometer. The knee flexors and extensors were tested with the participant seated and knees in 45^{o} of flexion. Hip flexors and extensors were tested with the participant supine and hip in 45^{o} of flexion. Hip abduction and adduction were tested with the participant side-lying and the hip in 15^{o} of abduction. Trunk extension was collected manually with the participant laying prone on a therapy bed. At least one warm-up/practice contraction was used for familiarization. Following the warmup, two three-second maximum effort contractions were completed with each contraction separated by a 40-second rest period. A ten-second rest period was provided between muscle groups[130]. Standardized verbal encouragement was provided during each contraction to ensure consistent maximal contractions[131].

4.3.4 Data Processing

Custom written MatLab R2018b (The Mathworks Inc., Massachusetts, USA) programs were used for data processing. Local and technical anatomical bone embedded coordinate systems for the pelvis, thigh, shank and foot were derived from virtual points, marker clusters and retroreflective skin markers. A 6Hz, low-pass, fourth-order Butterworth recursive filter was used to smooth all kinematic data. Joint angles were calculated using a six-degree of freedom model and a Cardan/Eular rotational sequence[132].

Flexion represented positive angles, while extension represented negative angles. All kinematic data were time normalized to 100% of the gait cycle (initial contact to ipsilateral initial contact) using a kinetic initial contact and pre swing detection method[133].

Raw EMG data were band-pass filtered using a 10-500Hz, fourth-order, recursive Butterworth filter, corrected for resting subject bias, gain adjusted, converted to microvolts, full-wave rectified, and low-pass filtered using a 6Hz, fourth-order Butterworth recursive filter. Surface EMG profiles were amplitude normalized using the maximum amplitude calculated from a 100ms moving average window for all corresponding MVIC trials[134]. Maximal strength was calculated using a 500ms moving average window to determine maximal torque generated during the three second MVIC trial[35]. The highest torque value calculated from either of the two MVIC trials was recorded as maximum and amplitude normalized to body mass (Nm/kg).

4.3.5 Data Analysis

For asymptomatic participants, the study limb was randomly selected for further analyses. Gait cycle kinematic data was averaged across all cycles within the 20-second data collection window for each of the five walking conditions. Sagittal plane hip kinematics were calculated for ROM (i.e., the difference between peak hip flexion and extension) during stance as well as peak hip extension and flexion angles. The peak and mean activation for MVIC normalized gluteus maximus and medius activation amplitudes during stance were also calculated.

4.3.6 Statistical Analyses

Means with standard deviations and 95% confidence intervals (95%CIs) were calculated for all sagittal plane hip kinematic and neuromuscular outcomes in each group

for each walking condition. Paired samples t-tests were used to detect differences in level ground walking before and after the inclination protocol in each group. A 2-way mixed methods analysis of variance (ANOVA) was used to test for group by condition interactions and main effects for all biomechanical variables and EMG amplitudes. Normality and equal variance were assessed using the Kolmogorov-Smirnov and Levene's tests, and sphericity was assessed using Mauchly's test of sphericity. If normality was violated, the data was transformed using a square root or Johnson transformation. If sphericity was violated, the Greenhouse-Geisser estimate was used. Due to the pilot nature of the data, planned contrasts were used to assess the effect of each challenged walking condition on the identified dependent variables of interest. Effect sizes were calculated using the mean difference between groups for each walking condition divided by the pooled standard deviation and interpreted using previously determined cut points for Cohen's d including 0.2 (small), 0.5 (moderate), and 0.8 (large)[138]. All statistical analyses were completed using SPSS (Version 25), and Minitab (Version 19.2).

4.4 Results

Due to COVID-19 research restrictions, anticipated recruitment of ten participants in each group was not achieved; therefore, the presented data includes a total of fourteen individuals (7 per group) who consented to participate in this study and were able to complete the study protocol. Participant demographics and clinical characteristics are presented in Table 4-1. No significant between group differences were observed for age and BMI, walking velocity, or muscle strength. Patient-reported outcomes for symptoms, function, quality of life and overall health were significantly lower (worse) in participants diagnosed with FAI. No differences were observed for biomechanical or neuromuscular

outcome measures during level ground walking before and after the inclination protocol for either group.

Table 4-1: Participant demographics and clinical characteristics (n=14)

	FAI (n=7)	Control (n=7)	p
Sex, no. of females/males	4 / 3	5 / 2	
Age, years	35 (11)	32 (8)	0.755
BMI, kg/m^2	28.4 (3.6)	25.8 (4.1)	0.363
Walking speed, m/s	1.11 (0.11)	1.17 (0.10)	0.350
Cam type FAI (no.)	0	-	
Pincer type FAI (no.)	0	-	
Mixed type FAI (no.)	7	-	
Alpha Angle, deg	68 (10)	-	
Crossover Sign #Y / #N	7 / 0	-	
iHOT-33 Total (0-100)	38 (20)	97 (4)	**0.001**
HOOS Total (0-100)	54 (20)	98 (4)	**0.018**
HAGOS Total (0-100)	42 (19)	98 (6)	**0.006**
NAHS Total (0-100)	55 (23)	99 (1)	**0.024**
Hip Extension Strength, Nm/kg	1.5 (0.6)	1.5 (0.7)	0.857
Hip Flexion Strength, Nm/kg	1.2 (0.4)	1.3 (0.4)	0.957
Hip Abduction Strength, Nm/kg	1.4 (0.3)	1.3 (0.3)	0.459
Hip Adduction Strength, Nm/kg	1.1 (0.3)	0.9 (0.3)	0.246

Values represent means and standard deviations; Bold p-values represent significant between-group differences; iHOT= International Hip Outcome Tool; HOOS= Hip Osteoarthritis Outcome Score; HAGOS= The Coppenhagen Hip and Groin Outcome Score; NAHS= Non-Arthritic Hip Score

Sagittal Plane Hip Range of Motion. Ensemble average waveforms for hip ROM in the sagittal plane during each walking condition are illustrated in Figure 4-1. Means and standard deviations for sagittal plane hip ROM in each group and walking condition, mean differences between groups with bootstrapped 95%CIs, and effect sizes are presented in Table 4-2. No significant group by condition interaction was observed for sagittal plane

hip ROM (F(4,48)=0.98; p=0.426). A significant main effect was observed for walking condition (F(4,48)=439.7; p≤0.001) but not group (F(1,12)=4.6, p=0.050). Planned contrasts revealed that compared to level ground walking both 5° (F(1,12)=153.7; p≤0.001) and 10° (F(1,12)=111.0; p≤0.001) declined walking conditions resulted in significantly smaller sagittal plane hip ROM, with largest reductions observed when walking at a 10° decline. The 5° (F(1,12)=163.3; p≤0.001) and 10° (F(1,12)=603.4; p≤0.001) inclined walking conditions resulted in significantly larger sagittal plane hip ROM, with largest increases observed when walking at a 10° incline. Between groups, large effect sizes were found for all walking conditions (d=0.91-1.37).

Table 4-2: Means (SD), mean differences (95%CIs) and effect sizes for sagittal plane hip range of motion (°) during level ground, inclined and declined walking

Walking Condition	FAI	ASYMP	Mean Diff (95%CI)	Effect Size [a]
Level Ground	32 (3)	36 (4)	-4 (-6.1, -0.13)	1.13
Five Degree Decline	25 (4)	30 (5)	-5 (-9.2, -0.66)	1.10
Ten Degree Decline	20 (5)	25 (6)	-5 (-9.7, 1.4)	0.91
Five Degree Incline	41 (4)	46 (6)	-5 (-10.5, -0.50)	0.98
Ten Degree Incline	53 (4)	60 (6)	-7 (-12.1, -1.0)	1.37

[a] Effect sizes were calculated as the mean difference between groups for each walking condition, divided by the pooled standard deviation.
Values for range of motion are presented in degrees (°).
Negative mean difference values indicate values are lower in individuals with FAI.

Peak Hip Extension. Means and standard deviations for peak hip extension in each group and walking condition, mean differences between groups with bootstrapped 95%CIs, and effect sizes are presented in Table 4-3. Peak hip extension violated the assumption of

sphericity; therefore, subsequent analyses utilized Greenhouse-Geisser (estimate of departure from sphericity was 0.66). No significant group by condition interaction was observed for peak hip extension ($F(2.4,29.3)=1.7$; $p=0.190$). A significant main effect was observed for walking condition ($F(2.4,29.3)=28.6$; $p\leq0.001$) but not group ($F(1,12)=3.2$; $p=0.099$). Planned contrasts revealed that compared to level ground walking both 5^o ($F(1,12)=21.3$; $p=0.001$) and 10^o ($F(1,12)=83.5$; $p\leq0.001$) declined walking conditions resulted in significantly lower peak hip extension. Although peak hip extension during the 5^o inclined walking condition ($F(1,12)=6.4$; $p=0.026$) was not significantly different from level ground walking, peak hip extension was significantly lower during the 10^o inclined walking condition ($F(1,12)=10.7$; $p=0.007$). Between groups, large effect sizes were found for all walking conditions ($d=0.81$-1.13).

Table 4-3: Means (SD), mean differences (95%CIs) and effect sizes for peak hip extension (°) during level ground, inclined and declined walking

Walking Condition	FAI	ASYMP	Mean Diff (95%CI)	Effect Size [a]
Level Ground	0 (7)	7 (9)	-7 (-15.5,1.4)	0.87
Five Degree Decline	-3 (8)	5 (9)	-8 (-17.7,0.10)	0.94
Ten Degree Decline	-7 (7)	0 (10)	-7 (-16.6,2.7)	0.81
Five Degree Incline	-2 (8)	7 (8)	-9 (-18.9,0.47)	1.13
Ten Degree Incline	-4 (8)	6 (12)	-10 (-21.3,0.24)	0.98

[a] Effect sizes were calculated as the mean difference between groups for each walking condition, divided by the pooled standard deviation.
Values for peak hip extension are presented in degrees (°).
Negative mean difference values indicate values are less in individuals with FAI.
Positive values indicate extension, negative values indicate flexion.

Peak Hip Flexion. Means and standard deviations for peak hip flexion in each group and walking condition, mean differences between groups with bootstrapped 95%CIs, and effect sizes are presented in Table 4-4. Peak hip flexion violated the assumption of sphericity; therefore, subsequent analyses utilized Greenhouse-Geisser (estimate of the departure from sphericity was 0.58). No significant group by condition interaction was observed for peak hip flexion ($F(2.3,27.7)=0.23$; $p=0.828$). A significant main effect was observed for walking condition ($F(2.3, 27.7)=593.0$; $p=<0.001$) but not group ($F(1,12)=0.66$; $p=0.434$). Planned contrasts revealed that compared to level ground walking both 5^o ($F(1,12)=43.7$; $p\leq0.001$) and 10^o ($F(1,12)=33.5$; $p\leq0.001$) declined walking conditions resulted in significantly decreased peak hip flexion, with the largest reductions observed when walking at a 10^o decline. The 5^o ($F(1,12)=242.5$; $p\leq0.001$) and 10^o ($F(1,12)=1936.2$; $p\leq0.001$) inclined walking conditions resulted in significantly increased peak hip flexion, with largest increases observed when walking at a 10^o incline. Effect sizes ranged from small ($d=0.32$) to moderate ($d=0.50$) between groups, with largest effect sizes observed during inclined versus declined walking.

Table 4-4: Means (SD), mean differences (95%CIs) and effect sizes for peak hip flexion (°) during level ground, inclined and declined walking.

Walking Condition	FAI	ASYMP	Mean Diff (95%CI)	Effect Size [a]
Level Ground	32 (6)	28 (10)	4 (-4.2, 12.7)	0.48
Five Degree Decline	28 (6)	25 (9)	3 (-4.2, 12.1)	0.39
Ten Degree Decline	28 (7)	25 (11)	3 (-6.8, 12.4)	0.32
Five Degree Incline	43 (7)	39 (9)	4 (-4.7, 12.1)	0.50
Ten Degree Incline	57 (6)	53 (10)	4 (-4.6, 12.4)	0.49

[a] Effect sizes were calculated as the mean difference between groups for each walking condition, divided by the pooled standard deviation.
Values for peak hip flexion are presented in degrees (°).
Negative mean difference values indicate values are lower in individuals with FAI.

Peak Gluteus Maximus. Ensemble average waveforms for gluteus maximus activation during each walking condition are illustrated in Figure 4-2. Means and standard deviations for peak gluteus maximus activation in each group and walking condition, mean differences between groups with bootstrapped 95%CIs, and effect sizes are presented in Table 4-5. Peak gluteus maximus activation violated the assumption of sphericity; therefore, subsequent analyses utilized Greenhouse-Geisser (estimate of the departure from sphericity was 0.40). No significant group by condition interaction was observed for peak gluteus maximus activation ($F(1.6,19.3)=1.08$; $p=0.347$). A significant main effect was observed for walking condition ($F(1.6,19.3)=8.5$; $p=0.004$) but not group ($F(1,12)=0.79$; $p=0.393$). Planned contrasts revealed that compared to level ground walking, 5° declined walking resulted in a significant decrease in peak gluteus maximus activation ($F(1,12)=15.7$; $p=0.002$), while walking at a 5° incline resulted in significantly increased peak gluteus maximus activation ($F(1,12)=10.8$; $p=0.007$). Neither 10° declined

(F(1,12)=3.4; p=0.090) nor 10° inclined (F(1,12)=3.9; p=0.071) walking showed a significant effect on peak gluteus maximus activation. Effect sizes between groups ranged from small (d=0.26) to moderate (d=0.73), with the largest effect sizes observed during both 10° challenged walking conditions.

Table 4-5: Means (SD), mean differences (95%CIs) and effect sizes for peak gluteus maximus (Gmax) activation (%MVIC) during level ground, inclined and declined walking.

Walking Condition	FAI	ASYMP	Mean Diff (95%CI)	Effect Size [a]
Level Ground	24 (24)	32 (36)	-8 (-41.7, 23.5)	0.26
Five Degree Decline	20 (19)	26 (26)	-6 (-30.2, 18.6)	0.26
Ten Degree Decline	14 (10)	26 (22)	-12 (-30.6, 4.64)	0.70
Five Degree Incline	28 (24)	39 (37)	-11 (-42.4, 20.0)	0.35
Ten Degree Incline	25 (15)	50 (46)	-25 (-60.8, 6.2)	0.73

[a] Effect sizes were calculated as the mean difference between groups for each walking condition, divided by the pooled standard deviation.
Values for peak Gmax activation are presented as %MVIC.
Negative mean difference values indicate values are lower in individuals with FAI.

Mean Gluteus Maximus. Means and standard deviations for mean gluteus maximus activation in each group and walking condition, mean differences between groups with bootstrapped 95%CIs, and effect sizes are presented in Table 4-6. Mean gluteus maximus activation violated the assumption of sphericity; therefore, subsequent analyses utilized Greenhouse-Geisser (estimate of the departure from sphericity was 0.41). No significant group by condition interaction was observed for peak gluteus maximus activation (F(1.7,20.0)=1.09; p=0.344). A significant main effect was observed for walking condition

(F(1.7,20.0)=18.3; p≤0.001) but not group (F(1,12)=0.47; p=0.505). Planned contrasts revealed that compared to level ground walking both 5° (F(1,12)=14.6; p=0.002) and 10° (F(1,12)=5.3; p=0.039) declined walking conditions significantly decreased mean gluteus maximus activation, with largest reductions observed when walking at 10° decline. The 5° (F(1,12)=55.2; p≤0.001) and 10° (F(1,12)=13.2; p=0.003) inclined walking conditions resulted in a significant increase in mean gluteus maximus activation, with largest increases observed when walking at a 10° incline. Effect sizes between groups ranged from no difference to moderate (d=0.64), with the largest effect sizes observed during both 10° challenged walking conditions.

Table 4-6: Means (SD), mean differences (95%CIs) and effect sizes for mean gluteus maximus (Gmax) activation (%MVIC) during level ground, inclined and declined walking.

Walking Condition	FAI	ASYMP	Mean Diff (95%CI)	Effect Size [a]
Level Ground	11 (10)	13 (11)	-2 (-12.8, 9.7)	0.19
Five Degree Decline	9 (9)	9 (7)	0 (-8.1, 8.5)	0.0
Ten Degree Decline	7 (6)	10 (7)	-3 (-8.8, 4.1)	0.46
Five Degree Incline	14 (12)	18 (15)	-4 (-16.7, 11.2)	0.29
Ten Degree Incline	14 (11)	24 (19)	-10 (-26.1, 5.6)	0.64

[a] Effect sizes were calculated as the mean difference between groups for each walking condition, divided by the pooled standard deviation.
Values for mean Gmax activation are presented as %MVIC.
Negative mean difference values indicate values are lower in individuals with FAI.

Peak Gluteus Medius. Ensemble average waveforms for gluteus medius activation during each walking condition are illustrated in Figure 4-3. Means and standard deviations for

peak gluteus medius activation in each group and walking condition, mean differences between groups with bootstrapped 95%CIs, and effect sizes are presented in Table 4-7. Peak gluteus medius activation violated the assumption of sphericity; therefore, subsequent analyses utilized Greenhouse-Geisser (estimate of the departure from sphericity was 0.37). No significant group by condition interaction was observed for peak gluteus medius activation ($F(1.7,20.8)=0.39$; $p=0.653$). A significant main effect was observed for walking condition ($F(1.73,20.8)=16.5$; $p\leq0.001$) but not group ($F(1,12)=2.1$; $p=0.171$). Planned contrasts revealed that compared to level ground walking, 5^{o} declined walking resulted in significantly decreased peak gluteus medius activation ($F(1,12)=7.7$; $p=0.017$) but not at 10^{o} declined ($F(1,12)=2.1$; $p=0.178$). Alternatively, 5^{o} inclined ($F(1,12)=3.3$; $p=0.093$) walking did not result in a significant change in peak gluteus medius activation but was significantly increased when walking at a 10^{o} incline ($F(1,12)=22.8$; $p\leq0.001$). Moderate ($d=0.60$) to large ($d=0.87$) effect sizes were observed between groups across all walking conditions.

Table 4-7: Means (SD), mean differences (95%CIs) and effect sizes for peak gluteus medius (Gmed) activation (%MVIC) during level ground, inclined and declined walking.

Walking Condition	FAI	ASYMP	Mean Diff (95%CI)	Effect Size [a]
Level Ground	34 (19)	47 (19)	-13 (-30.9, 8.5)	0.68
Five Degree Decline	29 (15)	43 (17)	-14 (-29.1, 4.3)	0.87
Ten Degree Decline	31 (19)	45 (27)	-14 (-30.3, 7.1)	0.60
Five Degree Incline	37 (22)	55 (26)	-18 (-43.4, 6.7)	0.74
Ten Degree Incline	45 (26)	65 (30)	-20 (-49.6, 9.2)	0.71

[a] Effect sizes were calculated as the mean difference between groups for each walking condition, divided by the pooled standard deviation.
Values for peak Gmed activation are presented as %MVIC.
Negative mean difference values indicate values are lower in individuals with FAI.

Mean Gluteus Medius. Means and standard deviations for mean gluteus medius activation in each group and walking condition, mean differences between groups with bootstrapped 95%CIs, and effect sizes are presented in Table 4-8. Mean gluteus medius activation violated the assumption of sphericity; therefore, subsequent analyses utilized Greenhouse-Geisser (estimate of the departure from sphericity was 0.37). No significant group by condition interaction was observed for peak gluteus medius activation ($F(4,48)=0.18$; $p=0.948$). A significant main effect was observed for walking condition ($F(1.73,20.8)=16.5$; $p\leq0.001$) but not group ($F(1,12)=2.1$; $p=0.171$). Planned contrasts revealed that compared to level ground walking both 5^o ($F(1,12)=19.7$; $p=0.001$) and 10^o ($F(1,12)=7.7$; $p=0.017$) declined walking conditions resulted in significantly less mean gluteus medius activation, with largest reductions observed when walking at a 10^o decline. The 5^o ($F(1,12)=25.9$; $p\leq0.001$) and 10^o ($F(1,12)=67.7$; $p\leq0.001$) inclined walking

conditions resulted in significantly larger mean gluteus medius activations, with largest increases observed when walking at a 10^{o} incline. Effect sizes between groups ranged from no difference to small (d=0.24), with largest effect sizes observed during inclined versus declined walking.

Table 4-8: Means (SD), mean differences (95%CIs) and effect sizes for mean gluteus medius (Gmed) activation (%MVIC) during level ground, inclined and declined walking.

Walking Condition	FAI	ASYMP	Mean Diff (95%CI)	Effect Size [a]
Level Ground	17 (13)	17 (7)	0 (-9.9, 11.6)	0
Five Degree Decline	13 (12)	14 (6)	-1 (-8.7, 9.9)	0.10
Ten Degree Decline	15 (12)	15 (5)	0 (-8.8, 10.9)	0
Five Degree Incline	21 (14)	24 (10)	-3 (-15.5, 9.9)	0.24
Ten Degree Incline	27 (18)	30 (13)	-3 (-19.4, 14.8)	0.19

[a] Effect sizes were calculated as the mean difference between groups for each walking condition, divided by the pooled standard deviation.
Values for mean Gmed activation are presented as %MVIC.
Negative mean difference values indicate values are lower in individuals with FAI.

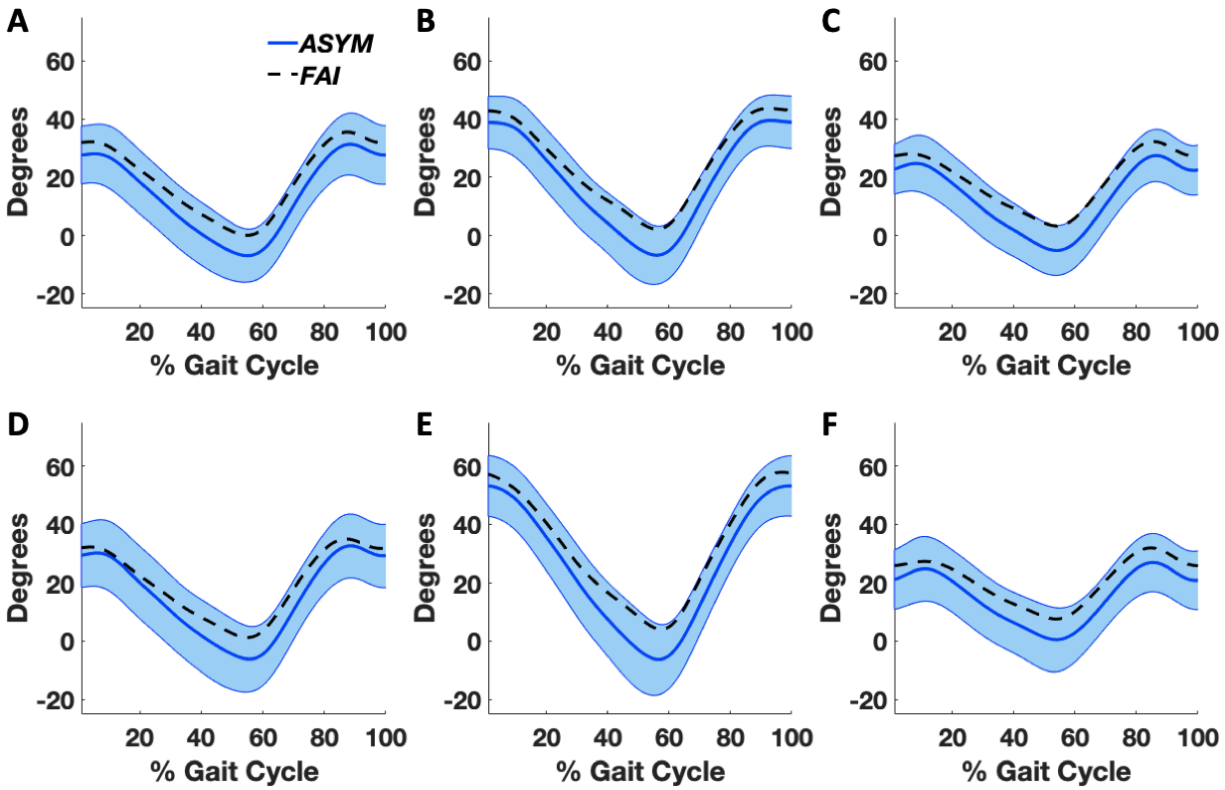

Figure 4-1: Ensemble average waveforms for sagittal plane hip range of motion during (A) level ground pre challenged walking, (B) 5° inclined walking, (C) 5° declined walking, (D) level ground post challenged walking, (E) 10° inclined walking and (F) 10° declined walking. Each waveform is time normalized to percentage of the gait cycle. Data are presented for asymptomatic participants (ASYM, solid line) and participants diagnosed with femoroacetabular impingement (FAI, dashed line). The shaded grey area represents 1SD above and below the mean for ASYM participants.

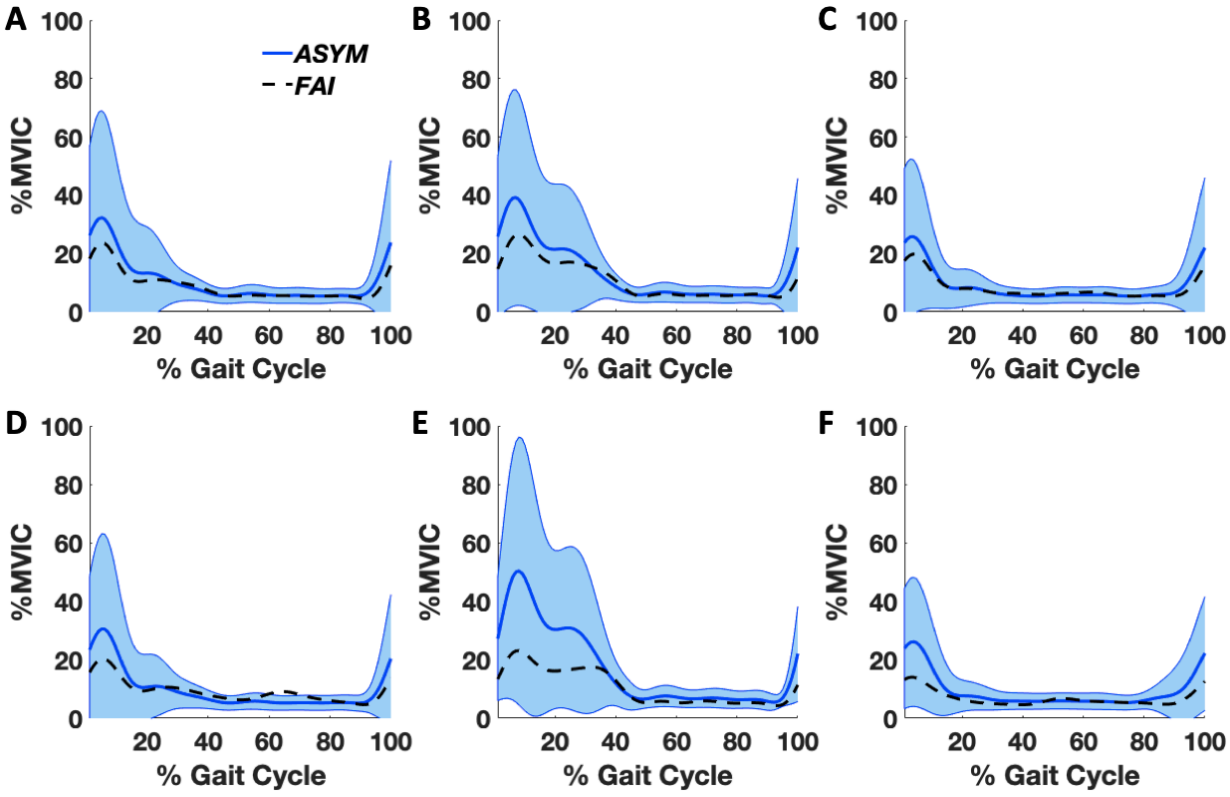

Figure 4-2: Ensemble average waveforms for gluteus maximus activation expressed as a percentage of maximum voluntary isometric contraction (MVIC) during (A) level ground pre challenged walking, (B) 5° inclined walking, (C) 5° declined walking, (D) level ground post challenged walking, (E) 10° inclined walking and (F) 10° declined walking. Each waveform is time normalized to percentage of the gait cycle. Data are presented for asymptomatic participants (ASYM, solid line) and participants diagnosed with femoroacetabular impingement (FAI, dashed line). The shaded grey area represents 1SD above and below the mean for ASYM participants.

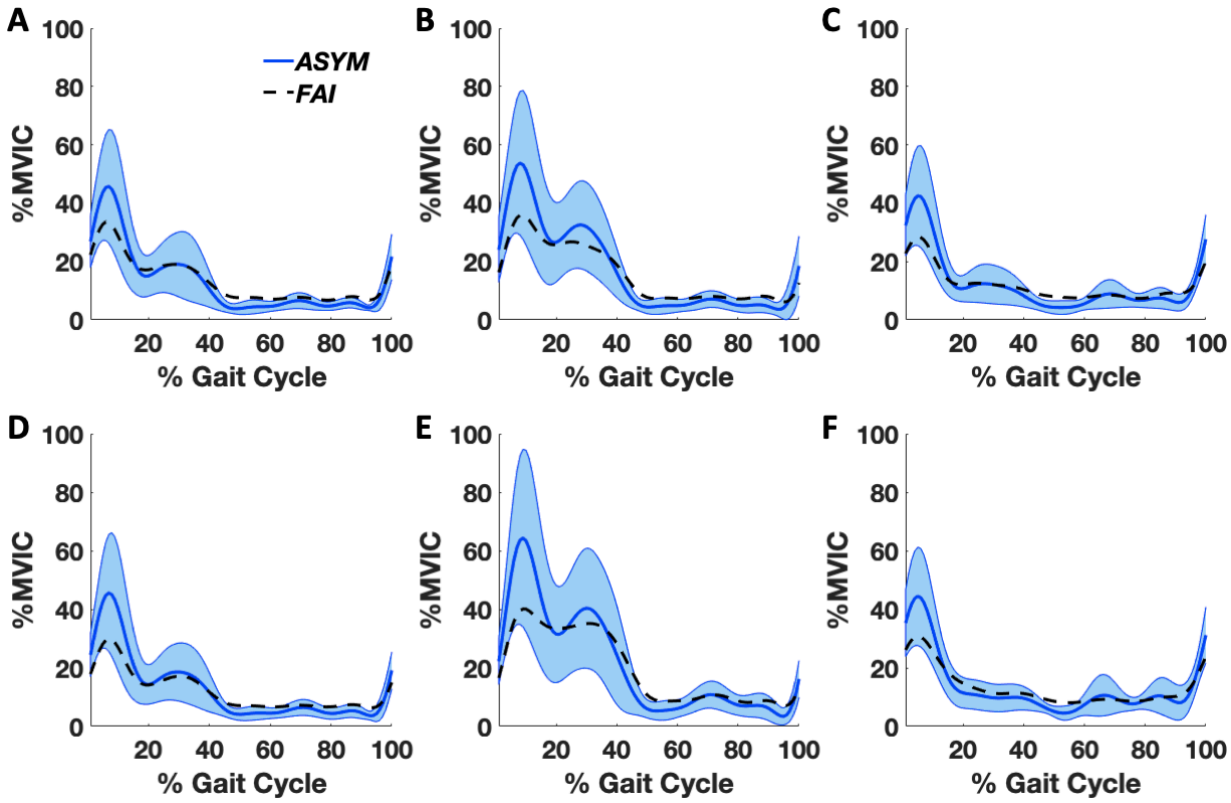

Figure 4-3: Ensemble average waveforms for gluteus medius activation expressed as a percentage of maximum voluntary isometric contraction (MVIC) during (A) level ground pre challenged walking, (B) 5° inclined walking, (C) 5° declined walking, (D) level ground post challenged walking, (E) 10° inclined walking and (F) 10° declined walking. Each waveform is time normalized to percentage of the gait cycle. Data are presented for asymptomatic participants (ASYM, solid line) and participants diagnosed with femoroacetabular impingement (FAI, dashed line). The shaded grey area represents 1SD above and below the mean for ASYM participants.

4.5 Discussion

Previous biomechanical investigations comparing individuals with FAI and asymptomatic individuals during level ground walking have been inconsistent, and question whether traditional level ground walking is sensitive enough to detect meaningful biomechanical differences between populations. Challenged walking tasks are a proposed method to increase hip ROM and stress the joint more than traditional level ground gait analysis. The current study compared the effects of challenged walking between individuals with FAI and asymptomatic individuals. Although the findings support that walking mechanics and neuromuscular activations are altered during challenged inclined and declined walking, the adapted movement patters were similar between individuals with FAI and asymptomatic individuals. Individuals with FAI self-reported worse symptoms, function and quality of life compared to asymptomatic individuals, yet their fundamental movement mechanics were consistent ($p=<0.05$).

Sagittal plane hip mechanics were not significantly different between groups for level ground or challenged inclined and declined walking conditions. The mean differences and calculated effect sizes suggest that individuals with FAI may walk with less hip extension through pre swing, which contributes to overall reductions in sagittal plane hip ROM. Previous studies investigating level ground gait biomechanics have demonstrated similar sagittal plane hip kinematic differences between individuals with FAI and asymptomatic individuals that were observed in the current study during challenged inclined and declined walking[37,38]. These findings are also consistent with reduced sagittal plane hip ROM and hip extension observed in individuals with hip OA compared to asymptomatic individuals[103,101] and may contribute to a biomechanical mechanism

between FAI and hip OA development[15,20]. Reduced peak hip extension, and therefore reduced sagittal plane hip ROM, have previously been observed[37,38], which are consistent with modelling studies that suggest increased hip extension to be associated with higher compressive forces in the anterior hip[98]. Although individuals with FAI appeared to avoid end-range hip extension to minimize aberrant loads on anterior hip structures, anticipated reductions in peak hip flexion during inclined walking were not observed. Across all conditions individuals with FAI walked with slightly higher peak hip flexion angles, which might suggest attempted offloading of the anterior hip structures in accordance with previous modelling studies. Alternatively, the magnitude of flexion needed to ambulate on a 10^{o} incline may not surpass positions of impingement (i.e., excessive flexion) that are more likely observed during higher impact activities and thus not observed in the current study[21].

Neuromuscular contributions to the mechanics of FAI have historically received very little attention. Although limited research challenges the interpretation of the current findings, the observed neuromuscular differences between individuals with FAI and asymptomatic individuals suggests a potential role in abnormal hip joint function. To our knowledge, only two studies have examined EMG during gait in individuals with FAI[35,43], and only one of these studies examined gluteus maximus and gluteus medius[35]. Although the current findings do vary from previous investigations, this study was the first to investigate challenged walking and its effects on neuromuscular activity. Previous reports suggest that increasing hip extension leads to higher anterior hip contact forces[98]. Individuals with versus without FAI in the current study walked up an incline with MVIC normalized peak gluteus maximus activation reduced up to 25% compared to

asymptomatic individuals, which is consistent with reductions in peak hip extension and possibly an adapted mechanism to lower anterior hip contact forces and protect hip joint structures. These observations also vary from increased midstance gluteus maximus activation in individuals with severe hip OA[103], which was not observed in individuals with mild hip OA or asymptomatic individuals[103]. Interestingly, the mild hip OA group had no difference in gluteus maximus activation compared to asymptomatic controls[103]. These opposing findings for gluteus maximus activation between pathologies highlight that different structural impairments associated with FAI versus hip OA may have a potential role and might explain the inconsistent neuromuscular findings across FAI types as well. Further research examining gluteus muscle activation across hip pathologies including FAI type and hip OA severity is needed.

Alternatively, pelvic kinematics may influence hip extensor activation and previous work suggests that when prone, anterior rotation of the pelvis is associated with reductions in hip extensor activity, although the mechanisms to clarify this relationship were unclear[139]. Individuals with versus without FAI have previously demonstrated increased anterior pelvic tilt during level ground walking[106]. Although pelvic markers were used to quantify hip joint angles in the current study, pelvic kinematics were not directly quantified but could help explain the observed decreased gluteus maximus activation and shed light on the interplay between pelvic motions and neuromuscular activity.

One previous study has examined gluteus medius during level ground walking in individuals with FAI; however, no significant differences between individuals with FAI and asymptomatic individuals were observed using principle component analysis[35]. Similarly, there were no significant differences between groups in the current study;

however, individuals with FAI walked with up to a 20% reduction in MVIC normalized peak gluteus medius activation and only a 3% reduction in mean gluteus medius activation compared to asymptomatic individuals during 10^{o} inclined walking. Frontal plane hip kinematics were not investigated; therefore, it remains unclear whether these small reductions in activation are associated with increased hip adduction or contralateral pelvic drop walking patterns. Previous research has shown that individuals with mild to moderate hip OA walk with increased frontal plane muscle co-contraction of the adductors (adductor brevis, longus, magnus, biceps femoris long head, gracilis, semimembranosus, semitendinosus) and abductors (gluteus medius, gluteus minimus, sartorius and tensor fascia latae) compared to asymptomatic individuals[140], yet despite increased co-activation, individuals with mild to moderate hip OA had less hip joint contact forces. Whether a similar relationship exists in individuals with FAI remains unclear.

Limitations. The small sample size in each group underpowered the a priori statistical analyses of the current study and prevented more conclusive interpretation of the results. Regardless of size, the groups were well matched with no significant differences between group demographics. Statistical analyses were still performed; however, descriptive between group differences with bootstrapped 95% confidence intervals and effect sizes interpreted using Cohen's d were used to help interpret the available findings despite the small sample size. Second, bilateral radiographic evidence for mixed-type FAI was present in both limbs of individuals with FAI; however, symptoms were only present in one limb. Radiographs were not available for the asymptomatic group, therefore radiographic evidence of FAI in this group is uncertain. Previously, a systematic review including 2114 asymptomatic hips found the prevalence of asymptomatic cam and pincer

type morphology was approximately 37% and 67%, respectively[141]; therefore, the asymptomatic participants in the current study could have unknowingly had structural hip abnormalities. Third, the limited number of males in the present study prevented further assessment of sex-related differences. Although sex-related investigations were outside the scope of this study, previous research has shown the potential for kinematic differences between males and females that could have an impact on the current findings[106].

Conclusion. Although statistical differences between groups were not observed, moderate to large effect sizes suggest that the effect of challenged walking on biomechanical and neuromuscular outcomes may differ between individuals with FAI and asymptomatic individuals. Individuals with FAI walked with less sagittal plane hip ROM and peak hip extension compared to asymptomatic individuals during level ground and all challenged walking conditions. The largest kinematic differences between groups were observed during 10^{o} inclined walking. From a neuromuscular perspective, moderate effect sizes were found for peak gluteus maximus activation during both 10^{o} inclined and declined walking, peak gluteus maximus activations during 10^{o} inclined walking and peak gluteus medius activations during level ground and all challenged walking conditions, suggesting lower overall glute activation in individuals with FAI. Lower gluteus maximus activation was consistent with lower peak hip extension in individuals with FAI, and suggest a possible neuromuscular mechanism for hip joint function. Similarities in hip joint kinematics and gluteus medius patterns observed between individuals with FAI and hip OA also suggest a potential biomechanical and neuromuscular mechanism in hip pathology progression and new directives for understanding hip mechanics and joint function associated with FAI. Results may inform future investigations examining challenged

walking in individuals with FAI, as well as provide insight into abnormal hip joint function that may facilitate tailoring rehabilitative strategies to mitigate the effects of hip pathology. Further research is needed to help understand hip joint mechanics and begin to examine the disease-related effects on biomechanical and neuromuscular outcomes and abnormal loading relevant to progression.

Chapter 5 – Discussion

Biomechanical and neuromuscular investigations during level ground walking have previously been investigated in individuals with FAI, with and without comparison to asymptomatic individuals. In contrast, unlevel or more challenged walking environments that are also commonly encountered across activities of daily living have not been well-investigated and their effects on joint function are poorly understood. The purpose of the current study was to determine whether hip joint kinematics and neuromuscular activation patterns differ between level ground walking and more challenged walking activities, including inclined and declined walking, between individuals with FAI and asymptomatic individuals. Individual waveforms for all kinematic and EMG variables for both the asymptomatic and FAI groups can be found in Appendices A and B.

5.1 Discussion

Although no statistically significant findings were observed in the current study, the reported mean differences and effect sizes between groups suggest possible biomechanical and neuromuscular activation differences exist between individuals with FAI and asymptomatic individuals. Morphology specifics are a frequent and sometimes poorly reported methodological difference between studies investigating FAI mechanics and has resulted in a limited understanding of potential mechanical differences between cam, pincer and mixed type disease. To date, a large number of studies investigating FAI mechanics have included participants with primarily cam type morphology[38,39,35]. Therefore, findings of the current study should be interpreted with caution due to a weaker understanding of the impact of pincer structural pathology on biomechanical and neuromuscular outcomes. Investigating individuals with mixed type morphology may

provide new insights into FAI mechanics and hip joint pathology. Future studies should consider specific morphological comparisons across FAI sub-types.

Despite previous evidence primarily focusing on cam type morphology, inconsistent findings still exist when describing sagittal plane hip kinematic differences associated with FAI, and there is little consensus whether decreased hip flexion or extension is predominantly responsible for this relationship. Femoral acetabular impingement is a proposed risk factor for hip OA development and previous gait studies have shown that individuals with hip OA walk with reduced sagittal plane hip ROM[103,101] compared to asymptomatic individuals, and that reduced sagittal plane hip kinematics are associated with reduced peak hip extension[103,101]. Results from the current study are consistent with the findings observed in participants with hip OA[101] and further support Hunt and colleagues (2013)[38] who also reported decreased peak hip extension and reduced sagittal plane hip ROM in individuals with FAI. Decreases in hip extension were observed during level ground and challenged walking, with moderate to large effect sizes at all inclinations compared to asymptomatic individuals. Calculated mean differences for peak hip extension showed that individuals with FAI walked with up to 7^{o} less peak hip extension during level ground and 10^{o} less extension during 10^{o} inclined walking compared to asymptomatic individuals. The mean differences during level ground walking are consistent with finding from Hunt and colleagues[38] who reported a 5^{o} reduction in peak hip extension in individuals with FAI compared to asymptomatic controls. The reductions in peak hip extension appear to have a progressive effect during the transition from FAI to moderate hip OA and severe hip OA. Eitzen and colleagues (2012)[101] reported mean differences of 10^{o} in individuals with moderate hip OA compared to asymptomatic

individuals, while Rutherford and colleagues (2015)[103] reported reductions of up to 25° in individuals with severe hip OA compared to asymptomatic individuals. These results indicate an apparent progressive deterioration in peak hip extension throughout the disease process and may further support the need for early intervention. Peak hip extension may be limited as an adaptive mechanism or movement strategy to reduce loading and compression forces in the hip joint[38], which is consistent with musculoskeletal modeling studies[98]. These studies suggest that higher anterior hip compression forces exist during increased hip extension[98] or the closed packed position of the hip, and that anterior soft tissue tension may pull the femoral head anteriorly into the acetabulum[98,38]. Interestingly, individuals with FAI had more hip flexion during level ground and challenged walking conditions but the differences between groups were consistently smaller than the differences observed for hip extension and not significant. Although contradictory to clinical evidence that individuals with FAI avoid increased hip flexion positions[23,24,25,26], these findings suggest that the magnitude of flexion needed to ambulate on a 10° incline may not surpass positions of impingement (i.e. excessive flexion) that are more likely observed during higher impact activities[143]. The walking demand at 5° and 10° inclines may require increased peak hip flexion for successful ambulation, resulting in little to no observable differences between groups.

Moderate effect sizes for reduced peak and mean gluteus maximus activation are consistent with the observed decreases in peak hip extension for individuals with FAI. A 25% reduction in gluteus maximus activation observed during 10° inclined walking suggests that individuals with FAI may have an inability or hesitation to recruit gluteus maximus and move their hip into a more extended position. This decrease in gluteus

maximus activation is not supported by previous research by Rutherford and colleagues (2018); however, notable differences in FAI sub-type criteria between studies exist[35]. Additionally, variability in participant age and analytical methods between studies may also contribute to the lack of consistency between findings.

Individuals with hip OA have also been shown to walk with altered neuromuscular activation patterns during level ground walking compared to asymptomatic indivudals[103]. Rutherford and colleagues (2015)[103] reported that individuals with severe hip OA walked with increased gluteus maximus activation during mid stance compared to asymptomatic individuals, and Diamond et al. (2020)[140] observed increased co-contraction of the hip flexors (adductor longus, iliacus, psoas major, rectus femoris, sartorius, tensor fasciae latae) and hip extensors (adductor magnus, biceps femoris long head, gluteus maximus, gluteus medius, semimembranosus and semitendinosus). Whether or not increased co-contraction of the hip flexors and extensors occurs in individuals with FAI during walking remains unclear and requires further research.

In the current study, neuromuscular activation of gluteus medius was also reduced in individuals with FAI compared to asymptomatic individuals, although findings were not statistically significant and inconsistent with previous literature[35]. Moderate to large effect sizes suggest that individuals with FAI may walk with reduced peak gluteus medius activation during level ground, inclined and declined walking conditions with up to 20% and 14% less activation compared to asymptomatic individuals during 10^{o} inclined and declined walking, respectively. Mean gluteus medius activation had very small effect sizes between groups with 3% less activation in individuals with FAI observed during 10^{o} inclined walking and no difference between groups during declined walking. This finding

suggests that individuals with FAI may retain prolonged gluteus medius activation during walking, but are unable to achieve peak muscle recruitment. Unlike the observed reductions in gluteus maximus activation, maintaining gluteus medius activation over the gait cycle may be consistent with progression to hip OA as individuals with severe hip OA have previously demonstrated increased overall, and early to mid-stance, gluteus medius activation compared to asymptomatic individuals[103].

Although group differences for frontal plane kinematics were not investigated, previous research has shown that individuals with FAI walk with less peak hip adduction[38]. The lack of effect sizes for mean activation may indicate that individuals with FAI are able to retain recruitment and prolonged activation of the hip primary abductor muscle, thus limiting movement into potentially painful or impinged positions. This phenomenon has also been seen within the hip OA population, where individuals with hip OA walked with increased mean activation and muscle co-contraction at the hip[144,140]. A comparison of the ensemble average waveforms between groups suggests a subtle yet dissimilar gluteus medius activation pattern across the gait cycle. Asymptomatic individuals demonstrated a bimodal activation pattern during the first 40% of the gait cycle that was not observed in individuals with FAI (Figure 4-3). Consistent with the pattern observed in individuals with FAI, a similar pattern was observed by Rutherford and colleagues (2015)[103] in individuals with severe hip OA, where individuals with severe hip OA walked with a more unimodal gluteus medius activation pattern. Importantly, the current study did not specifically analyze neuromuscular patterns, however the observed waveform differences between individuals with FAI and asymptomatic individuals may be reflective of hip OA mechanics.

Across the literature, patient-reported symptoms, function and quality of life are consistently worse in individuals with FAI compared to asymptomatic individuals; yet, inconsistent biomechanical and neuromuscular outcomes highlight a limited understanding of hip mechanics in this population. The findings of this thesis support the use of challenged walking as a strategy to improve interpretation of biomechanical and neuromuscular outcomes between individuals with FAI and asymptomatic individuals. The observations in the sagittal plane align with previous literature[37,38]; although, notable variability exists for neuromuscular activation of gluteus maximus and medius with earlier, but limited, findings[35]. However, these inconsistencies are not surprising given the recent shift in focus to understanding neuromuscular activity in this population. Moderate to large effect sizes suggest possible biomechanical and neuromuscular differences between groups are more apparent during challenged walking than level walking and may be associated with hip mechanics previously observed in individuals with hip OA.

To our knowledge, this study is the first to examine inclined and declined challenged walking; however, continued research is warranted including larger sample sizes. Using the current data, post hoc power analyses suggest that a sample size of 17 participants in each group would provide sufficient power using an alpha level of 0.5, target power of 0.8, a maximum sagittal plane ROM difference of 4^{o} and assumed standard deviation of 5. During 10^{o} walking with a maximum sagittal plane ROM difference of 7^{o} and assumed standard deviation of 6, a sample size of 13 participants per group was determined, and increased to 17 participants per group if a ROM difference of 5^{o} and standard deviation of 5 were used during 10^{o} decline walking. Therefore, 17 participants

(10 new participants) per group will be recruited when research restarts following the pandemic closure.

5.2 Limitations

The work conducted in this thesis does have limitations. The small sample size in each group prevented more conclusive interpretations for the data and underpowered the statistical analyses; therefore, non-statistically significant findings were expected. Although the a priori analyses were still performed, descriptive between group differences with bootstrapped 95% confidence intervals and effect sizes interpreted using Cohen's d were used to help interpret the available findings in the presence of a small sample size. Despite their size, the groups were well matched with no significant differences between group demographics. Radiographic evidence indicating hip pathology was available for both limbs in individuals with FAI but not available for the asymptomatic participants. The presence of FAI or structural hip pathology in this group is unknown. A previous systematic review including 2,114 asymptomatic hips reported that the prevalence of asymptomatic cam and pincer type morphology was approximately 37% and 67%, respectively[141]; therefore, the asymptomatic participants in the current study could have unknowingly had structural hip abnormalities. Due to the sample size in the current study, it was not feasible to assess for any potential sex differences between groups. Previous research has shown the potential for kinematic differences between sex, which could be reflected in the current results[106]. Last, the use of MVIC's as a normalization method has been questioned regarding its ability to elicit a true maximal effort[145]. In the current study, non-normalized, absolute strength measures indicated mean group differences up to 30Nm,

with larger strength values for individuals with FAI. These findings were not statistically significant; however, they do contradict previous literature[35,40,41,110].

5.3 Future Directions

The findings from the current work have identified several research directions that warrant further investigation. First, increasing the number of participants in each group to achieve statistical power is needed. The increased sample size will provide adequate power for performing the a priori analyses and facilitate interpretation of the data. Second, investigating the effect of arthroscopic surgery, a commonly used operative treatment in individuals with FAI, on immediate postoperative biomechanical and neuromuscular outcomes may help interpret the responsiveness and effectiveness of structural disease outcomes that may be associated with progression to hip OA. This thesis may have identified key biomechanical and neuromuscular outcomes between individuals with FAI and asymptomatic individuals that may be used to evaluate the responsiveness to the current surgical gold standard for FAI treatment[55]. Third, we plan to further investigate these challenged walking conditions with increased walking speed as well as increased incline and decline walking conditions. In doing this we can further identify these key biomechanical and neuromuscular outcomes related to the FAI-OA continuum. Fourth, investigating potential biomechanical and neuromuscular differences associated with the different structural subtypes of FAI would be novel. To date, little attention has been focused on investigating or comparing FAI subtypes and their possible effect on hip mechanics. These investigations may help interpret or clarify existing between study inconsistencies and perhaps better inform mechanical-related rehabilitation interventions for patients. Potential sex-related biomechanical and neuromuscular differences between

males and females with FAI needs investigation, as very limited evidence exists that explores these potential sex-related differences in individuals with FAI during walking and may further help tailor rehabilitation interventions. Last, the potential effect of pelvic motion on hip kinematics as well as the high prevalence of anterior pelvic tilt found in individuals with FAI warrants examination of pelvic tilt during standing calibration as a possible confounding variable in future biomechanical investigations.

5.4 Concluding remarks

The primary aim of this thesis was to assess the effect of inclined and declined challenged walking on individuals with FAI and asymptomatic individuals. Although statistical differences between groups were not observed, moderate to large effect sizes were found for sagittal plane hip ROM, peak hip extension, peak and mean gluteus maximus and peak gluteus medius activations. The sagittal plane findings were consistent with previous results for level ground walking in individuals with FAI, with similar and slightly larger responses for challenged walking. The observed findings also reflect similar hip mechanics previously observed in individuals with hip OA, suggesting a potential role for abnormal hip mechanics in hip pathology progression. The results of the study may inform future investigations examining challenged walking in individuals with FAI, as well as provide new insight into abnormal hip joint function that may facilitate tailoring rehabilitative strategies to mitigate the effects of hip pathology. The current study has generated new information relevant to the mechanisms associated with FAI and its impact on the hip musculoskeletal system. Further research examining the disease-related effects on biomechanical and neuromuscular outcomes is needed to help understand the hip joint mechanics and abnormal loading relevant to progression. Additionally, evidence to support

the use of surgical and non-surgical interventions in this clinical population is lacking; yet, an important research direction to help early prevention of OA-related personal and societal burdens as well as support long-term alleviations for the current strain on the healthcare system.

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APPENDIX A: Individual Participant Waveforms for individuals with FAI

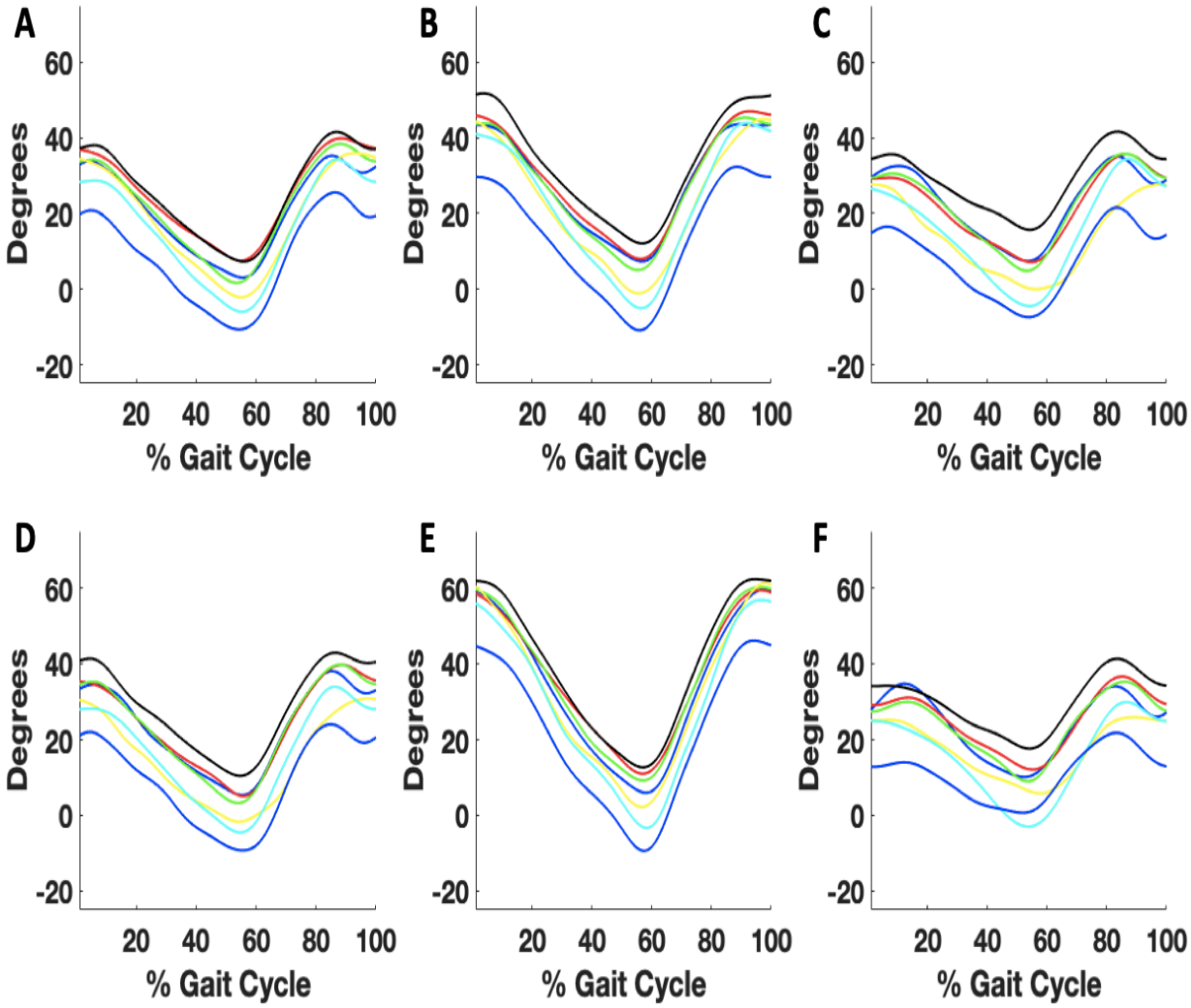

Figure A-5-1: Individual waveforms for individuals with FAI illustrating sagittal plane hip range of motion during (A) level ground pre challenged walking, (B) 5º inclined walking, (C) 5º declined walking, (D) level ground post challenged walking, (E) 10º inclined walking and (F) 10º declined walking. Each waveform is time normalized to percentage of the gait cycle.

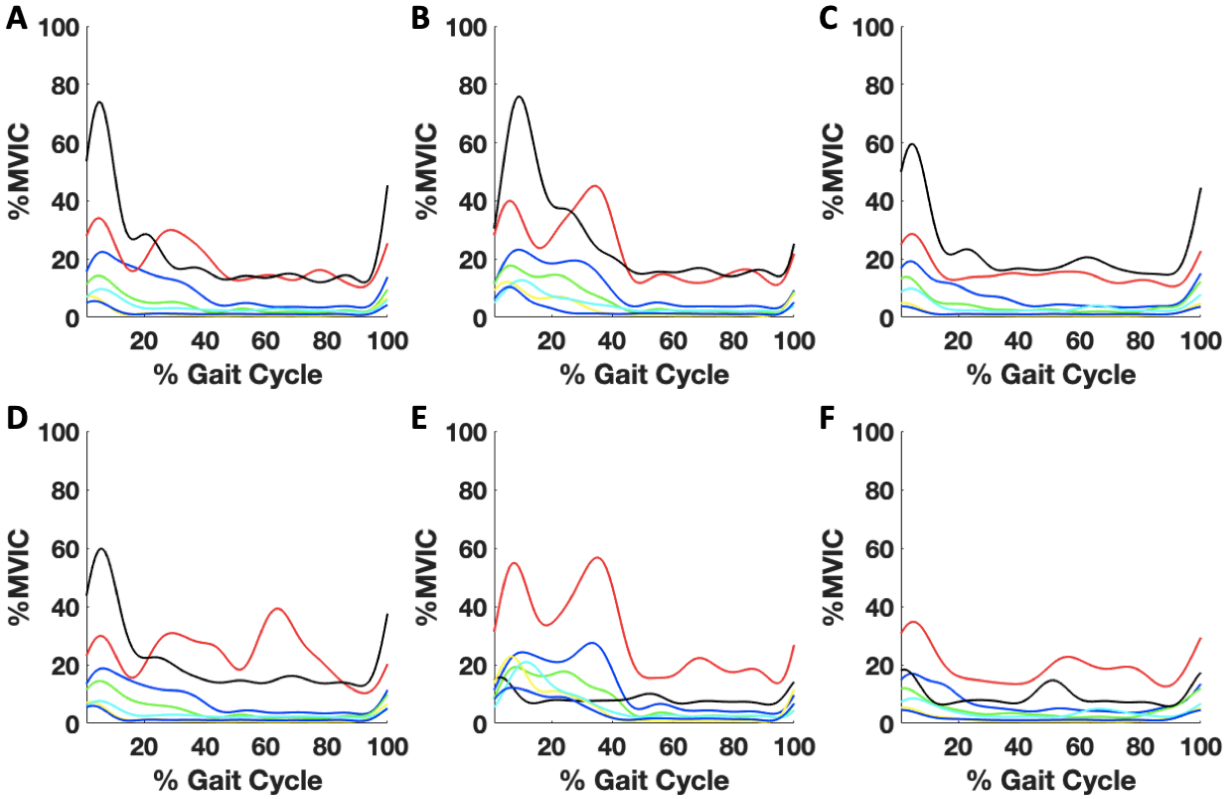

Figure A-5-2: Individual waveforms for individuals with FAI illustrating gluteus maximus activation expressed as a percentage of maximum voluntary isometric contraction (MVIC) during (A) level ground pre challenged walking, (B) 5º inclined walking, (C) 5º declined walking, (D) level ground post challenged walking, (E) 10º inclined walking and (F) 10º declined walking. Each waveform is time normalized to percentage of the gait cycle.

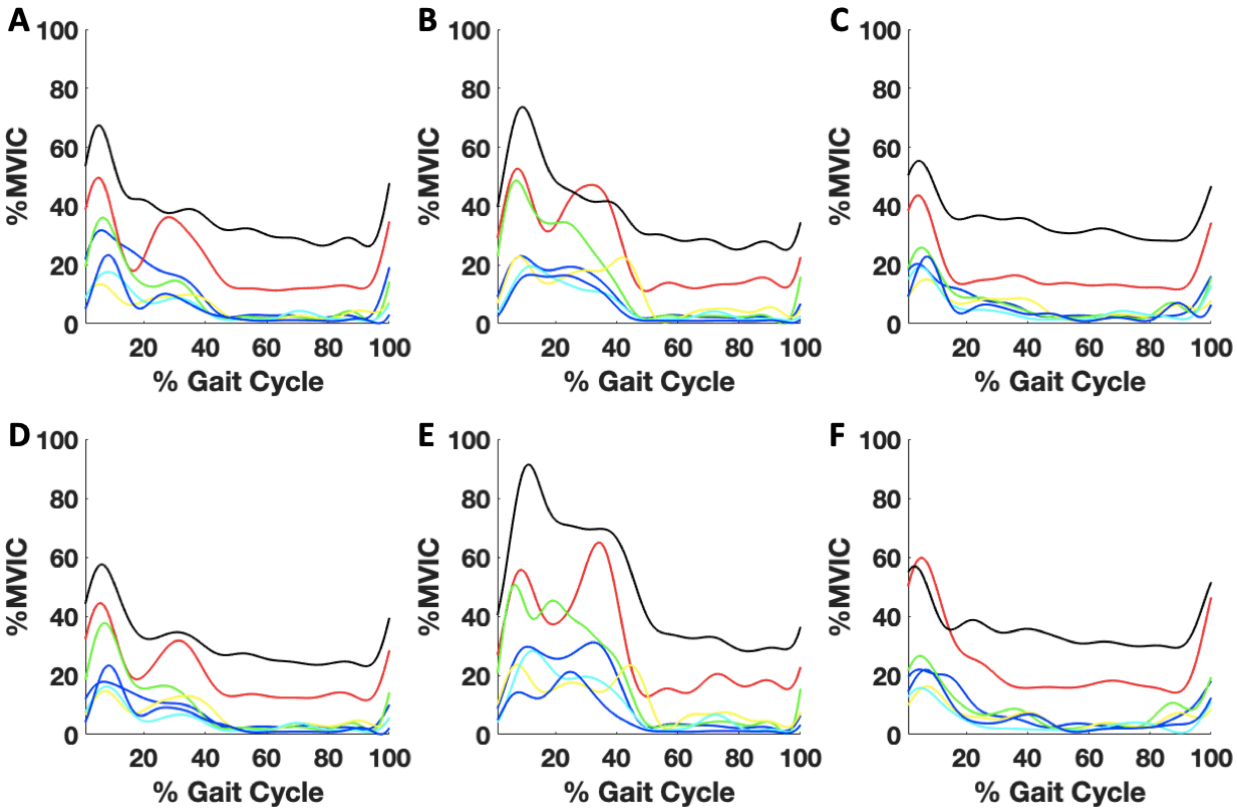

Figure A-5-3: Individual waveforms for individuals with FAI illustrating gluteus medius activation expressed as a percentage of maximum voluntary isometric contraction (MVIC) during (A) level ground pre challenged walking, (B) 5º inclined walking, (C) 5º declined walking, (D) level ground post challenged walking, (E) 10º inclined walking and (F) 10º declined walking. Each waveform is time normalized to percentage of the gait cycle.

APPENDIX B: Individual Participant Waveforms for asymptomatic individuals

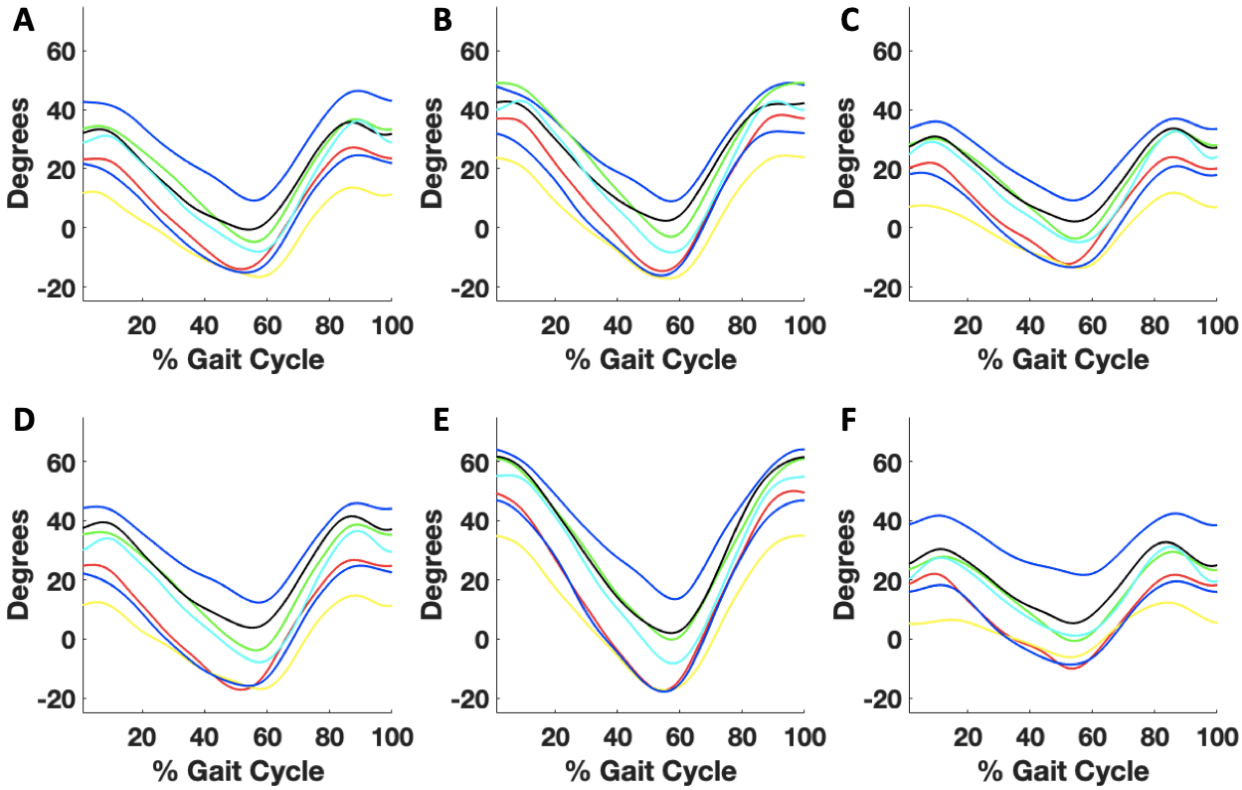

Figure B-1: Individual waveforms for asymptomatic individuals illustrating sagittal plane hip range of motion during (A) level ground pre challenged walking, (B) 5° inclined walking, (C) 5° declined walking, (D) level ground post challenged walking, (E) 10° inclined walking and (F) 10° declined walking. Each waveform is time normalized to percentage of the gait cycle.

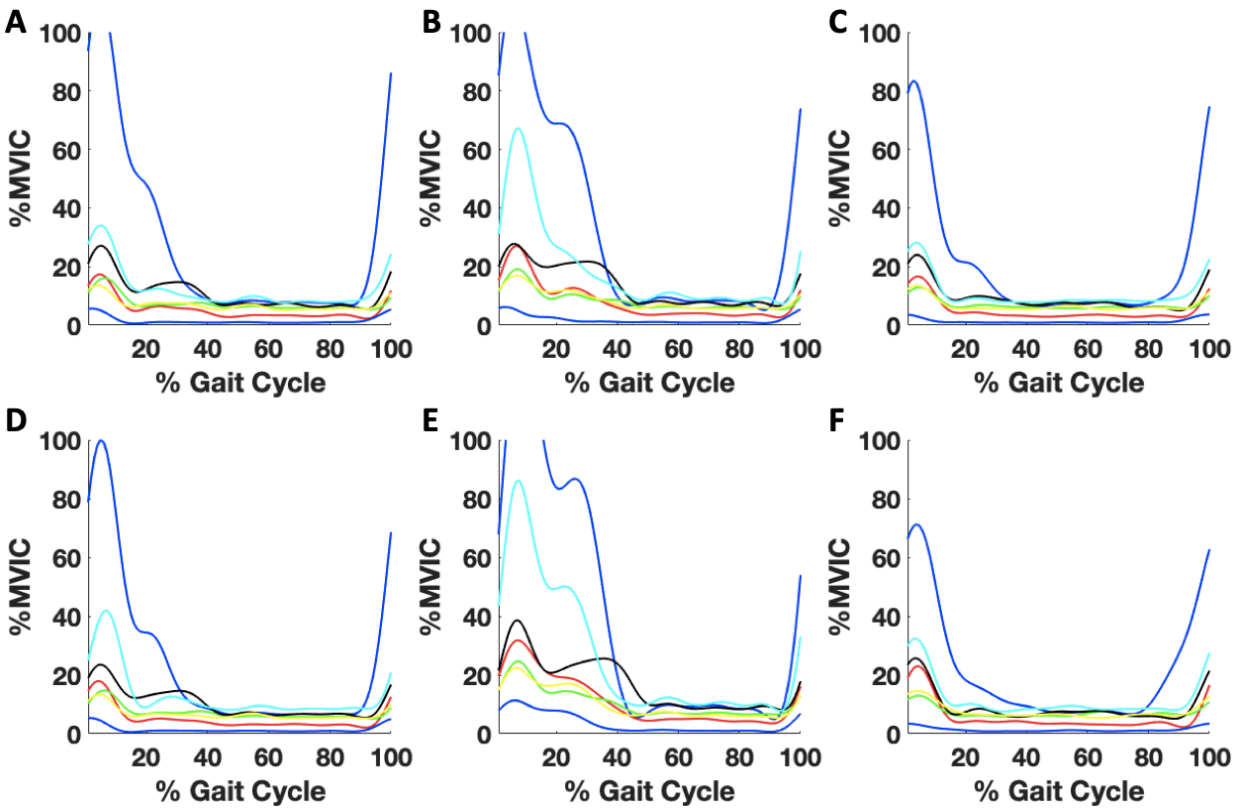

Figure B-2: Individual waveforms for asymptomatic individuals illustrating gluteus maximus activation expressed as a percentage of maximum voluntary isometric contraction (MVIC) during (A) level ground pre challenged walking, (B) 5° inclined walking, (C) 5° declined walking, (D) level ground post challenged walking, (E) 10° inclined walking and (F) 10° declined walking. Each waveform is time normalized to percentage of the gait cycle.

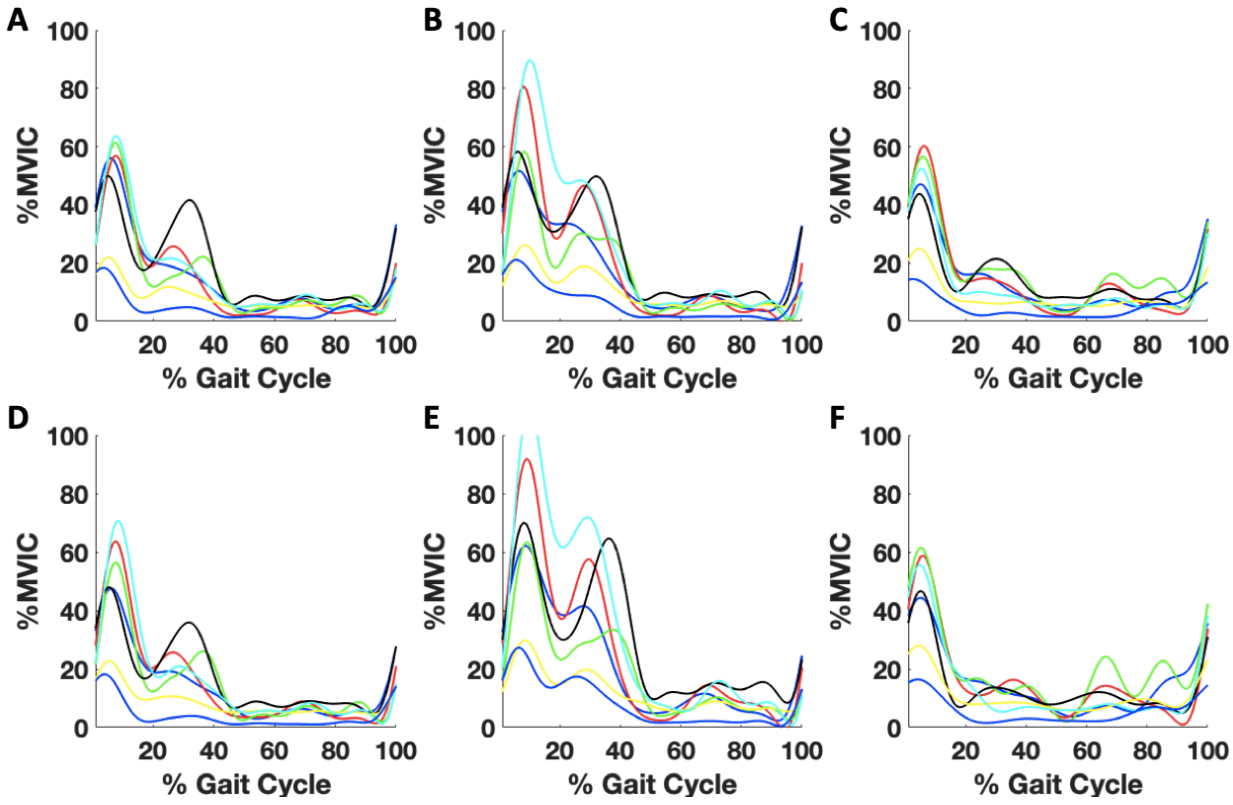

Figure B-3: Individual waveforms for asymptomatic individuals illustrating gluteus medius activation expressed as a percentage of maximum voluntary isometric contraction (MVIC) during (A) level ground pre challenged walking, (B) 5° inclined walking, (C) 5° declined walking, (D) level ground post challenged walking, (E) 10° inclined walking and (F) 10° declined walking. Each waveform is time normalized to percentage of the gait cycle.