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Impact of Parity on Gait Biomechanics

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Impact of Parity on Gait Biomechanics

A Thesis Presented

by

BEKAH P. STEIN

Submitted to the Graduate School of the
University of Massachusetts Amherst in partial fulfillment
of the requirements for the degree of

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Impact of Parity on Gait Biomechanics

A Thesis Presented

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ABSTRACT

IMPACT OF PARITY ON GAIT BIOMECHANICS

MAY 2020

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Background: Symptomatic knee osteoarthritis (OA) is an incurable condition that affects nearly 50% of adults, and women are twice as likely as men to develop OA. Throughout pregnancy, women experience large changes in morphology and gait mechanics, as well as changes in joint loading. It is possible these adaptations could cause lasting changes postpartum, which may potentially contribute to initiation of OA, thereby increasing the overall risk of OA for women.

Purpose: This exploratory study looked to identify differences between lower limb gait mechanics of healthy nulliparous women and healthy parous women.

Methods: 28 healthy female participants (14 parous, 14 nulliparous) were recruited for the study. Nulliparous participants had never given birth to a child, and were self-reported not pregnant. Parous participants had given birth to at least one full term infant (37 – 42 weeks) without complications between one to five years before data collection. Kinematic and kinetic data was collected for the lower body, using motion capture and in-ground force plates. Participants completed one quiet standing trial, and walked over-ground through the motion capture space at their preferred, fast, and set walking speeds (1.4 m/s). An ANOVA was performed to test if there were significant differences in between groups.

Results: Q angle did not differ between groups. There was a significant main effect of group indicating a larger knee flexion angle at toe off ($p = 0.060$), smaller knee extension moment at

heel strike ($p = 0.0006$), smaller first peak knee flexion moment ($p = 0.040$), and smaller peak hip adduction moment for the parous group compared to the nulliparous group ($p = 0.003$).

Conclusions: Our data revealed a decrease in the moments experienced, which could possibly lead to degradation of cartilage due to under loading of the joint. We think this may be an indication that pregnancy could increase risk of OA, and therefore more research into this possibility is warranted.

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CHAPTER I

INTRODUCTION

Background on Knee OA

Symptomatic knee osteoarthritis (OA) is an incurable condition that affects nearly 50% of adults aged 45-85 years (Murphy et al., 2008). The knee is the most common place to be diagnosed with OA, and women are more likely than men to develop knee OA. This difference can be seen in Figure 1. By 2030, knee OA could affect up to 36 million Americans, as older adults are projected to number greater than 19.3% of the population (Report on aging, 2014). OA is the degeneration of joint cartilage and the underlying bone (Figure 2). Pain associated with OA

can often lead to significant functional deficits, therefore restricting patients from normal day-to-day functioning. OA is often associated with joint inflammation, joint stiffness, and decreased mobility.

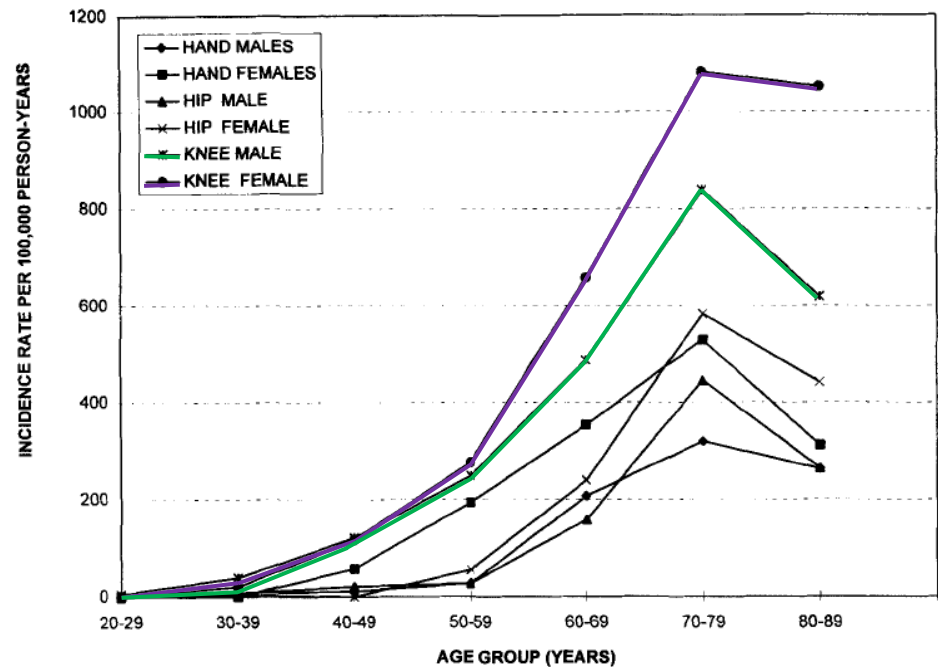


Figure 1: Incidence rate of men and women for different types of OA
Adapted from Oliveria et al., 2006

Along with the functional difficulties presented by OA, the disease is associated with increases in comorbidities such as cardiovascular disease and ischemic heart disease (Osteoarthritis: A Serious Disease, 2016). To date, there are no drugs which can stop, prevent, or

slow the progression of OA. Since there is no known cure or treatment to prevent the need for a total joint replacement, health care costs associated with OA are extremely high (Osteoarthritis: A Serious Disease, 2016). In the United States it is estimated that medical expenditures for people with OA averaged \$340 billion each year from 2008 to 2011 (The Burden of Musculoskeletal Diseases in the United States, 2014).

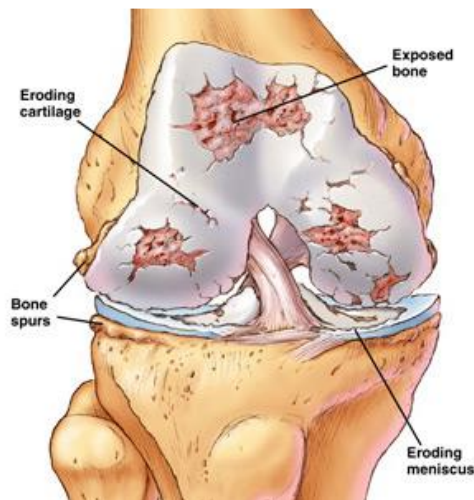


Figure 2: A depiction of an osteoarthritic knee
Adapted from Oxford University Hospital
2019, Retrieved from <https://www.ouh.nhs.uk/hinandknee/information/knee/arthritis.aspx>

Not only do people with OA experience pain, comorbidities, and huge costs, they also eventually will experience loss of independence and quality of life. The currently approved treatments are aimed only at reducing pain, rather than improving the condition of the patient's knee. These available drugs have many adverse effects and can increase the risk of comorbidities (Trelle et al., 2011). The last-resort treatment is a total knee replacement, and involves a

replacement of the entire knee joint with an artificial joint. This requires a major surgery, hospitalization, and physical therapy thereafter. Additionally, this frequently results in no less pain for the patient, and the artificial joints have a limited lifespan (Sakellariou et al., 2016). This means there is a huge need for non-surgical treatments. To improve the effectiveness of non-surgical options, there is a need to understand factors leading to the initiation of knee OA. Because there is no cure or disease modifying treatment for OA, prevention of initiation is critical.

OA Risk for Women

Women are twice as likely as men to develop OA (Oliveria et al., 1995). While the overall number of people with OA is expected to rise greatly in the coming years, the portion of those diagnosed who are women is expected to become a continually

increasing percentage of the overall OA population as can be seen in Figure 3 (Center for Disease Control and Prevention, 2003). This means women are at a higher risk for OA and their risk of OA will continue to increase. The prevalence of women diagnosed with OA is consistently higher than men diagnosed with OA. This is true throughout the progression of the disease, and continuing through to total knee replacement.

Body Mass Index (BMI) is a known risk factor for OA. The need for total knee replacement increases with increasing BMI, and for every BMI level, the percentage of women who need a total knee replacement is higher than that of men (National Institutes of Health Osteoarthritis Initiative). This means for a man with the same BMI as a woman, the woman is at a higher risk of needing a total knee replacement. Women additionally have higher losses of function and disability associated with OA (Keefe et al., 2000; Parmelee et al., 2012). Parous

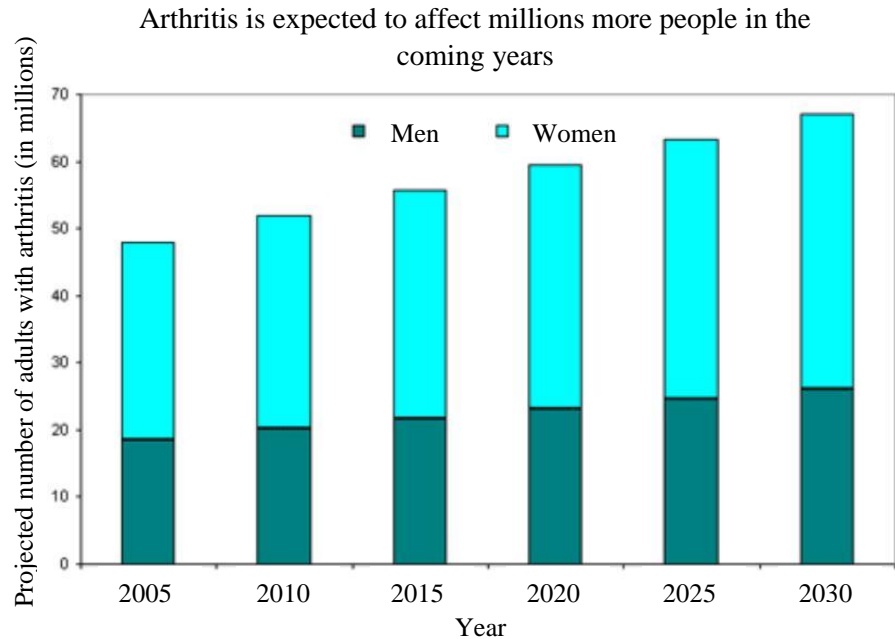


Figure 3: Projected prevalence of doctor-diagnosed arthritis among U.S. adults aged 18 years and older.

Adapted from Center for Disease Control and Prevention, 2003

women, women who have had children, on average have higher BMIs than nulliparous women, women who have not had children (Bobrow et al., 2013). Older parous women have been shown to have lower cartilage volume in the knee, as compared with older nulliparous women. This suggests a relationship between parity (having experienced pregnancy) and knee joint health (Wei et al., 2011). It is possible that the higher risk of OA in women may be driven by an additional risk factor men cannot experience: pregnancy.

In the United States, 57% of women have had one or more children throughout her lifetime (United States Census Bureau, 2017). Throughout these pregnancies, women experience large changes in morphology as well as changes in gait mechanics such as increased distensibility of the pelvic floor, greater anterior pelvic tilt, medial-lateral instability, increased base of support, decreased navicular height, and greater thoracic extension at heel strike (Alvarez et al., 1988; Branco et al., 2014; Branco et al., 2015; Butler et al., 2006; McCrory et al., 2014; Ponnappula et al., 2010; Schauburger et al., 1995; Van Veelen et al., 2013; Wei et al., 2011). These changes could cause lasting adaptations postpartum, and increase the overall risk of OA for women. No widely available disease modifying treatments exist to stop or reverse knee OA structural changes, and therefore research remains critical to aim to reduce the overall societal burden of knee OA, particularly in women. Thus, due to this inequality, understanding the factors that may increase risk for OA initiation in women is specifically necessary.

Research has shown the risk discrepancy of OA to women may be partially due to hormonal changes experienced by women such as menopause, or the morphological differences between men and women (NIH Conference, 2000; Srikanth et al., 2005). In the literature, it remains unclear if or how pregnancy and childbirth impact OA risk. However, greater risk of OA in women could also be due to differences in body mass index (BMI) or physical activity

level. Low physical activity is on the rise within the ranks of OA risk factors, and increasing in the general population as well (Risk factors and burden of osteoarthritis, 2016; Osteoarthritis: A Serious Disease, 2016). In low-income countries, 12% of men and 24% of women were insufficiently physically active, and in high-income countries, 26% of men and 35% of women were insufficiently physically active (Physical Activity. WHO Fact Sheet, 2015). In both cases, women do less physical activity, putting them at a higher risk for OA (Chronic rheumatic conditions, 2018). The inequality of OA risk for woman is a global issue. The more we know about the variables which put women at higher risk, the closer we will be to understanding OA. Understanding which factors cause women to be at a higher risk could help researchers further understand the initiation and progression of OA.

Theory of OA Initiation

Mechanical loading during activities of daily living is a stimulus for healthy cartilage remodeling in non-OA afflicted knees (Andriacchi et al., 2004; Felson, 2013). Healthy cartilage adapts to have greater cartilage thickness in locations which

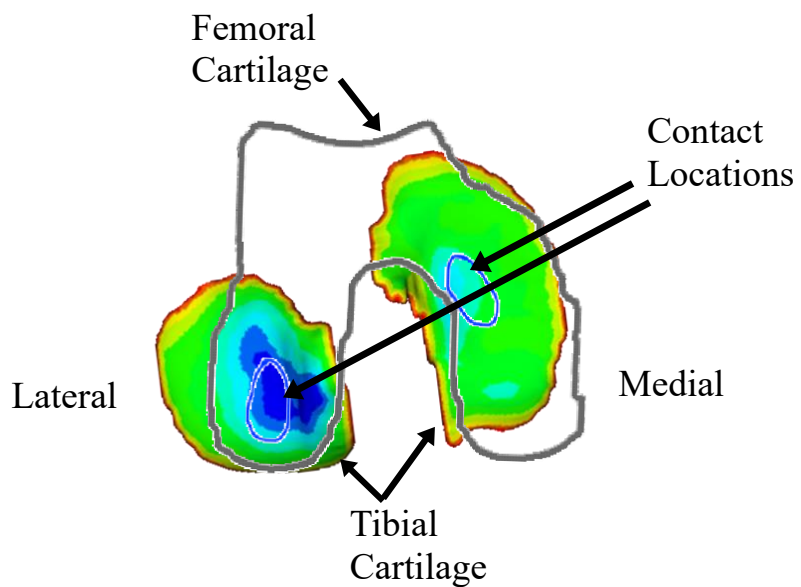


Figure 4: Depiction of tibial cartilage (colored areas, where blue is thicker cartilage) and femoral cartilage (grey line), and their contact locations
Adapted from Andriacchi et al., 2009

experience the greatest loading (Andriacchi et al., 2009; Koo & Andriacchi, 2007; Koo et al., 2011). In Figure 4, the blue spots represent thicker cartilage, and the grey circles represent the

contact locations, showing that these two align nicely in a healthy knee joint. During the gait cycle, the highest loads occur when the knee is near full extension at heel strike. The greatest cartilage thickness in the knee is found at the tibiofemoral contact locations when the knee is near full extension (Koo et al., 2011). Changes in contact locations are believed to be a primary contributor to the initiation of OA (Andriacchi et al., 2004; Andriacchi & Mündermann, 2006). The current accepted theory of OA initiation is shown in Figure 5. A healthy knee exists on the homeostatic left circle. Due to some change in joint mechanics, OA is initiated and the knee is then driven over to the right spiral of joint degradation. Abnormal knee joint kinematics can result in changes in tibiofemoral contact locations. With these changes, high loading occurs in areas which under normal circumstances are not heavily loaded (Andriacchi & Mündermann, 2006). Cartilage is a slowly adapting tissue, due to its limited access to nutrients, therefore as the cartilage attempts to adapt in the newly heavily loaded contact areas, these contact areas can display fibrillation (Andriacchi et al., 2004). This shift of tibiofemoral contact locations, and

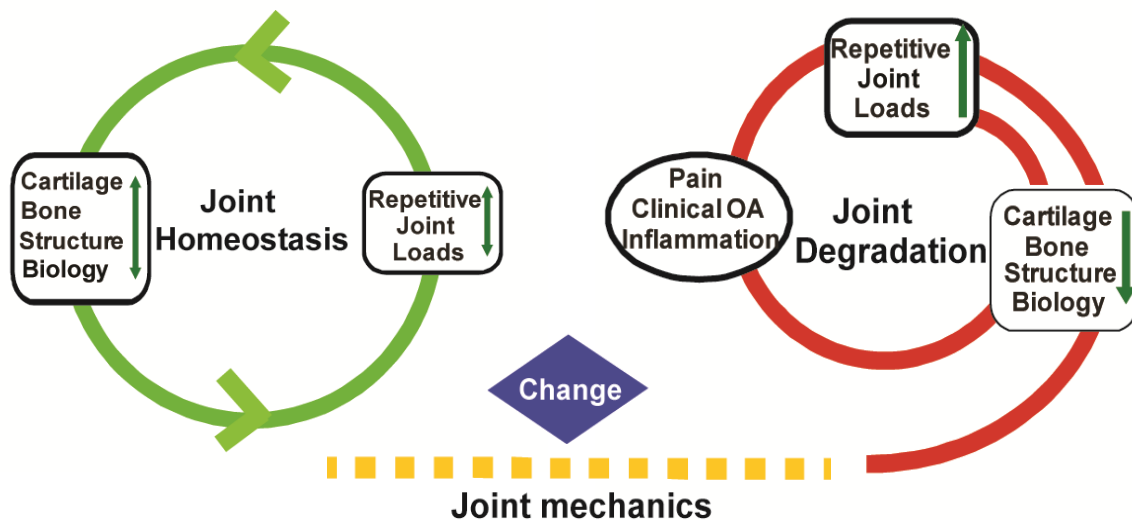


Figure 5: Depiction of osteoarthritis initiation theory
Adanted from Andriacchi & Münderman. 2006

these new contact areas not being able to adapt to the sudden increase in mechanical loading is believed to be the initiation phase of OA (Andriacchi et al., 2004; Andriacchi & Favre, 2014).

It is well understood that ACL injuries, specifically tears and reconstructions, lead to higher incidence of OA later in life. This is thought to be due to the change in mechanics which occurs due to the injury or due to the surgery (Andriacchi et al., 2004; Felson, 2013; Lohmander et al., 2007). When the ACL is reconstructed, the position of high stress zones in the cartilage shift. This shift in contact locations is likely the cause for increased risk of OA in people with ACL injuries, and more specifically ACL reconstruction. It is possible, that similar to an ACL injury, pregnancy causes hormone induced ligament changes, and the pregnant woman experiences significant mechanically increased stresses, and alters the mechanics of the knee. The change in mechanics of gait and morphology during pregnancy is fairly well documented, but the lasting changes are not. Documenting whether or not there are lasting changes due to pregnancy, and if these lasting changes correlate with mechanical changes associated with OA will help researchers gain a better understanding of OA initiation.

Biomechanical Changes with Pregnancy

Among other changes, during pregnancy women experience lumbar lordosis, posterior upper body tilt, increased sagittal pelvic tilt, relaxation of the ligaments, and a host of hormone changes (Dumas et al., 1997; Franklin et al., 1998; Zarrow et al., 1954). With the many fast-paced changes women experience throughout pregnancy, it is not surprising that some of these changes are long-lasting. Chu et al. showed 4 months postpartum there are lasting changes to ligament laxities in the knee (Chu et al., 2018). There is also evidence showing lasting changes in the structure of the feet at 8 weeks postpartum and distensibility of the pelvic floor at 6 months postpartum (Alvarez et al., 1988; Van Veelen, 2014). Due to the lack of motion capture research

exploring changes postpartum, there is a large gap in understanding of the lasting implications pregnancy may have on movement mechanics and future injury risk. The current literature suggests there are lasting changes in the feet, pelvis, and knees. It is possible some of the lasting morphological changes may cause gait changes. These changes in gait may align with changes associated in the literature with OA initiation, such as increases in the peak knee adduction moment, peak flexion moment, or the internal rotation moment. Many studies looking at changes during pregnancy have included data on women postpartum; however, frequently only measuring for changes shortly after birth (Marnach et al., 2003; Schauburger et al., 1996; Wurdinger et al., 2002). The studies which do measure further into the postpartum period suggest there may be lasting morphological changes due to childbirth (Alvarez et al., 1988; Branco et al., 2015; Chu et al., 2018; Van Veelen et al., 2013; Wei et al., 2011). The impact of these morphological changes postpartum on gait mechanics remains unclear.

Possible Increased OA Risk

Kinematic changes at the knee, such as changes associated with altered ligament stiffness, can cause degenerative changes to cartilage (Andriacchi et al., 2004). Joint laxity has been shown to be related to an increased incidence of osteoarthritis (Scott et al., 1979; Sharma et al., 1999). Chu et al. (2018) found that after pregnancy, multiplanar ligament laxity in the knee persisted, and there were lasting changes in compliance at the knee. These lasting morphological changes, if they impact gait mechanics, could potentially be one of the factors that cause women to be at increased risk for lower body injuries and osteoarthritis (Chu et al., 2018; Scott et al., 1979; Sharma et al., 1999). Older women who have had children have been shown to have lower cartilage volume, as compared with older women who have never had children (Wei et al., 2011). This suggests there may be a link between cartilage changes and mechanical changes

during or due to pregnancy (Wei et al., 2011). The average age of women during their first birth in the United States is 25 years old, so the vast majority of these women's lives are experienced postpartum (National Vital Statistics Reports, 2002). If there are lasting morphological changes during the postpartum period, there are likely lasting changes in gait mechanics as well. To date, there have been no motion capture analyses comparing gaits of non-pregnant nulliparous woman (women who have never had a baby) to the gait of parous women (women who have had one or more children) to our knowledge.

Overall Hypothesis

We hypothesized there would be a measurable difference in the kinetics and kinematics between the nulliparous and parous groups

- **Aim 1:** Quantify the impact of parity (the state of having borne offspring) on 3D lower extremity joint kinematics in women
 - H 1.1: Parous women, compared to nulliparous women, will have increased peak ankle eversion and peak hip flexion over stance, and increased knee flexion local maxima during the first half of the stance phase.
 - H 1.2: Parous women, compared to nulliparous women, will have reduction of the mean internal rotation angle of the knee across stance phase, and a reduction in the range of motion of the internal rotation angle.

Aim 1, hypotheses 1.1 is supported by research which states women experience more everted feet after pregnancy, pelvic floor distensibility causing core strength issues, and there is decreased ligament laxity in the knee (Alvarez et al., 1988; Van Veelen et al., 2014; Chu et al., 2018). With pelvic floor distensibility causing decreased core strength, lumbar lordosis could

continue after pregnancy. This would cause anterior pelvis tilt, increasing flexion of the hip and decreasing extension of the hip. Aim 1, hypotheses 1.2 is supported by research which suggests ligaments in the knee experience lasting changes postpartum due to pregnancy. We expect the mean internal rotation angle and the range of motion of the internal rotation angle will be reduced for parous women because they may compensate for an increase in ligament laxity by co-contracting, decreasing the rotational movement of the knee. Hamstring co-contraction can increase stability of the knee by decreasing rotation of the tibia, and we believe this may be the case with the parous group (Hirokawa et al., 1991). This is supported by ACL research showing as absence or changes in the ligament properties results in kinematic changes. Known major factors in the progression of knee OA are rotational changes, such as those which follow ACL injury (Andriacchi et al., 2004).

- **Aim 2:** Quantify the impact of parity on 3D lower extremity joint kinetics in women
 - H 2.1: Parous women, compared to nulliparous women, will have increased peak knee adduction moment, flexion moment, and internal rotation moment during walking.

Aim 2 and respective hypothesis are supported by the general knowledge that changes in the ground reaction force vector position relative to the joint center can alter the kinetics experienced in the hip, knee, and ankle. Therefore, changes in the center of mass position and lower extremity alignment due to lasting impact of pregnancy on the pelvis and foot-ankle motion may change the knee joint kinetics by altering the GRF vector position relative to the joint center. Changes due to GRF changes are important, as they may contribute to previously reported reduction in volume of cartilage in the tibial compartment of the knee in women who have had children (Chu et al., 2018; Wei et al., 2011). Increases in the peak knee adduction moment during walking

provide an external measure which has been shown to be able to gauge changes in cartilage thickness. This is because changes in the peak knee adduction moment influence the distribution of the force between the medial and lateral compartments of the knee (Andriacchi et al., 2009).

- **Aim 3:** Quantify the impact of parity on static alignment in women
 - H 3.1: Parous women, compared to nulliparous women, will have abnormally large Q angles.

Aim 3 and respective hypothesis is supported by research which states abnormal Q angles are associated with degenerative changes in the knee (Huberti, 1984). A change in the Q angle means changes in alignment of the knee. This change in knee alignment could result in changes of the tibio-femoral contact location changing during walking, which as has been previously stated, can lead to the initiation of OA.

Quantifying the lower body change from nulliparous to parous walking will help to better understand some of the lasting changes due to pregnancy, and understand further the possible connection between parity and OA. This study will help continue the exploration into the field of Biomechanics within the specialty of women's health. Access to women before they become pregnant, as well as during and after pregnancy, is limited due to the unpredictability of pregnancy. Because of this limitation, studies frequently use the first trimester data or the postpartum data as the comparative control (Bird et al., 1999; Dumas & Reid, 1997; Franklin & Conner-Kerr, 1998; Lou et al., 2001). This is an issue due to the large release of hormones experienced during the first trimester (Schauberger et al., 1996).

If pregnancy is a risk factor for OA, it could affect more than a quarter of our population directly (United States Census Bureau, 2017). Having a more thorough understanding of how

pregnancy could affect women postpartum might change how doctors treat parous women, as well as women who chose not to have children. Such knowledge could inform the development of injury prevention techniques, and could help impact the understanding of some diseases and injuries such as osteoarthritis (Chu et al., 2018). If biomechanical changes are detected, and pregnancy is found to be a risk factor for osteoarthritis, it is possible that rehabilitation could be developed for women during or after birth to help to attempt to counteract the negative effects of pregnancy before they cause harm. Future research could explore how long after birth these changes take effect, and whether or not exercise before, during, or after this happens is beneficial. This could possibly even help physicians know when it is healthy for parous women to return to their previous activity level.

CHAPTER II

LITERATURE REVIEW

Introduction

In the United States, 57% of women have had one or more children throughout her lifetime (United States Census Bureau, 2017). Throughout these pregnancies, women experience large changes in morphology as well as changes in gait mechanics (Alvarez et al., 1988; Branco et al., 2014; Branco et al., 2015; McCrory et al., 2014; Ponnappula et al., 2010; Schauburger et al., 1995; Van Veelen et al., 2013; Wei et al., 2011). With the many fast paced changes women experience throughout pregnancy, it is possible that some of these changes may be lasting. Due to the lack of motion capture research in this area there is a large gap in understanding of the lasting implications pregnancy may have on movement mechanics and future injury risk. One of the potential musculoskeletal diseases pregnancy may increase overall risk factor for is Osteoarthritis (OA) (Chu et al., 2018). Symptomatic knee OA is an incurable condition that affects nearly 50% of adults aged 45-85 years, and women are twice as likely as men to develop OA (Murphy et al., 2008; Oliveria et al., 1995). While the overall number of people with OA is shown to rise greatly in the coming years, the portion of those diagnosed who are women is expected to become a continually increasing percent of the overall OA population (Center for Disease Control and Prevention, 2003). This means women are at a higher risk for OA, and their risk of OA is continuing to increase.

Osteoarthritis

OA is characterized by the degeneration of joint cartilage and the underlying bone. A major symptom of OA is pain, and therefore OA largely restricts patients from normal day-to-day functioning. The most common symptoms associated with knee OA are pain, joint inflammation, joint stiffness, and decreased mobility. Along with the difficulties presented by OA, the disease is associated with increases in comorbidities such as cardiovascular disease and ischemic heart disease (Osteoarthritis: A Serious Disease, 2016). To date, there are no drugs which can stop, prevent, or restrain the progression of OA. The current approved treatments are aimed only to reduce pain, rather than improve the condition of the patient's knee. The drugs prescribed to help with OA leave the patient with more adverse effects to worry about, and can increase risk of comorbidities (Trelle et al., 2011). The last resort treatment for OA is a total knee replacement which requires major surgery, hospitalization, and physical therapy afterward. Additionally, this frequently results in no less pain for the patient, and the artificial joints have a limited lifespan. This indicates there is a huge need for non-surgical treatments and more importantly prevention of knee OA before treatment is needed. Before large-scale intervention programs can be developed, a greater understanding of the factors leading to the initiation and progression of knee OA is needed, particularly in women.

Pregnancy may have lasting effects on ligament mechanical properties and functions in-vivo. A strong link between ligament function in-vivo and knee OA initiation has previously been established in individuals with a history of Anterior Cruciate Ligament (ACL) injury and repair. Due to the possibility that pregnancy has lasting effects on ligaments in the knee postpartum, it is important to understand what is already known about the relationship of OA to changes in the ligaments of the knee. In the literature, there is evidence which links ACL tears to

the development of premature knee OA in young adults (Felson, 2013; Lohmander et al., 2007). Lohmander and colleagues found a mean rate of more than 50% of people with ACL injury had OA 10 to 20 years after the injury (Lohmander et al., 2007). The main role of an ACL is to resist anterior-posterior translation as well as internal-external rotation of the knee joint. Patients with an ACL tear or who have had ACL reconstruction show distinct changes to both the anterior-posterior displacement and internal-external rotation, as compared to their healthy knee (Andriacchi et al., 2009; Andriacchi et al., 2004). The changes experienced due to ACL injury are associated with changes in gait mechanics, and the initiation of OA is believed to be associated with changes in gait mechanics (Andriacchi et al., 2009). Furthermore, the research suggests tibial cartilage thinning occurs in the region associated with the patient's specific kinematic changes (Andriacchi et al., 2009). Kinematic changes following an ACL injury have been associated with patterns of cartilage thinning in young adults as well as older adults. Some of the commonly found changes with ACL tears are increased knee flexion, increased anterior femoral displacement at heel strike in walking, and tibial rotation with respect to the femur during the stance phase of gait (Andriacchi & Favre, 2014; Shabani et al., 2014; Favre et al., 2014; Netravali et al., 2010). The kinematic changes experienced due to ACL injury have been shown to be linked to the initiation of OA (Koo et al., 2010). If these kinematic changes due to ACL tears are increasing the risk of OA, it is possible that other changes to the ligaments in the knee, such as those possibly experienced by parous women, might cause a similarly increased risk.

Changes During Pregnancy

During pregnancy, women go through large morphological and hormonal fluctuations. These alterations, coupled with the changes in mechanical load, due to both morphological changes and weight gain, could be indicative of lasting changes. Although the largest spike in most hormones is the first trimester, there are constant hormonal influences throughout pregnancy and into postpartum, due to lactation. Because of this, many women develop musculoskeletal disorders postpartum due to these hormones, as well as the mechanical and ergonomic stresses of pregnancy, child care, and related activities (Borg-Stein & Dugan, 2007).

Some of the known postural changes during pregnancy can be seen in Figure 6, and include lumbar lordosis, posterior upper body tilt, and increased sagittal pelvic tilt. The increased sagittal pelvic tilt is an adaptation believed to deal with the ventrally driven center of gravity (Franklin & Conner-Kerr, 1998). Alterations of mechanics such as these require weight bearing joints to adapt by absorbing additional force

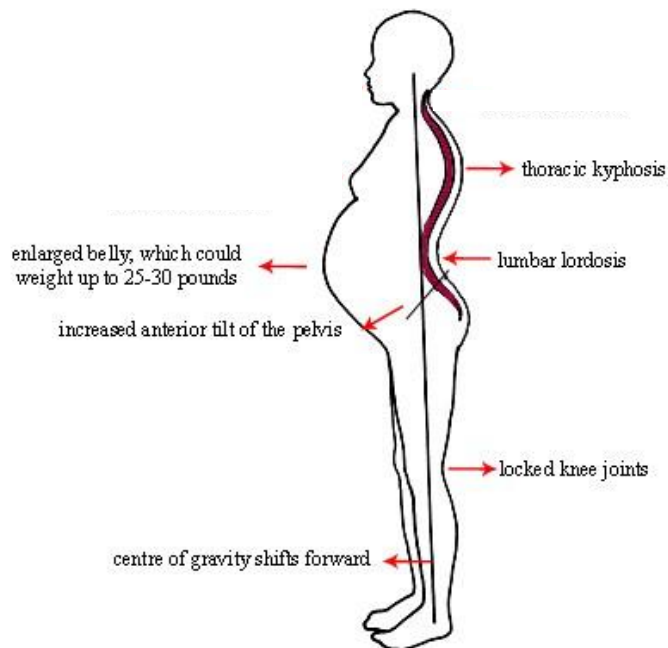


Figure 6: Postural changes experienced during pregnancy
Adapted from Mike Luque Training, by M. Luque, 2016, Retrieved from mikeluque.training/gyrotonic-training/shoulder-release

(Ponnappala & Boberg, 2010). During pregnancy, the change in the center of gravity anteriorly and weight gain also cause relatively rapid gait changes. Bird and colleagues found a 30% increase in the base of support between the first trimester and the third trimester, meaning the

women walked with a wider stride width toward the end of pregnancy (Bird et al., 1999). Alterations of the base of support in gait can cause changes in kinematics and kinetics of the lower extremities. Because these alterations are sustained over many months, the women may have experienced lasting changes attributable to the temporary changes in gait.

Kinetic changes during pregnancy may occur due to weight gain, and placement of this weight. While weight gain is natural and healthy during pregnancy, an increase in weight of 20% may increase the force on a joint by as much as 100% (Borg-Stein & Dugan, 2007). Not only do pregnant women gain weight, the weight gained is primarily focused in one area, the anterior portion of the pregnant women's trunk. Hyperlordosis during pregnancy may be the result of forces induced by this concentrated area weight gain. Hyperlordosis caused by pregnancy may be exaggerating anterior pelvic tilt, because the sacroiliac joints resist this forward rotation due to the pregnant belly. Both forward rotation of the pelvis and hyperlordosis increase as the sacroiliac ligaments become relaxed, and as pregnancy progresses, this can cause a widening of sacroiliac joints (Ritchie, 2003). Additionally, this shift in center of gravity causes pregnant women to hyperextend their knees to maintain a balanced and upright posture (Yoo et al., 2015).

As pregnancy progresses from the first trimester to the third, and into the postpartum period, postural stability has been shown to decline. A study by Butler et al. in 2006 found 25% of the pregnant women experienced a fall during their pregnancy (Butler et al., 2006). This fall rate is akin to the fall rate of the > 65-year-old general population (Dunning et al., 2003). Although it has been shown that women widen their base of support, this still does not seem to compensate for the large morphological changes pregnant women experience (Bird et al., 1999).

Relaxation of the pubic symphysis joint is a natural part of a healthy pregnancy to help the pelvis expand for a safe delivery. This relaxation is thought to be related to hormonal changes due to pregnancy. In the first trimester of pregnancy, women go through the largest

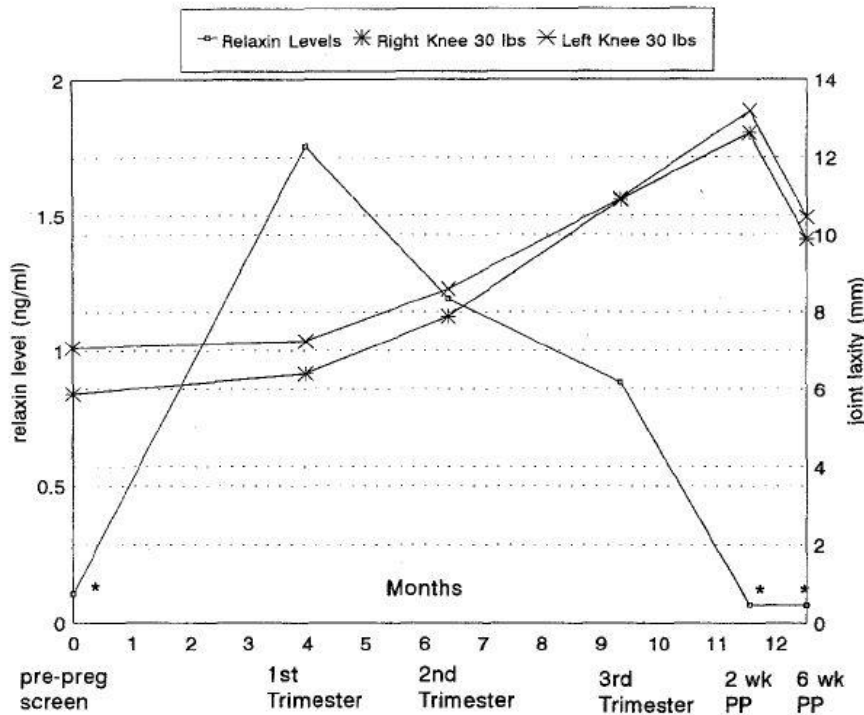


Figure 7: Relaxin levels and knee joint laxity measures during pregnancy.

* Not detectable in most samples, PP: Postpartum

Adapted from Schauburger et al., 1995

during pregnancy (Calguneri et al., 1982). This peak occurs during the first trimester, as can be seen in Figure 7. Also seen in Figure 7 is the peak knee ligament laxity at birth. This peak having a delayed effect compared to the relaxin peak is thought to be due to the delay in the increase in mechanical strains as weight gain continues throughout pregnancy (Schauburger et al., 1995). If this is true, it would indicate that relaxin has effects which last long enough to still be affecting ligaments months after its peak.

High levels of relaxin during pregnancy and nursing have been associated with pelvic pain, and women who experienced incapacitating pain had the highest levels of relaxin

hormone spike they will experience throughout their pregnancy. One of the hormones in this spike is called relaxin. Relaxin is known for its association to the relaxation of ligaments. A 10-fold increase of relaxin weakens soft tissue structures and increases joint flexibility

(MacLennan et al., 1986). Relaxin has also been shown to affect not only the ligaments, but to have effects on other tissues. In mice, it has been shown to induce cartilage and bone erosion, and cause transformation of hyaline cartilage caps into fibrous connective tissue. If these physiologic changes are equivalent in humans, they could further increase joint stresses, and cause damage to the weight bearing joints (O'Byrne et al., 1982). There is some disagreement in the literature if relaxin directly causes the relaxation of ligaments during pregnancy, although there is agreement that relaxation of ligaments does occur during pregnancy (Schauberger et al., 1996). This disagreement stems from the peak of relaxin release happening in in the first trimester, and the ligament laxity of various joints peaking at different points throughout pregnancy, such as second trimester, third trimester, two weeks postpartum, or six weeks postpartum. Thus no correlation was found between relaxin release, and ligament laxity (Schauberger et al., 1996).

Although the relaxation of the pubic symphysis joint is needed for a vaginal birth, this is clearly not the only ligament which experiences relaxation, and the relaxation of ligaments throughout the body can cause complications. Up to 28% of women experience pelvic pain during pregnancy, 50% experience back pain, and with successive births, lower back pain increases further (Mousavi et al., 2007; Borg-Stein & Dugan, 2007; Mogren & Pohjanen 2005). Lower back pain is also reported in 30% to 45% of women in the postpartum period (To & Wong, 2003). Additionally, during pregnancy 22% of women experienced knee pain, and 64% of women reported hip pain (Ponnapula & Boberg, 2010). In the postpartum period, parous women were twice as likely as the nulliparous group to have leg and foot pain (Vullo et al., 1996).

The requirement of calcium increases as the fetus grows and results in a calcium-deprived maternal state (Mull & Bill, 1934). Due to this, the body tries to compensate by increasing the metabolic bone turnover, resulting in a decreased callous bone mass during pregnancy, and throughout lactation (Akesson et al., 2004). Changes in weight bearing bone can cause changes in weight bearing cartilage. Because cartilage is much slower to adapt to changes, lasting damage could occur in the cartilage due to this adaptation during pregnancy and throughout lactation. With elevated hormonal activity during pregnancy as well as increased stress from weight gain in weight bearing joints and associated connective tissue, hip pain has been linked to osteonecrosis of the femoral head (Cheng et al., 1982). Osteonecrosis at the hip is known to possibly lead to hip osteoarthritis (Gurzu et al., 2017). It was found that limited hip flexion during pregnancy results due to pain, stiffness, and occasionally osteoporosis. The decreased hip moment which results requires an increased knee moment, which is then effected by pregnancy resultant ligament laxity. This results in joint instability and patellofemoral dysfunction, which then intensify strain on the hip and knee (Lou et al., 2001; Smith et al., 1995).

Changes in shoe design are known to have consequences in gait mechanics throughout the lower extremities. Knowing this, it seems evident that if the shape of our feet changes, our gait could change as well. Anecdotally, pregnant and parous woman complain of their feet increasing in size, hurting more than normal, and becoming flat-footed, and this change enduring into the postpartum period. According to Nyska (1997) and further supported by Ramachandra et al. (2016), some of these claims are not only anecdotal. Nyska showed during pregnancy laxity and attenuation of the tibialis posterior tendon can allow up to a 1-cm lowering of the talar head, causing a lowering of the arch and a biomechanically pronated foot during gait. The resulting midfoot pronation and lower arch creates a flattening of the foot architecture during pregnancy

(Nyska, 1997). Ramachandra et al. showed a decrease in navicular height, with the height continuing to decrease throughout pregnancy, and not returning to its original height 6 weeks postpartum. This significance was additionally maintained when the navicular height is normalized to foot length. It was also found that static pressures under the feet change significantly during pregnancy, and do not return to their original value 6 weeks postpartum (Ramachandra et al., 2017).

Changes Postpartum

Much more is known about what happens during pregnancy, than is known about the lasting effects postpartum. It is important to know what happens postpartum because for women who chose to have children, most of their life is postpartum, not pregnant. This being said, it is important to understand what occurs during pregnancy to inform what might happen after. There have been some papers published regarding lasting effects, and these provide further evidence for lasting changes postpartum.

A new study in 2018 by Chu et al. found lasting changes in joint laxity of the knee. They investigated joint laxity of 48 women, comparing first trimester joint laxity with laxity at 4 to 5 months postpartum. The researchers expected to find lasting increase in laxity and compliance at the knee due to the relaxation of the ligaments during pregnancy, but surprisingly found the opposite. They found decreases in laxity in the coronal plane of 20% to 22%, and a 51% decrease in the posterior direction for all of their participants. The experiment also aimed to find a difference between primiparous (one birth) and multiparous women (multiple births), and found there was a statistically significant increase in joint laxity at the knee of the primiparous women in the anterior direction (Chu et al., 2018). This difference could be due to lasting changes which have already taken effect in the multiparous groups, and have only just taken

place for the primiparous group over the course of the study. Compliance decreased for both groups in the posterior direction, and compliance increased in the anterior direction for the primiparous group. Although these findings were not what were hypothesized, they still support the notion of lasting changes after birth, and further support that some of these changes may compound with each birth the mother has experienced (Chu et al., 2018). The most notable limitation of this study was using the first trimester as a baseline. Although we do not understand completely what causes the ligament laxity during pregnancy, there is evidence that the hormones possibly responsible are released largely in the first trimester. This experiment is one of the first of its kind, so there is still much to be explored in further research.

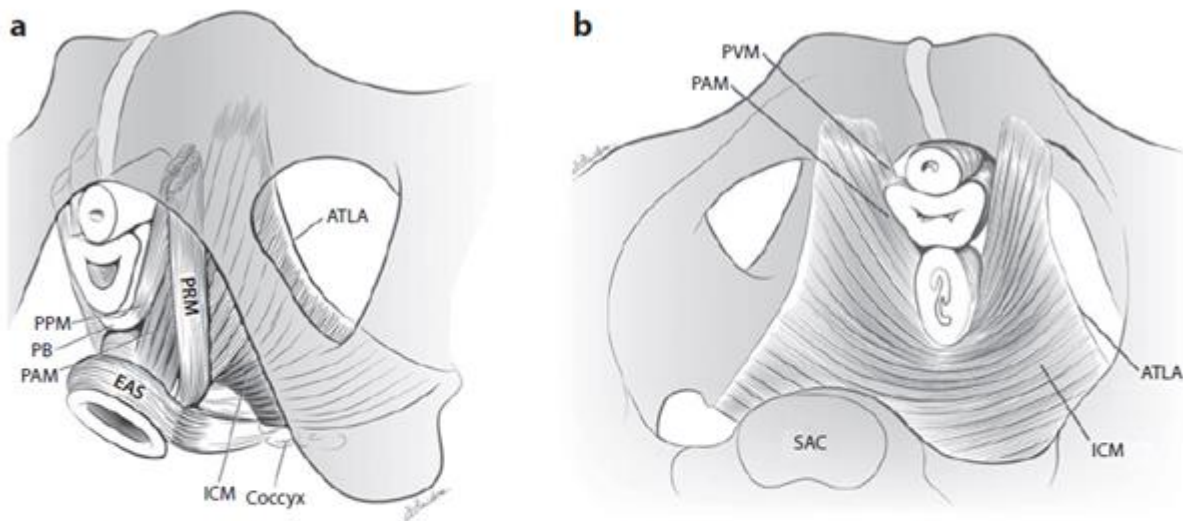


Figure 8: (a) Schematic view of the levator ani muscles from below, including the arcus tendineus levator ani (ATLA), the external anal sphincter (EAS), the puboanal muscle (PAM), the perineal body (PB) uniting the two ends of the puboperineal muscle (PPM), the iliococcygeal muscle (ICM), and the puborectal muscle (PRM). (b) The levator ani muscle seen from above, including the sacral promontory (SAC), and the pubovaginal muscle (PVM).

Adapted from Ashton-Miller & Delancey, 2009

Wise et al. (2013) found an association between parity and incident knee replacement, as well as an association between parity and incident and prevalence of radiographic OA. Wei et al. (2011) performed a cross-sectional study of 489 women between 50 and 80 years of age. The

experiment was designed to describe the associations of parity, the use of hormone replacement therapy, and oral contraceptives with cartilage volume, cartilage defects and radiographic OA in the knee. The researchers found no association with hormone replacement or oral contraceptives, but found parity was independently associated with a deficit in total knee cartilage volume. They also found that increasing the number of births was associated with decreasing the cartilage volume. This decrease in cartilage volume was in both the tibial compartment cartilage and total knee cartilage. Parity was also found to be independently associated with greater cartilage defects in the patella compartment (Wei et al., 2011).

After a first pregnancy, increased distensibility of the levator hiatus during Valsalva has been found. A schematic view of the levator ani muscles from below and above can be seen in Figure 8. This change has been shown to last, having been found up to 6 months after childbirth. Increased distensibility of the levator hiatus can lead to pelvic floor dysfunction later in life, causing pain or even prolapse. These changes are thought to be a consequence of adaptations of connective tissue properties during pregnancy and birth (Van Veelen et al., 2013). The mechanical changes experienced by the tissue involved in pregnancy are massive. The pelvic floor has been found to have a stretch ratio (final length of a structure divided by the initial length) of 3.26 by the end of the second stage of labor (Ashton-Miller & Delancey, 2009). These changes experienced in the pelvic floor, along with the changes in the lumbar region, pelvis, hip, and knee would not surprisingly have an effect on the quadriceps angle (Q angle), as was investigated in our experiment.

Access to women before they become pregnant, during, and after is limited due to the unpredictability of pregnancy. Because of this limitation, studies frequently use the first trimester data or the postpartum data as the comparative control (Bird et al., 1999; Dumas & Reid, 1997;

Franklin & Conner-Kerr, 1998; Lou et al., 2001). This is an issue due to the large release of hormones experienced during the first trimester (Schauberger et al., 1996). If a woman's body experiences lasting changes postpartum, these may not be apparent in studies using first trimester values as a baseline. Additionally, studies investigating during pregnancy changes, which use postpartum as their baseline, may be getting skewed results due to possible lasting changes due to pregnancy.

CHAPTER III

METHODS

Participants

To address the aims of this study, 28 participants (n=28: 14 nulliparous females, 14 parous females) were recruited. The inclusion criteria were as follows: 1) female; 2) between the ages of 25 – 45 years old; 3) have a body mass index less than 30 kg/m²; 4) have the ability to walk unaided for more than 20 minutes at a time; 5) Nulliparous: have not given birth to a child, nor are self-reported pregnant; and 6) Parous: have given birth to a full term infant (37 – 42 weeks) without complications between one to five years before data collection. Participants were excluded if they had a current acute injury to the lower extremities, moderate to severe low back pain, a history of significant heart problems or neurological disorders, or had undergone any lower body surgery. For parous individuals, participants were excluded if they were breastfeeding at the time of recruitment.

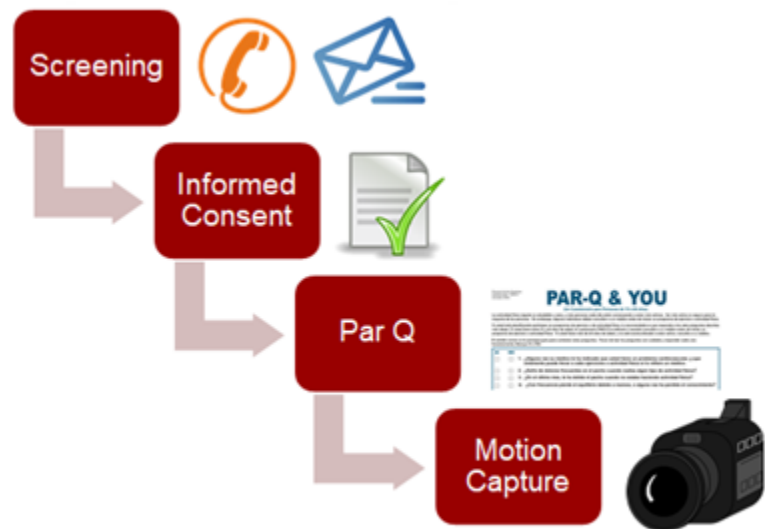


Figure 9: Schematic of experimental protocol.

Experimental Protocol

Participants completed one laboratory testing session lasting approximately 2 hours in the Biomechanics lab in the Totman building at the University of Massachusetts Amherst, with an experimental protocol as can be seen in Figure 9. Prior to attending the lab session, individuals interested in participating underwent a phone or email screening to determine eligibility. Once the participant qualified, they were asked to come to the Biomechanics lab. After arriving, the informed consent document was reviewed with the participant, and the participant was given time to ask any questions and read over the document. After they agreed to the terms, they signed the University of Massachusetts Institutional Review Board (IRB) approved informed consent form. To confirm eligibility, data was collected on their age, height, weight, and parous status. The participant (if they were in the parous group) also completed a questionnaire inquiring about their previous births and both groups completed a Par-Q questionnaire.

Motion Capture

Kinematic and kinetic data was collected for the lower body, using motion capture technology, and in-ground force plates. To use this method, participants were fitted unilaterally on the right leg with 32 retro-reflective markers which were tracked by 12 infrared motion capture cameras (Oqus 7 series, Qualysis, Inc., Gothenburg, Sweden). Marker trajectories were recorded at 200 Hz. A total of 24 markers were placed on the right lower limb, with 9 markers placed as a cluster on the thigh, and 6 placed as a cluster on the shank. The remaining 9 markers were placed at the following landmark locations: medial and lateral femoral epicondyles, medial and lateral tibial plateau, medial and lateral malleoli, fifth metatarsal head, and medial and lateral heel. Markers were then be placed on the pelvis at the left and right anterior superior iliac spine, left and right posterior superior iliac spine, left and right iliac crest, and left and right greater

trochanter (8 markers). This marker set allowed for derivation of limb motion through the point cluster technique discussed in the data processing section.

Once markers were placed, the subject was asked to stand in the data collection space to record a standing calibration trial of the markers. Participants were asked to stand with two inches between their first metatarsal heads on each foot. Preferred walking speed was determined by walking over-ground through the motion capture space at their preferred walking speed, and the timing gaits were used to collect their preferred speed. Three practice trials were completed to obtain an average preferred speed, and from this an average and a range of $\pm 5\%$ was calculated. Participants were then asked to walk over-ground through the motion capture space at that preferred walking speed. After, they were asked to walk at “the speed they would walk to catch a bus” three times through the motion capture space, and a range for their fast walking speed was calculated in the same manner as the preferred speed. Participants were then asked to walk over-ground through the motion capture space at their preferred fast (catch a bus) speed. Following this, participants were asked to walk over-ground through the motion capture space at a set speed of 1.4 m/s. During the collection, participants walked over in-ground force plates (AMTI, Watertown, MA, USA) located in the middle of the capture space, which collected GRFs at 1000 Hz. Participants completed 5 successful trials at the three speeds for a total of 15 successful trials. A successful trial meant the speed varied by no more than 5% from the respective current speed, and the foot of the right leg fully contacted the force platform embedded in the floor.

Data Processing

Kinematic and Kinetic Data

Kinematic and kinetic data collected was used calculate measures of interest for 28 participants in this study. The point cluster technique (PCT) as can be seen in Figure 10 was used

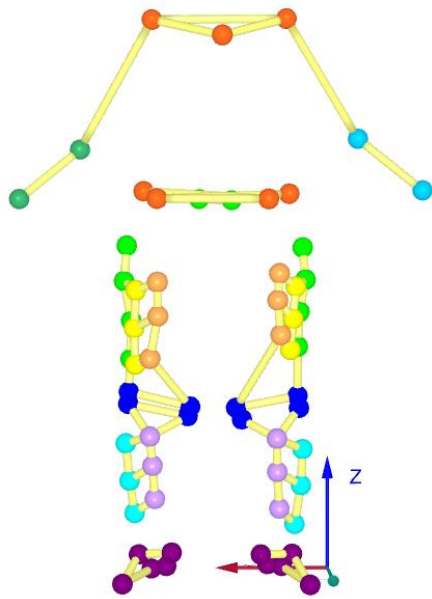


Figure 10: Lower body point cluster technique (PCT) shown on a person with simple marker set up on upper body

to calculate segment motion for each participant from the markers placed on the skin. The PCT method helps reduce the effect of the soft tissue artifact associated with the non-rigid movement of markers that are placed on the skin, allowing for reduced errors in calculating segment motion (Andriacchi et al., 1998). With the PCT marker set, the clusters of reflective markers placed on the thigh and shank estimate the movement of the underlying femur and tibia by creating and tracking a coordinate system for each cluster. The coordinate systems are determined by calculating the principal axes of the PCT marker clusters, assuming a unit weight for each marker in the cluster. The definition of principal axes allows for correction in the coordinate system due to non-rigid movement (Andriacchi et al., 1998). The accuracy of the point cluster technique has been validated using mobile biplane X-ray imaging (Gray et al., 2019).

The joint centers were calculated using cardan decomposition with a rotation sequence of XYZ. The center of the knee joint is calculated as the midpoint between the medial and lateral femoral epicondyle markers. The ankle joint center is calculated as the midpoint between the medial and lateral malleoli markers. The center of the hip joint is calculated using the Bell et al.

(1989; 1990) regression equations. For the right hip joint center, the medial-lateral position is calculated by $0.36 * ASIS_Distance$. The Anterior-posterior position of the right hip joint center is calculated by $-0.19 * ASIS_Distance + (0.5 * RPV_Depth - Target_Radius_ASIS)$. The axial distance of the right hip joint center is calculated by $-0.3 * ASIS_Distance$. Where the $ASIS_Distance$ is the 3D distance between the Right and Left ASIS (anterior superior iliac spine) markers, the RPV_Depth is the 3D distance between the Mid-Point of the ASIS and the Mid-Point of the PSIS (posterior superior iliac spine), and the $Target_Radius_ASIS$ is the radius of the marker placed on the ASIS landmark. Visual 3D along with custom Matlab code was used to process PCT kinematic and kinetic data to calculate 3D lower limb joint angles and moments for each trial of each participant. Kinematic and kinetic data was filtered using a low pass Butterworth filter with cutoff point of 8 and 15 Hz, respectively. Joint moments and angles were normalized to height and weight of the participant, and interpolated to 101 data points, representing the length of the gait cycle (0 to 100%). Discrete time points for heel strike and toe off of the right leg were determined when vertical GRFs exceeds 20N for heel strike and is less than 20N for toe off.

Quadriceps Angle

To quantify static alignment and calculate quadriceps angle (Q angle) using retroreflective markers, the method proposed by Mündermann et al. (2008) was used to align the subject to the coordinates of the laboratory, and the joint centers were calculated as is outlined above. As seen in Figure

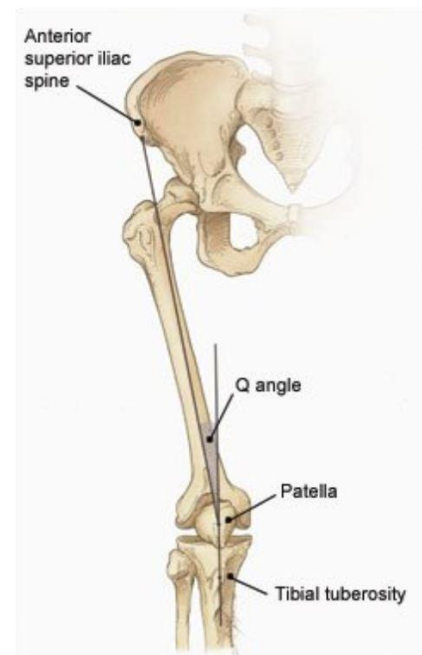


Figure 11: Depiction of Q-angle
Adapted from The Corps Pilates Blog
2009, Retrieved from
nilatesonfifth.wordpress.com

11, the Q angle is the angle between the vector from the center of the patella to the Anterior Superior Iliac Spine (ASIS), and the vector from the center of the tibial tuberosity to the center of the patella. As the motion capture system captures the center of the markers, the radius of these markers must be taken into account of the calculation of the correction factors and joint centers. The Q angle for our purposes was calculated as the angle between the vector connecting the knee joint center (approximating the center of the patella) to the ASIS, and another vector connecting the knee joint center to the ankle joint center (approximating the vector between the tibial tuberosity and the patella). This calculation is completed on the subject's standing calibration trial. To reduce variation due to the orientation of the standing trial, the patient's position was first aligned to the laboratory coordinate system. The angle (γ) between the line drawn from the heel marker to the 5th metatarsal head (v_{foot}) and the anterior posterior axis (y) was calculated using the following equation:

$$\gamma = \frac{180}{\pi} * \arccos\left(\frac{v_{foot} \cdot y}{|v_{foot}| * |y|}\right)$$

Following this calculation, the limb is computationally rotated by this angle around the z axis. The Q angle (δ) was then calculated using the vector connecting the knee joint center with the ASIS center projected into the zx plane ($v_{Q,zx}$), and the vector connecting the ankle joint center and knee joint center projected into the zx plane ($v_{tibia,zx}$). This was calculated using the following equation:

$$\delta = \frac{180}{\pi} * \arccos\left(\frac{v_{Q,zx} \cdot v_{tibia,zx}}{|v_{Q,zx}| * |v_{tibia,zx}|}\right)$$

Outcomes and Statistics

The statistical methods used for both Aims 1, 2, and 3 were the same. All statistical tests used an alpha criterion level of ($\alpha = 0.1$). A two way ANOVA was used to calculate if there were differences across speeds and parity for each variable, then Fisher's Least Significant Difference (LSD) post hoc test was used to calculate p values for within speed results. The ages, heights, weights, speeds, and Q angle were tested for group differences using two sample t-tests. The 90% confidence intervals and Cohen's d effects sizes were calculated for all variables. The primary measures of interest for Aim 1 include peak ankle eversion and peak hip flexion over stance, knee flexion local maxima during the first half of stance, mean internal rotation angle of the knee across stance, and range of motion of the internal rotation angle across stance. The primary measures of interest for Aim 2 include peak knee adduction moment, flexion moment, and internal rotation moment during walking. For Aim 3, the primary measure of interest is Q angle.

Power calculations were performed for primary outcome variables, using data from the literature. In Aims 1, 2, and 3 there are many variables of interest and many different potential analyses. We explored sample size and power in the context of assessing pooled t-test main effects ($\alpha = 0.05$). Assuming a sample size of $n = 15$ for each group, we assessed the power to detect relevant differences between the 2 study groups using mean and standard deviations for young adults from the literature. Most of the variables had a power greater than 0.8 for a group size of 15. Table 1 shows the power for key variables for the % difference at which the minimum power was first > 0.75 .

Table 1: Power calculations for main effects of primary outcome variables. For each variable, the highest available SD (from the various age/gender groupings) was used for the calculations. KAM = knee adduction moment; KIEA = knee internal/external angle; AEA= ankle eversion angle; HIRA = hip internal rotation angle. Additional gait variables were calculated, but not shown.

Variable	Units	Data Source	Mean, YA	SD	Abs. Δ	% Δ	Power
Q angle	°	(Weiss et al., 2013)	13	2.6	4	31	0.55
Mean KIEA	°	(Boyer et al., 2012)	2.67	1.26	1.34	50	0.82
First peak KAM	(Nm/kg)	(Chumanov et al., 2008)	7.7	1.9	2	26	0.82
Peak AEA	°	(Chumanov et al., 2008)	3.9	2.1	2.2	56	0.82
Peak HIRA	°	(Chumanov et al., 2008)	3.1	4.3	4.3	140	0.78

CHAPTER IV

RESULTS

Participant Characteristics

Data was collected on 28 participants, including 14 parous women and 14 nulliparous women. All 28 participants were included in each part of the analysis. The heights and weights of the two groups were not found to be significantly different; however the ages of the groups were significantly different (Table 2, $p = 0.003$). The average age of the parous women at first pregnancy was 30.1 ± 3.5 years. The average number of children of the parous women was 1.7 ± 0.6 children. Pregnancies for all parous participants reached full term. There were no participants with multiple child births (e.g. twins). For participants with multiple children, the average time between births was 3.6 ± 1.8 years. There were no reported pelvic floor injuries due to birth. Only one subject reported recovery complications and postpartum physical therapy following complications. She reported that the issue resolved after therapy. The average weight difference from before their first pregnancy to the date of collection was 3.3 ± 4.3 kg. The average length of time since their most recent birth was 3.2 ± 1.3 years. The average weight of their most recent child at birth was 3.4 ± 0.6 kg. The average weight gained during their most recent pregnancy was 15.2 ± 5.9 kg. All of the parous women included in the study breastfed, and the average duration was 1 ± 0.6 years. Out of the 14 parous women, 12 of them had natural births for their most recent birth (two had C-sections). Q angle was not found to be significantly different between groups (Table 2, $p = 0.44$). Each of the three speeds was also not found to be significantly different between groups (Table 3).

Table 2: Means, standard deviations, and p values of participant characteristic groups. The variables with p values smaller than 0.1 are highlighted in grey. CI 90% stands for confidence interval of 90%, and ES stands for effect size.

	Parous	SD	CI 90%	Nulliparous	SD	CI 90%	p value	ES
Age (y)	36	4.1	34.2 – 37.80	30.1	5.2	27.81 – 32.39	0.003	1.26
Height (m)	1.66	0.06	1.63 – 1.69	1.65	0.04	1.63 – 1.67	0.77	0.2
Weight (kg)	63.93	10.32	56.39 – 68.47	61.01	8.35	57.34 – 64.68	0.43	0.31
Q angle (°)	3.94	2.74	2.74 – 5.14	3.25	1.79	2.46 – 4.04	0.44	0.3
Step Width (m)	0.17	0.03	0.15 – 0.18	0.16	0.03	0.14 – 0.17	0.32	0.38
Step Height (m)	0.69	0.06	0.66 – 0.71	0.71	0.09	0.67 – 0.75	0.44	-0.29

Table 3: Averages and p values of three speeds for each group. Speeds were not found to be significantly different between groups. Speeds are reported in m/s. CI 90% stands for confidence interval of 90%, and ES stands for effect size.

	Parous	SD	CI 90%	Nulliparous	SD	CI 90%	p value	ES
Preferred Speed	1.41	0.17	1.34 – 1.48	1.47	0.16	1.40 – 1.54	0.41	-0.36
Fast Speed	1.94	0.14	1.88 – 2.00	1.91	0.21	1.82 – 2.00	0.58	0.17
Set Speed	1.41	0.03	1.40 – 1.42	1.41	0.03	1.40 – 1.42	0.87	0

Kinematic Variables

The mean, standard deviation, and p values of the kinematic variables calculated are reported in Table 4. There was not a main effect of group for the peak ankle eversion angle ($p = 0.59$), heel strike knee flexion angle ($p = 0.92$), or first peak knee flexion angle ($p = 0.54$). For the knee flexion angle at toe off, a group effect was found with the toe-off knee flexion angle larger for the parous group compared to the nulliparous group ($p = 0.060$, Figure 12). There was not a main effect of group for the mean knee internal/external angle ($p = 0.75$), range of motion

of the knee internal rotation angle ($p = 0.82$), peak hip flexion angle ($p = 0.15$), peak hip adduction angle ($p = 0.74$), or toe off hip adduction angle ($p = 0.11$). The interaction effect between the different speeds and the two groups was found not to be significant for any kinematic variables, indicating the differences between groups do not depend on the speed at which the participants were tested.

Table 4: Means, standard deviations, and p values of kinematic variables separated by group. The variables with p values smaller than 0.1 are highlighted in grey. Values are reported in units of degrees. Main effect of group is reported and if found significant, specifics are reported for that variable. Main effect of group is calculated across all speeds. CI 90% stands for confidence interval of 90%, and ES stands for effect size.

Variable	Parous	SD	CI 90%	Nulliparous	SD	CI 90%	p value	ES
Peak Ankle Eversion Angle	9.04	4.07	7.25 – 10.83	9.44	2.62	8.29 – 10.59	0.59	-0.12
Heel Strike Knee Flexion Angle	4.11	3.9	2.40 – 5.82	4.21	5.24	1.91 – 6.51	0.92	-0.02
First Peak Knee Flexion Angle	20.56	5.14	18.29 – 22.82	21.3	6.02	18.66 – 23.95	0.54	-0.13
Toe Off Knee Flexion Angle	8.86	6.87	5.85 – 11.88	6.06	6.31	3.29 – 8.84	0.060	0.42
<i>Preferred</i>	9.55	7.52	6.24 – 12.86	6.68	6.91	3.64 – 9.72	0.26	0.4
<i>Fast</i>	7.65	6.02	5.00 – 10.29	5.38	5.82	2.82 – 7.94	0.37	0.38
<i>Set</i>	9.4	7.31	6.18 – 12.61	6.14	6.57	3.25 – 9.02	0.2	0.47
Mean Knee Internal/External Angle	3.95	3.13	2.58 – 5.33	3.72	3.52	2.17 – 5.26	0.75	0.07
ROM of Knee Internal Rot Angle	17.05	5.92	14.45 – 19.66	17.3	3.8	15.63 – 18.97	0.82	-0.05
Peak Hip Flexion Angle	31.5	7.01	28.42 – 34.58	33.78	7.38	30.54 – 37.02	0.15	-0.32
Peak Hip Adduction Angle	-14	3.13	-15.38 – -12.62	-13.49	3.8	-15.16 – -11.82	0.5	-0.15
Toe off Hip Adduction Angle	-0.89	2.48	-1.98 – 0.20	-0.21	2.96	-1.51 – 1.09	0.27	-0.25

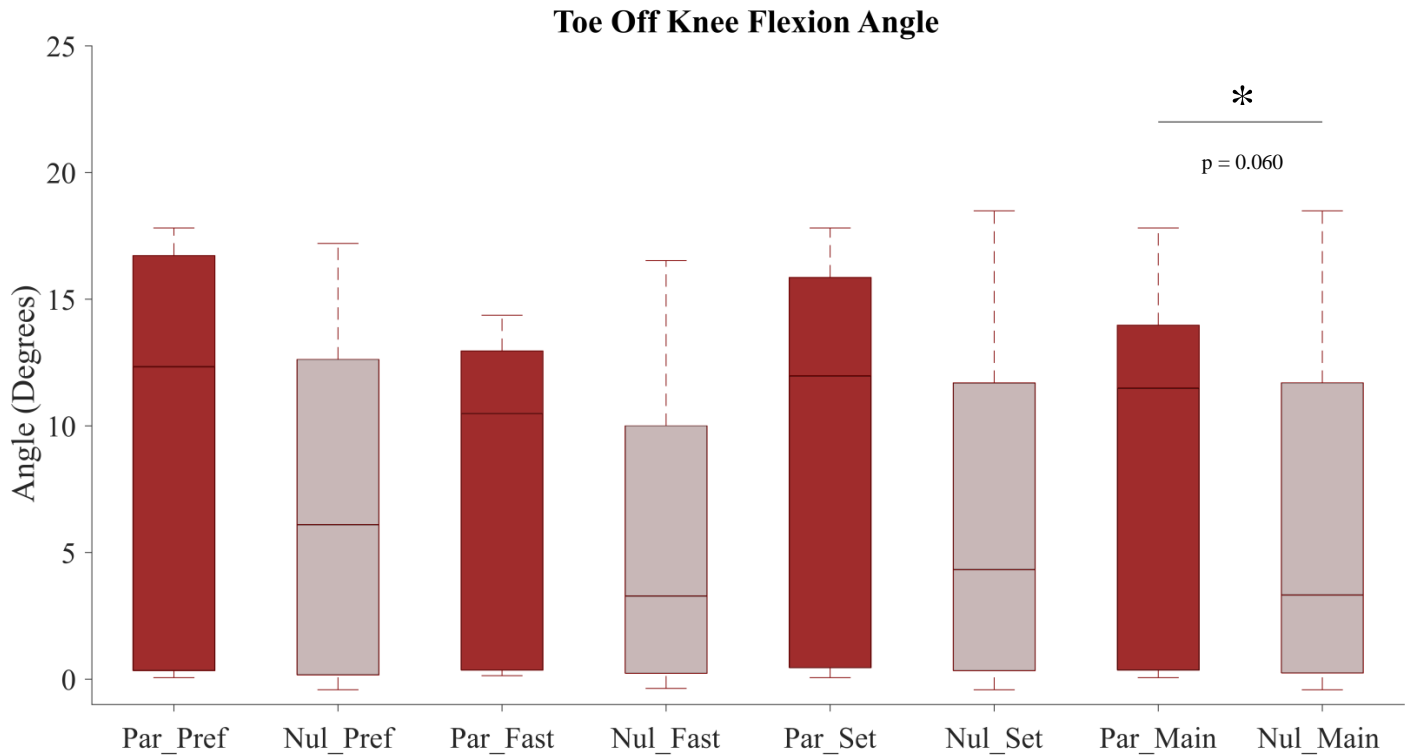


Figure 12: Box plot for knee flexion angle at toe off. The y axis is in units of degrees, and the x axis represents, from left to right respectively, parous preferred speed; nulliparous preferred speed; parous fast preferred speed; nulliparous preferred speed; parous set speed; nulliparous set speed; parous main effect; nulliparous main effect. . Main effect of group is calculated across all speeds. Maroon represents the parous group and grey represents the nulliparous group. The box represents the interquartile range (25th to 75th percentile), while the horizontal line in the box represents the median. The whiskers above show the upper quartile + 1.5*interquartile range, while the lower whiskers show the lower quartile – 1.5*interquartile range. Outliers are represented by empty circles. The horizontal line with an asterisk above it identifies any pairs which were found to be significantly different, and the

Kinetic Variables

The mean, standard deviation, and p values of the kinetic variables calculated are reported in Table 5. There was a significant effect of group for the knee extension moment at heel strike ($p = 0.0006$, Figure 13) and post-hoc testing indicated that at all speeds the moment was smaller for the parous group compared to the nulliparous group (preferred: $p = 0.036$; fast preferred: $p = 0.084$; set: $p = 0.039$). There was a significant effect of group for the first peak knee flexion moment ($p = 0.040$, Figure 14), where the moment was smaller for the parous group compared to the nulliparous group. There was not a main effect of group for the peak knee

adduction moment ($p = 0.28$) or peak knee internal rotation moment ($p = 0.98$). There was a significant group effect for the peak hip adduction moment ($p = 0.003$, Figure 15) and post-hoc testing indicated that at preferred and set speeds, the moment was smaller for the parous group compared to the nulliparous group (preferred: $p = 0.087$; set: $p = 0.057$). The interaction effect between the different speeds and the two groups was found not to be significant for any kinetic variables, indicating the differences between groups do not depend on the speed at which the participants were tested.

Table 5: Means, standard deviations, and p values of the main effect of kinetic variables separated by group. The variables with p values smaller than 0.1 are highlighted in grey. Where significant main effects of group were found, the means, standard deviation and p-values for within speed post-hoc testing are reported. Values are reported in units of %body weight*height. Main effect of group is reported and if found significant, specifics are reported for that variable. Main effect of group is calculated across all speeds. CI 90% stands for confidence interval of 90%, and ES stands for effect size.

Variable	Parous	SD	CI 90%	Nulliparous	SD	CI 90%	p value	ES
Heel Strike Knee Extension Moment	-0.71	0.27	-0.82 – -0.59	-0.97	0.4	-1.15 – -0.80	0.0006	0.8
<i>Preferred</i>	-0.7	0.25	-0.81 – -0.59	-0.98	0.4	-1.16 – -0.81	0.036	0.84
<i>Fast</i>	-0.75	0.28	-0.88 – -0.63	-0.99	0.39	-1.16 – -0.81	0.082	0.68
<i>Set</i>	-0.66	0.28	-0.78 – -0.54	-0.96	0.43	-1.15 – -0.77	0.025	0.82
First Peak Knee Flexion Moment	4.97	2.13	4.03 – 5.90	6.17	3.29	4.72 – 7.61	0.04	-0.43
<i>Preferred</i>	4.28	1.8	3.49 – 5.08	5.26	2.46	4.18 – 6.34	0.33	-0.45
<i>Fast</i>	6.25	2.44	5.17 – 7.32	7.77	3.97	6.02 – 6.34	0.13	-0.46
<i>Set</i>	4.37	1.58	3.67 – 5.07	5.47	2.83	4.22 – 6.71	0.27	-0.48
Toe Off Knee Flexion Moment	4.46	5.82	-3.82 – -1.63	5.49	6.39	-3.31 – -0.84	0.45	-0.24
Peak Knee Internal Rot Moment	1.25	0.3	1.12 – 1.38	1.25	0.36	1.10 – 1.14	0.98	-0.01
First Peak Knee Adduction Moment	-2.72	2.5	1.90 – 7.02	-2.07	2.82	2.68 – 8.30	0.28	-0.17
Peak Hip Adduction Moment	-7.63	1.2	-8.16 – -7.10	-9.02	2.56	-10.14 – -7.89	0.003	0.69
<i>Preferred</i>	-7.66	1	-8.35 – -6.89	-8.81	2.2	-10.60 – -7.86	0.14	0.64
<i>Fast</i>	-7.62	1.66	-8.00 – -7.21	-9.23	3.11	-10.10 – -7.92	0.041	0.75
<i>Set</i>	-7.61	0.9	-8.16 – -7.10	-9.01	2.47	-10.14 – -7.89	0.074	0.69
Peak Hip Internal Rotation Moment	2.68	0.72	2.37 – 3.00	2.85	0.76	2.51 – 3.18	0.28	-0.22

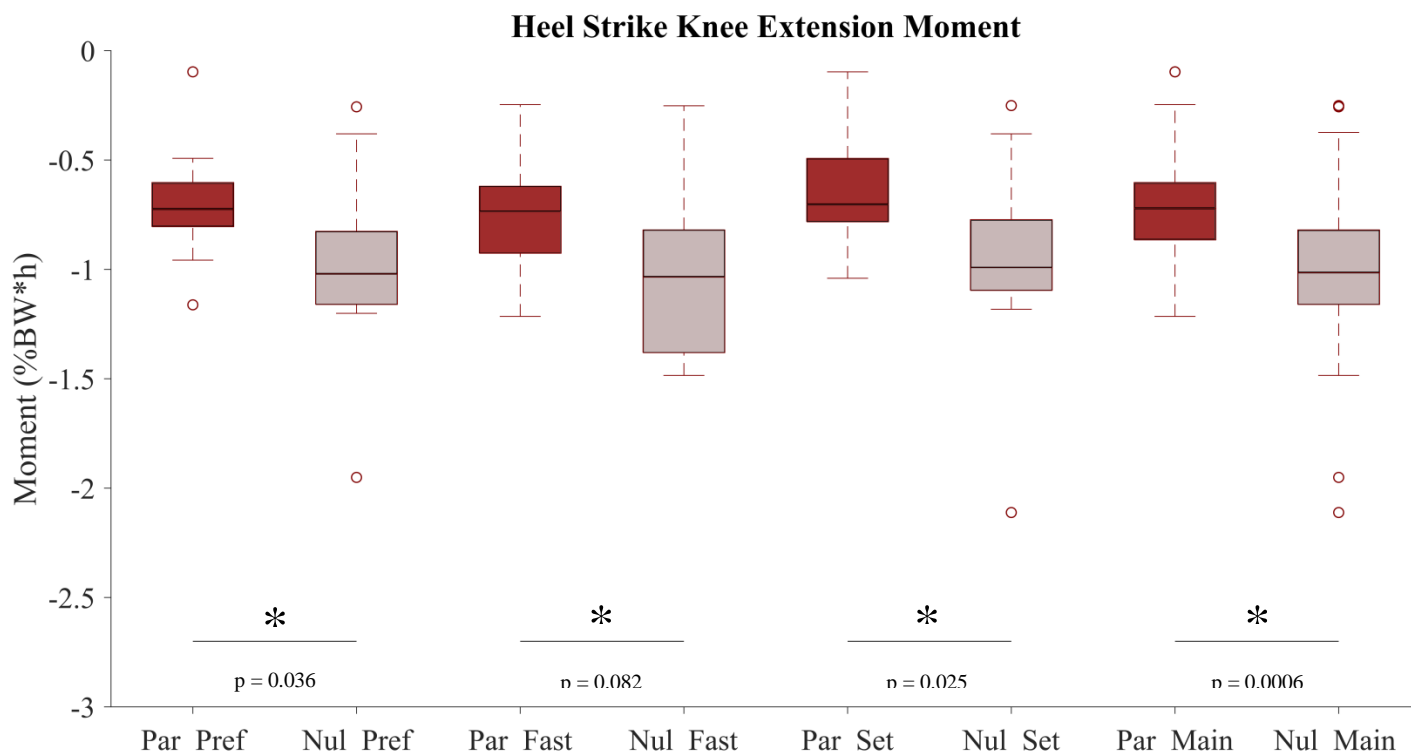


Figure 13: Box plot for knee extension moment at heel strike. The y axis is in units of %body weight*height, and the x axis represents, from left to right respectively, parous preferred speed; nulliparous preferred speed; parous fast preferred speed; nulliparous preferred speed; parous set speed; nulliparous set speed; parous main effect; nulliparous main effect. Main effect of group is calculated across all speeds. Maroon represents the parous group and grey represents the nulliparous group. The box represents the interquartile range (25th to 75th percentile), while the horizontal line in the box represents the median. The whiskers above show the upper quartile + 1.5*interquartile range, while the lower whiskers show the lower quartile – 1.5*interquartile range. Outliers are represented by empty circles. The horizontal line with an asterisk above it identifies any pairs which were found to be significantly different, and the p value for that pair is included below the line.

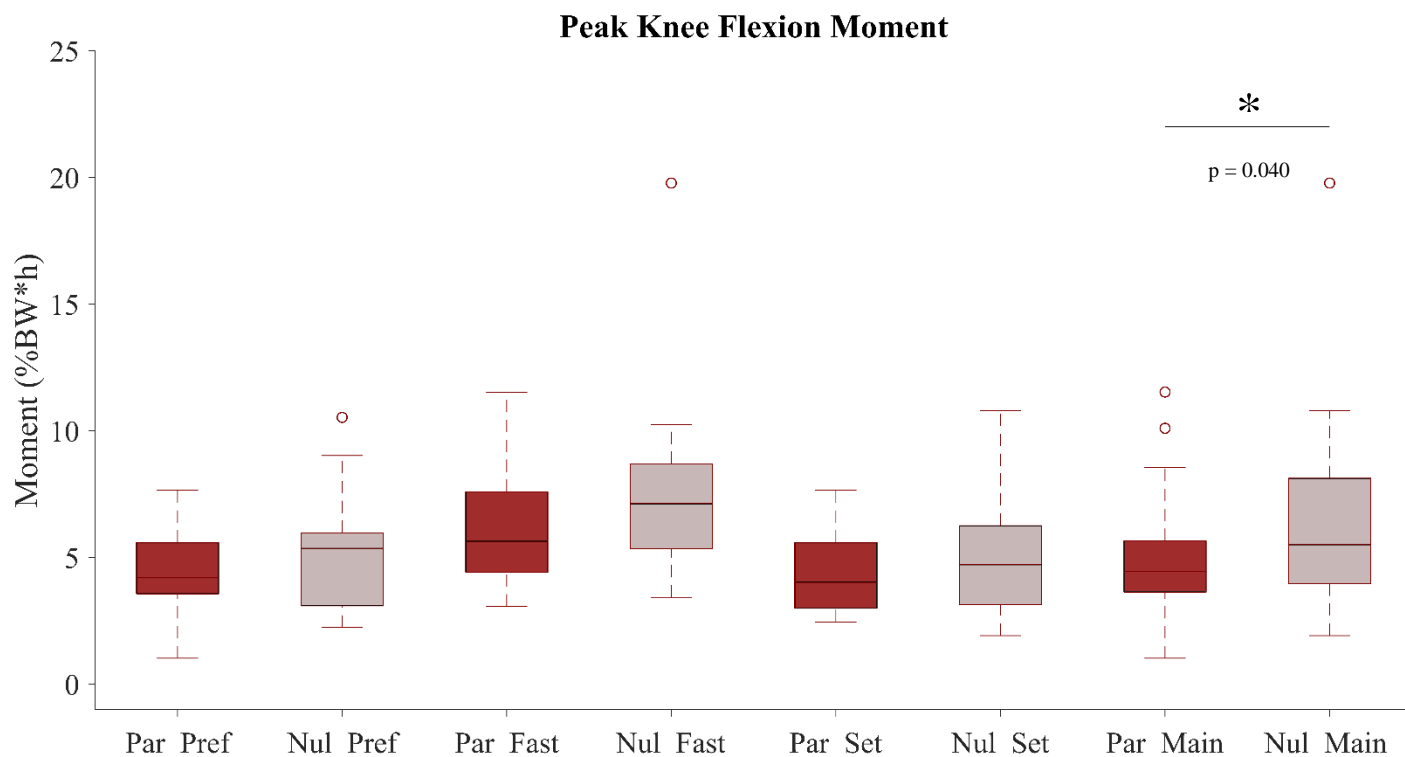


Figure 14: Box plot for peak knee flexion moment. The y axis is in units of %body weight*height, and the x axis represents, from left to right respectively, parous preferred speed; nulliparous preferred speed; parous fast preferred speed; nulliparous preferred speed; parous set speed; nulliparous set speed; parous main effect; nulliparous main effect. Main effect of group is calculated across all speeds. Maroon represents the parous group and grey represents the nulliparous group. The box represents the interquartile range (25th to 75th percentile), while the horizontal line in the box represents the median. The whiskers above show the upper quartile + 1.5*interquartile range, while the lower whiskers show the lower quartile – 1.5*interquartile range. Outliers are represented by empty circles. The horizontal line with an asterisk above it identifies any pairs which were found to be significantly different, and the p value for that pair is included below the line.

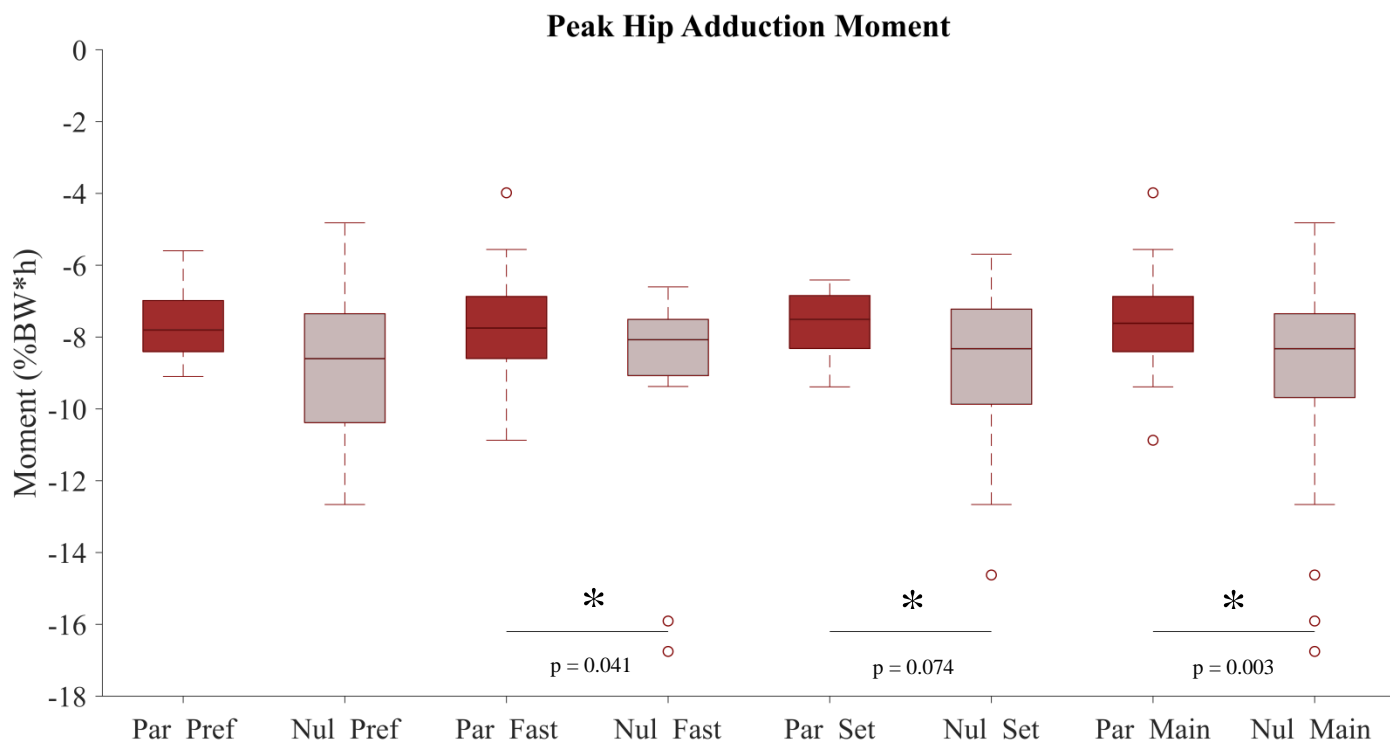


Figure 15: Box plot for peak hip adduction moment. The y axis is in units of %body weight*height, and the x axis represents, from left to right respectively, parous preferred speed; nulliparous preferred speed; parous fast preferred speed; nulliparous preferred speed; parous set speed; nulliparous set speed; parous main effect; nulliparous main effect. Main effect of group is calculated across all speeds. Maroon represents the parous group and grey represents the nulliparous group. The box represents the interquartile range (25th to 75th percentile), while the horizontal line in the box represents the median. The whiskers above show the upper quartile + 1.5*interquartile range, while the lower whiskers show the lower quartile – 1.5*interquartile range. Outliers are represented by empty circles. The horizontal line with an asterisk above it identifies any pairs which were found to be significantly different, and the p value for that pair is included below the line.

CHAPTER V

DISCUSSION

This study investigated the differences in mechanics of walking between parous women and nulliparous women. The change in mechanics of gait and morphology during pregnancy is fairly well documented (Alvarez et al., 1988; Borg-Stein et al., 2007; Bird et al., 1999; Butler et al., 2006; Calguneri et al., 1982; Chu et al., 2018; Dumas et al., 1997; Franklin et al., 1998; Lou et al., 2001; Marnach et al., 2003; Ponnappula et al., 2010; Ramachandra et al., 2017; Smith et al., 1995; van Veelen et al., 2014; Yoo et al., 2015), but the lasting changes postpartum are not. We hypothesized there would be measurable differences in the kinetics and kinematics between the parous and nulliparous groups. This hypothesis was based on previous studies which showed women experience large changes in morphology as well as changes in gait mechanics during pregnancy (Alvarez et al., 1988; Branco et al., 2014; Branco et al., 2015; Butler et al., 2006; McCrory et al., 2014; Ponnappula et al., 2010; Schauburger et al., 1995; Van Veelen et al., 2013; Wei et al., 2011). Additionally, research showing multiplanar ligament laxity in the knee persists after pregnancy, as well as lasting changes in compliance at the knee persisting longer than four months, led to the formulation of the study hypotheses (Chu et al., 2018). Our kinematic hypotheses expected parous women would have increased peak ankle eversion over stance, peak hip flexion over stance, first peak knee flexion, a reduction of the mean knee internal rotation angle across stance phase, and a reduction in the range of motion of the internal rotation angle. Our kinetic hypothesis expected parous women would have increased peak knee adduction moment, flexion moment, and internal rotation moment over stance. Additionally, we expected

parous women to have abnormally large Q angles. None of our specific hypotheses were supported, however we did have some interesting findings none the less.

Through our study, we aimed to explore the potential for differences in gait postpartum to better understand the impact of parity on the musculoskeletal system. The impact of parity on kinematics was smaller than was expected. A group effect was found only for toe-off knee flexion angle, with the angle being larger for the parous group compared to the nulliparous group ($p = 0.060$, Figure 12). The impact of parity on kinetics was greater than the impact on kinematics. Parous and nulliparous groups differed significantly for knee extension moment at heel strike, first peak knee flexion moment, and peak hip adduction moment. In all cases moments were smaller for the parous compared to the nulliparous group. From these results, we know the effect on kinematics is small; however, it seems the effect on kinetics is not. This suggests there is an effect of parity, which still has the potential to influence OA risk, however not through the current theoretical pathway to OA initiation due to a change in contact location.

Our hypotheses were primarily based on the theory of initiation of OA that suggests changes in tibio-femoral cartilage contact locations would cause the initiation of OA in the knee (Andriacchi & Münderman, 2006). We did not find many changes in kinematics, so it's unlikely there are any changes in cartilage contact locations in response to parity. The changes we did find, however, suggest a different possible pathway to OA initiation via unloading of the cartilage (Carter et al., 2004). Although the contact locations may not have changed, it is possible that the loading cartilage is experiencing has changed, possibly due to changes in the upper body movement or changes in the ground reaction forces. This alteration in load the cartilage experiences may lead to pathological changes in cartilage similar to what may occur in response to a change in cartilage contact locations.

Our results for peak ankle eversion angle, mean knee internal/external rotation angle, range of motion of knee internal/external rotation angle, hip adduction angle, hip flexion angle, heel strike knee flexion angle, toe off hip adduction angle, toe off knee flexion angle, and knee flexion angle were comparable values to previous walking studies when compared to other young healthy adult populations (Boyer et al., 2012; Khalid et al., 2017). Our first two hypotheses stated that parous women as compared to nulliparous women would have increased peak ankle eversion and peak hip flexion over stance, and increased knee flexion local maxima during the first half of the stance phase (H.1.1) as well as a reduction of the mean internal rotation angle of the knee and a reduction in the range of motion of the internal rotation angle across stance phase (H.1.2). We did not find evidence of significant differences between the groups for these outcomes. It is possible the parous groups didn't experience the expected changes or that the changes weren't large enough to result in a significant kinematic difference. Although prior work has found lasting changes in ligament properties postpartum which could lead to kinematic changes, our results suggest that parous women are able to compensate for these changes (if present) to limit the impact on joint kinematics. If changes in tissue mechanics occur, there are multiple strategies possible to adapt to perform the same task. It is possible they have compensated for changes in ligament laxity with increased muscle activation to maintain the same kinematics as they experienced before pregnancy, however as this was not covered by this study, future research should examine this.

Only one of the kinematic variables tested was found to be significantly different between groups, knee flexion angle at toe off, suggesting a difference in push-off mechanics. The group effect found for knee flexion angle at toe off was larger for the parous group compared to the nulliparous group ($p = 0.060$, Figure 12). This supports our overall hypothesis

that there would be a kinematic difference between parous and nulliparous groups; however, because it was the only difference found, it seems the change in kinematics is not very large. It is also possible that although there were not many differences in kinematics, there were differences in muscle activation, although because this was not included in the breath of this study, this is speculation and must be investigated further. Future studies should investigate whether there are activation differences in the primary muscles used in gait between parous and nulliparous groups. Additionally, it is feasible we only found one significant difference for the kinematic variables tested due to our small sample size, which may have affected our ability to identify small changes between groups. Some of the variables we tested had larger standard deviations than those used in our power analysis. This would indicate a larger sample size may have been needed to find significant differences for those variables. Although there was a significant difference in ages between groups, this is not expected to have affected our results because women in this age range are not going through any large age related hormonal or physical changes.

It is also possible the changes in ligament laxity did not occur as expected or were not long lasting enough to cause differences in our study. The role of relaxin and other hormones during pregnancy which change ligament laxity are still not fully understood, let alone postpartum. Although much is unknown about the constantly changing hormonal cocktail released throughout pregnancy, studies have shown the ligaments are more relaxed during pregnancy (Dumas et al., 1997). Due to the aforementioned changes, weight gain or lack of weight gain during pregnancy could also affect outcomes of lasting changes due to the extended mechanical loading experienced by relaxed ligaments during pregnancy (Ashton-Miller & Delancey, 2009; Schauburger et al., 1995).

Our third specific hypothesis, H 2.1, that parous women compared to nulliparous women will have increased peak knee adduction moment, flexion moment, and internal rotation moment during walking were not supported. Our results for first peak knee adduction moment, peak knee flexion moment, heel strike knee extension moment, and toe off knee flexion moment were comparable values to previous walking studies when compared to other young healthy adult populations (Fischer et al., 2018; Hafer, 2017). Increased peak knee adduction moment and internal rotation moment during walking were not significantly different between groups. However, our hypothesis that parous women would have an increased peak knee flexion moment as compared to nulliparous women was shown to the contrary. We found the peak knee flexion moment was smaller for parous women than nulliparous women during walking ($p = 0.040$, Figure 14).

Previous research has shown that decreasing joint loading can be bad for long-term health of cartilage (Carter et al., 2004), and mechanical loading during everyday activities is a stimulus for healthy cartilage remodeling in non-OA knees (Andriacchi et al., 2004; Felson, 2013). Our participants were healthy, so the decrease in moments could be lowering the cyclic loading, which is critical to the maintenance of healthy cartilage (Carter et al., 2004). The decrease in moments the parous women are experiencing could lead to thinning and softening of their cartilage, as well as cartilage degradation if normal loading is not reinstated (O'Connor, 1997). Previous research has shown a reduction of the peak knee flexion moment during gait after ACL reconstruction (Andriacchi et al., 2005). It is readily accepted in the literature that there is a higher incidence of OA later in life for individuals who experience an ACL tear or reconstruction. Further research has shown patients who have undergone an ACL reconstruction who display a lower peak knee flexion moment have greater morphological changes in the

medial tibial cartilage (Scanlan et al., 2007). However, the reduction in peak knee flexion moments for the parous group was not to the extent that is experienced by the ACL reconstructed groups, where to be considered in the low loading group, the peak knee flexion had to be less than $2.8\%bw*ht$ (Scanlan et al., 2007). Previous work has also shown both the peak knee flexion moment and peak hip adduction moment are lower for those with OA than for an asymptomatic group, which is consistent with our findings in this study (Asthephen et al., 2007). Together these results suggest that the parous women may be at a greater risk for knee OA initiation due to the kinetic changes following pregnancy.

Our finding that knee extension moment at heel strike was decreased for the parous women ($p = 0.0006$, Figure 13) aligns with previous OA research showing knee extension moment at heel strike was smaller for groups with OA than that of a young asymptomatic group, however this difference may be due to age differences rather than differences due to OA (Favre et al., 2014). It is possible, that similar to an ACL injury, pregnancy causes hormone-induced ligament changes, and the pregnant women experience internally changed stresses or adaptations of the muscles to strive for the same kinematics as before pregnancy. As previously stated, a decrease in joint loading as experienced in this study could be bad for long term cartilage health, which has been hypothesized for ACL reconstructed patients as well (Carter et al., 2004).

Although no changes in kinematics during the weight acceptance peaks (peaks during the first part of stance phase) were identified in our study (Table 4), further research needs to be completed to find what is causing a change in peak knee and hip moments during stance. The decrease found in peak knee flexion and hip adduction moments for parous women ($p = 0.040$, Figure 14; $p = 0.003$, Figure 15) possibly suggests changes in the upper body movement or changes in the ground reaction forces between groups. Changes in the ground reaction force

vector position relative to the joint center can alter the kinetics experienced in the hip, knee, or ankle without necessarily altering the kinematics. Body center of mass has been shown to change in the lateral and anterior directions for up to 28 weeks postpartum (Catena et al., 2019). Therefore, changes in the center of mass position due to lasting impacts of pregnancy may change the knee joint kinetics by altering the GRF vector position relative to the joint center. Further research should investigate this possibility.

Q angle was not found to be different between groups (Table 2). This was a surprising finding because our expectation that Q angle would be larger for the parous group was supported by research which found lasting increased distensibility of the levator hiatus 6 months after birth. Increased distensibility of the levator hiatus means the muscles are more able to stretch under loading and can lead to pelvic floor dysfunction (Van Veelen et al., 2013). Training of the pelvic floor muscles has been shown to lead to changes in gait (Fraser et al., 2014). Changes of the levator hiatus postpartum are thought to be a consequence of adaptations of connective tissue properties during pregnancy and birth (Van Veelen et al., 2013). Widening of the pelvis happens naturally during birth and can possibly result in increased width of sacroiliac joint or increased pubic symphysis width (Garagiola et al., 1989). The material changes experienced by the tissue involved in pregnancy are massive. These changes experienced in the pelvic floor, and with the changes in the ligaments and other connective tissues of the lumbar region, pelvis, hip, and knee would not surprisingly have an effect on the Q angle of parous women.

There is no standard way to calculate Q angle, so it is hard to compare values across studies; however the means calculated in this experiment were lower than other comparable studies reporting average Q angles for women (Hahn et al., 1997; Sanchez et al., 2014; Wu et al., 2019). The participants were instructed to stand with two inches between their first metatarsals

on each foot to reduce error due variation in unspecified quiet standing, however there is error introduced by the participant's estimation of distance. For future research, indicators should be permanently on the floor showing participants where to place their feet to reduce this error. There is also a possibility that there are groups within the parous group that have larger Q angles due to birth, and those who did not experience these changes for various possible reasons. Our standard deviation was very large, more than another half time bigger than that of the nulliparous group. This large standard deviation could suggest there are two groups, responders and non-responders to pelvis changes from birth. This proposed split could be related to weight gain during pregnancy. When we split out data between women who had above the median weight gain during pregnancy and women who had below or equal to the median weight gain during pregnancy, the average Q angle is higher for the above median group. This was not found to be statistically significantly different, but this may be due to the parous group being divided further into smaller groups for this calculation, and therefore we lacked the power needed to show this difference (Appendix, Table 6). As was previously mentioned, weight gain has the potential to influence lasting changes due to extended mechanical loading on relaxed ligaments, and therefore further investigation is warranted.

Summary

This study investigated the resulting impact of pregnancy on gait mechanics in a healthy population within 5 years of giving birth. Although this was an exploratory study, it has shown the potential for a connection between parity and specific joint mechanics, which may have implication for OA risk. However, much more research is needed to see if parity is a risk factor for OA, and further, what can be done to lower this risk. It is clear that altered gait mechanics have an impact on OA initiation; however, it is unclear what measures directly drive this change

in progression. The changes that occur due to childbirth could be one driver of changes in gait mechanics. Our data revealed a decrease in the moments experienced, which could possibly lead to degradation of cartilage due to under loading of the joint. We think this may be an indication that pregnancy could increase the risk of OA, and therefore more research into this possibility is warranted. Outcomes from this project provide some insight into the effects of pregnancy on women's gait, and could possibly lead the field toward whether or not pregnancy is a risk factor of OA.

APPENDIX

WAVEFORM DATA

Table 6: Q angles split with regards to weight gained during pregnancy. This difference was not found to be significantly different between groups.

	Average	SD	p value	CI 90%	ES
Greater than 13.6 kg	4.8	3.4	0.32	3.31 - 6.29	0.56
Less than or equal to 13.6	3.3	1.7		2.55 - 4.05	

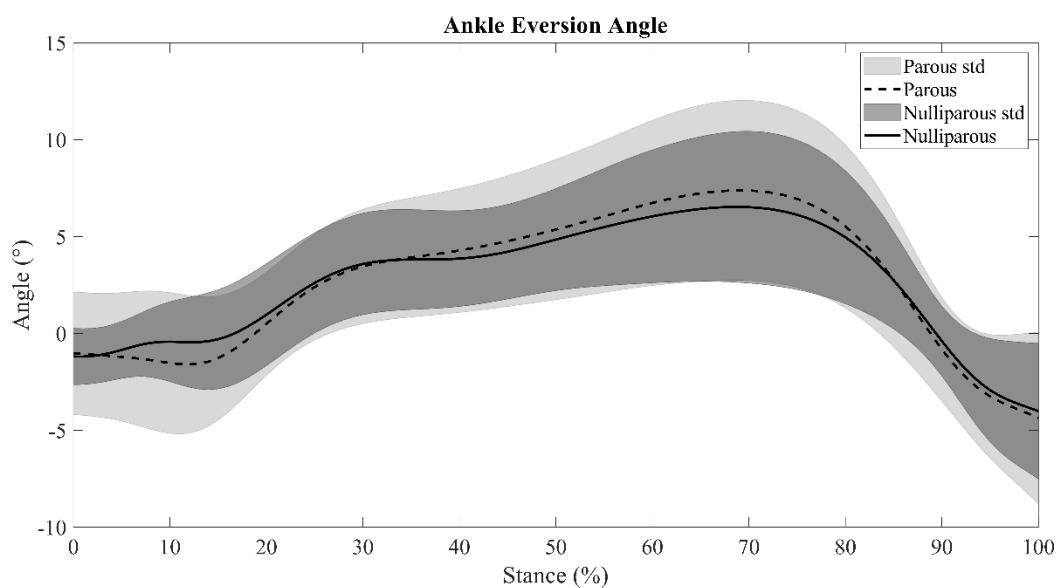


Figure 16: Waveform for main effect of ankle eversion angle across stance phase.

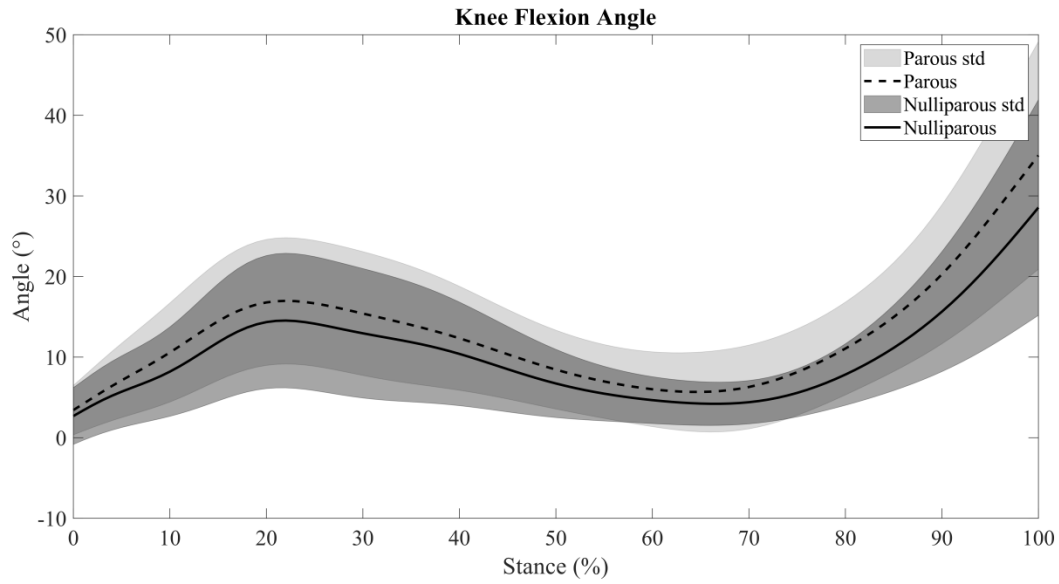


Figure 17: Waveform for main effect of knee flexion angle across stance phase.

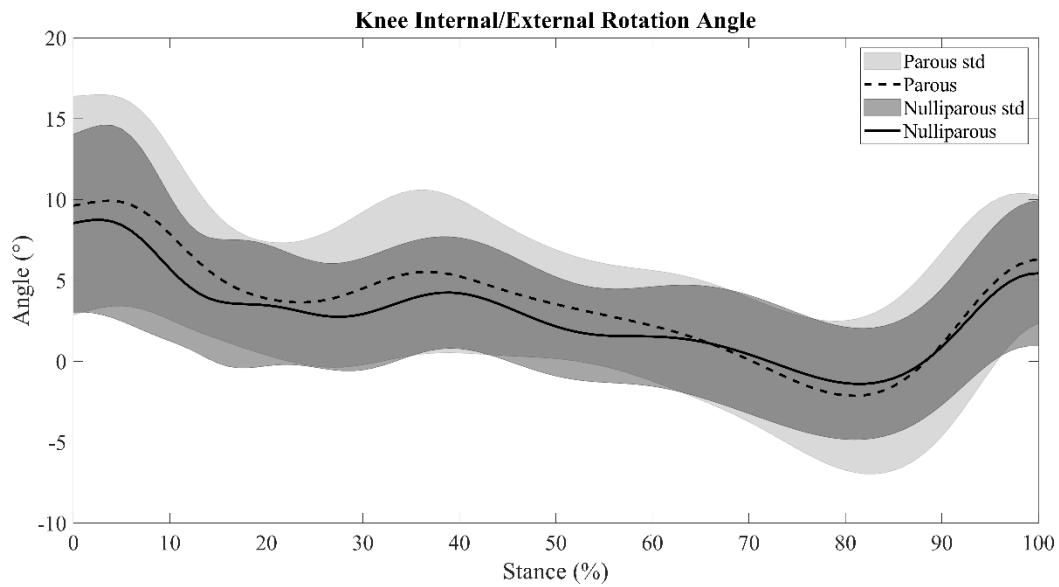


Figure 18: Waveform for main effect of knee internal/external angle across stance phase.

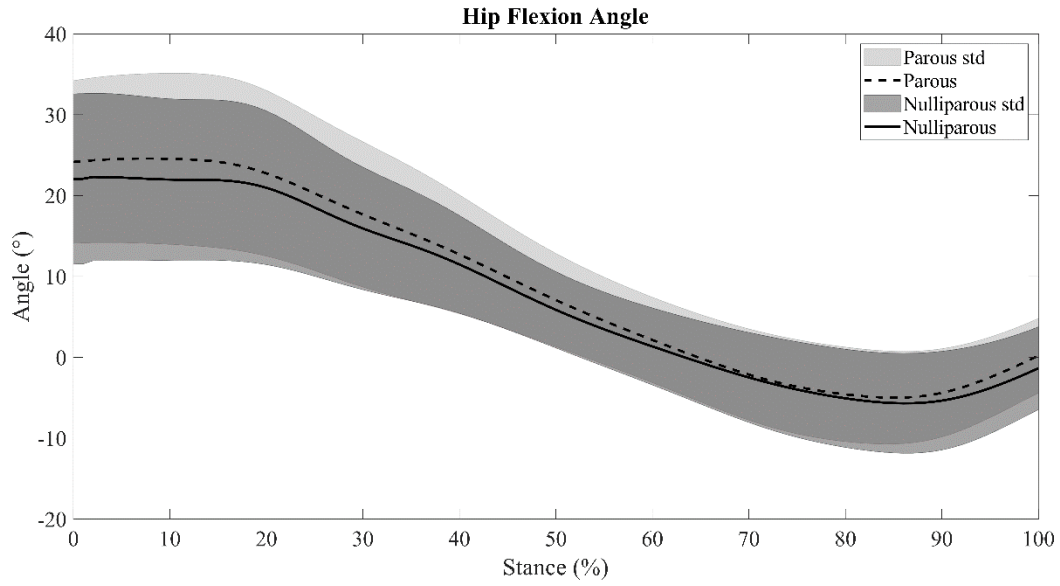


Figure 19: Waveform for main effect of hip flexion angle across stance phase.

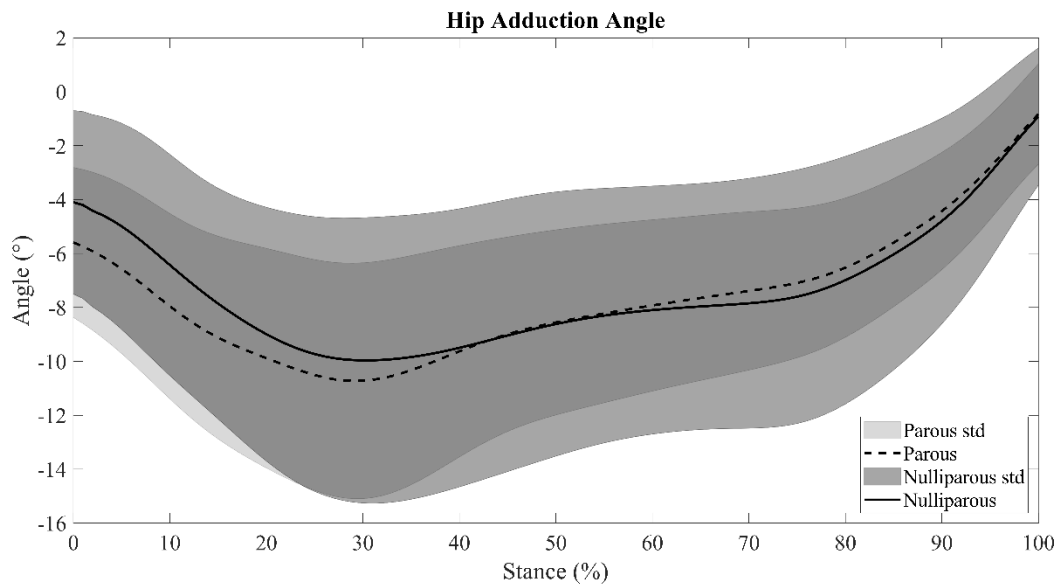


Figure 20: Waveform for main effect of hip adduction angle across stance phase.

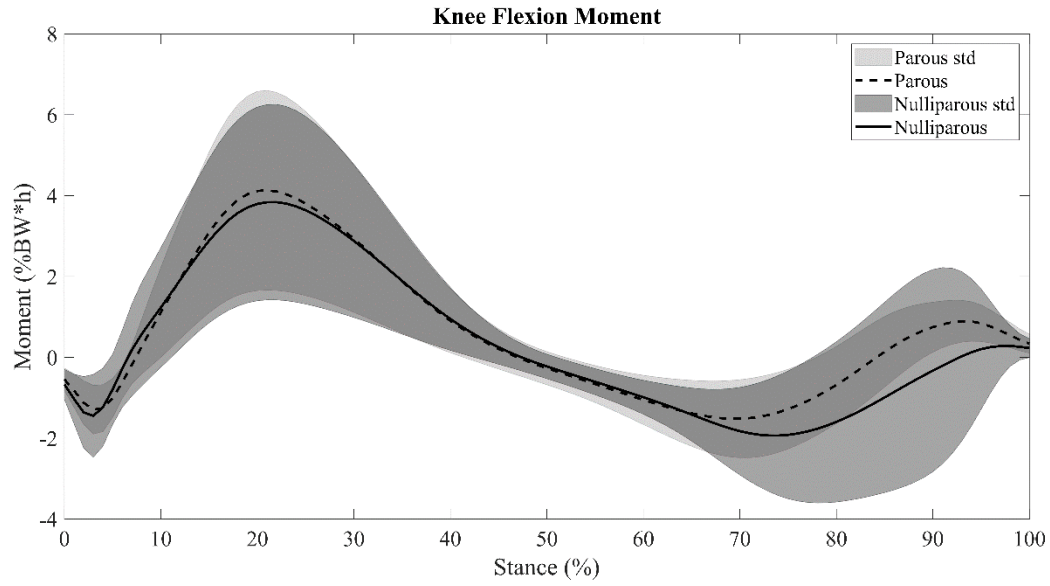


Figure 21: Waveform for main effect of knee flexion moment across stance phase.

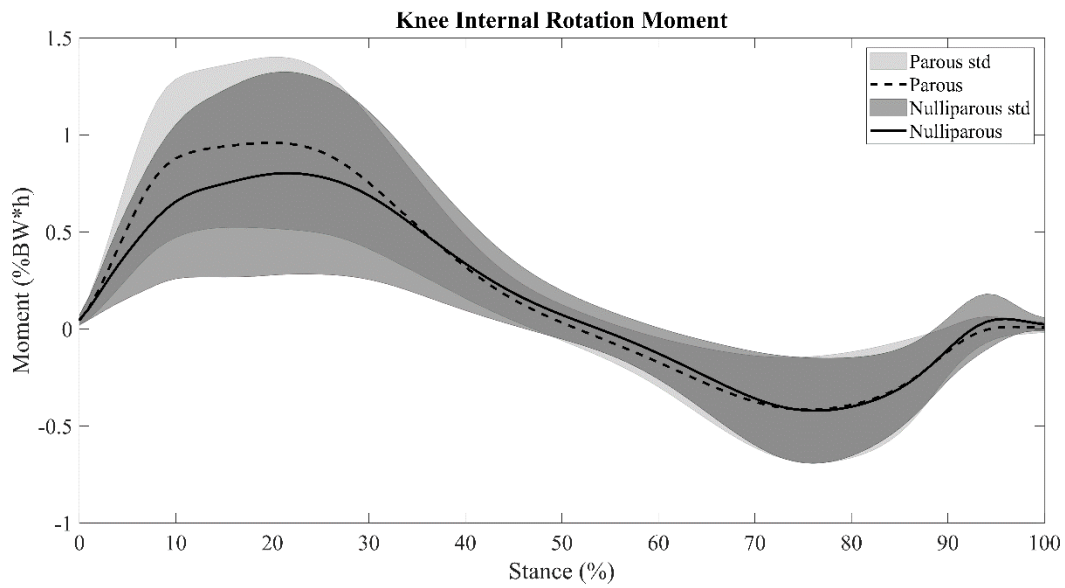


Figure 22: Waveform for main effect of knee internal rotation moment across stance phase.

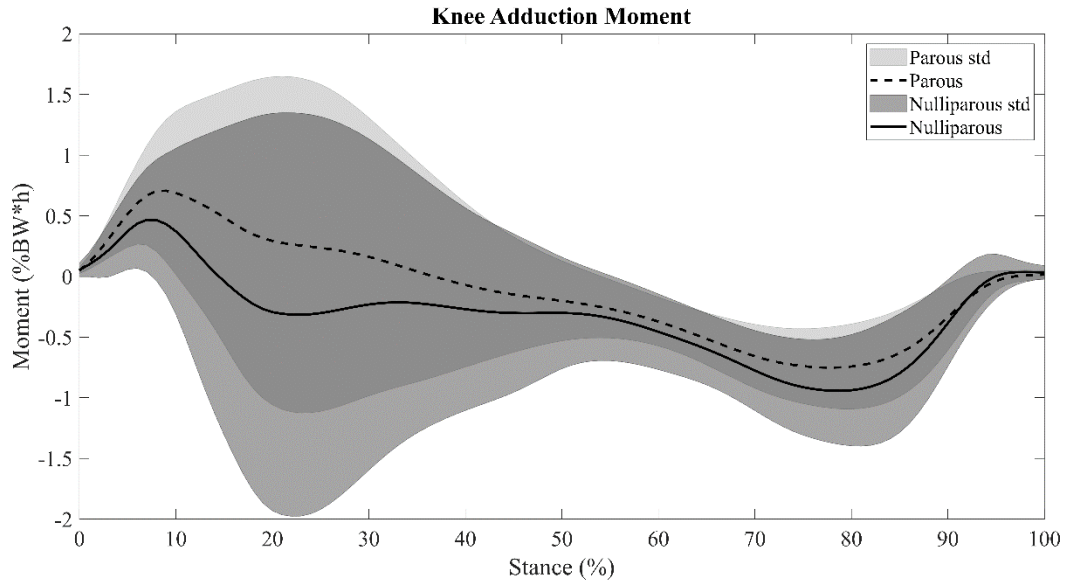


Figure 23: Waveform for main effect of knee adduction moment across stance phase.

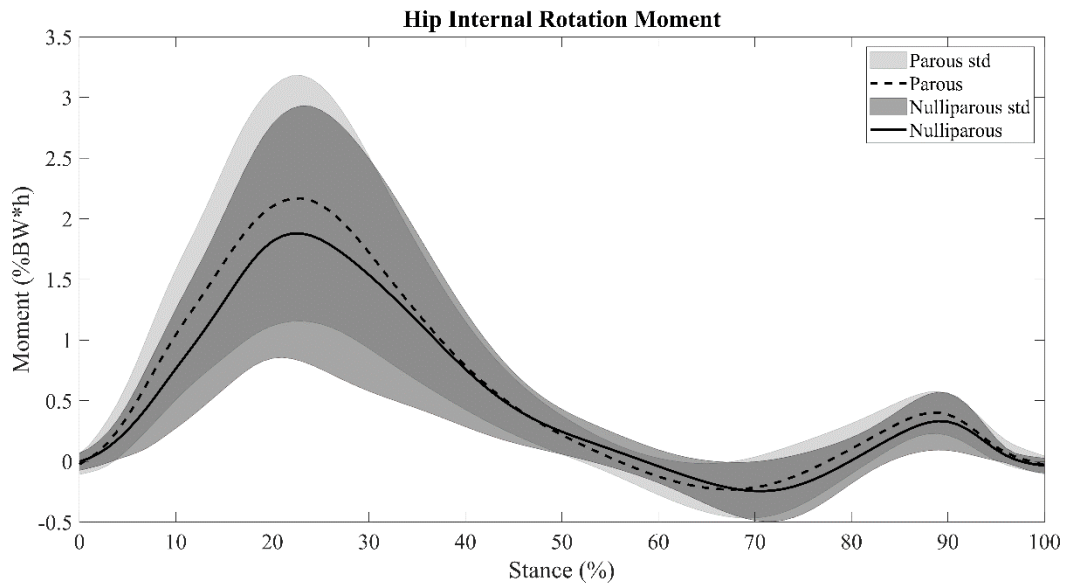


Figure 24: Waveform for main effect of hip internal rotation moment across stance phase.

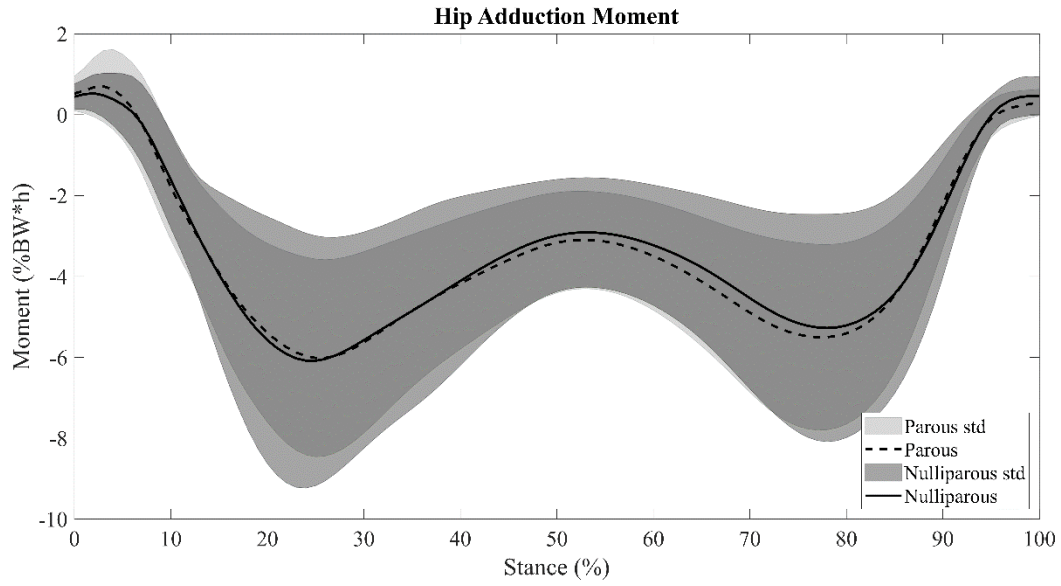


Figure 25: Waveform for main effect of hip adduction moment across stance phase.

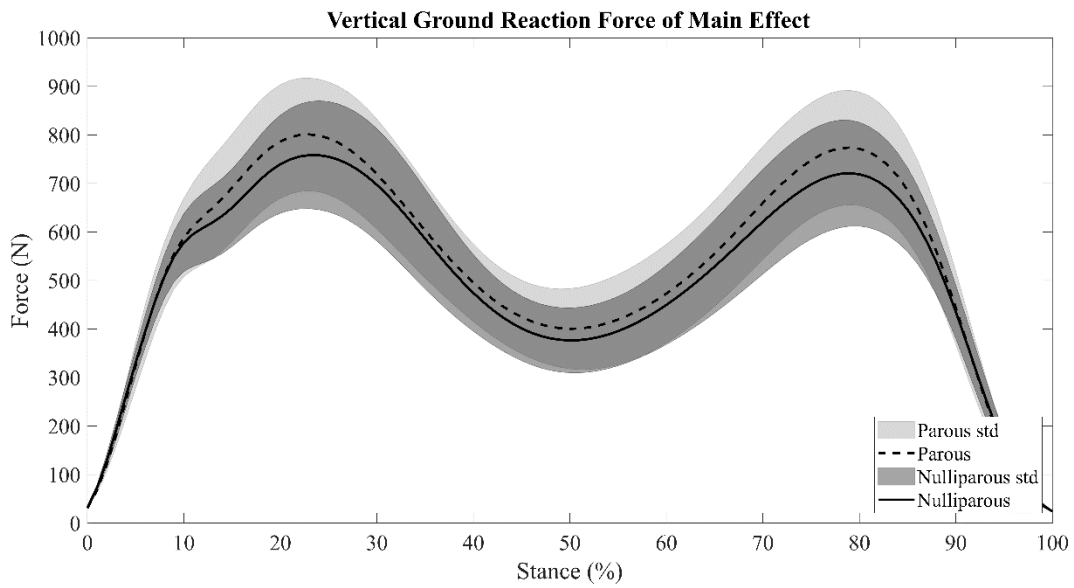


Figure 26: Waveform for main effect of vertical ground reaction forces across stance phase.

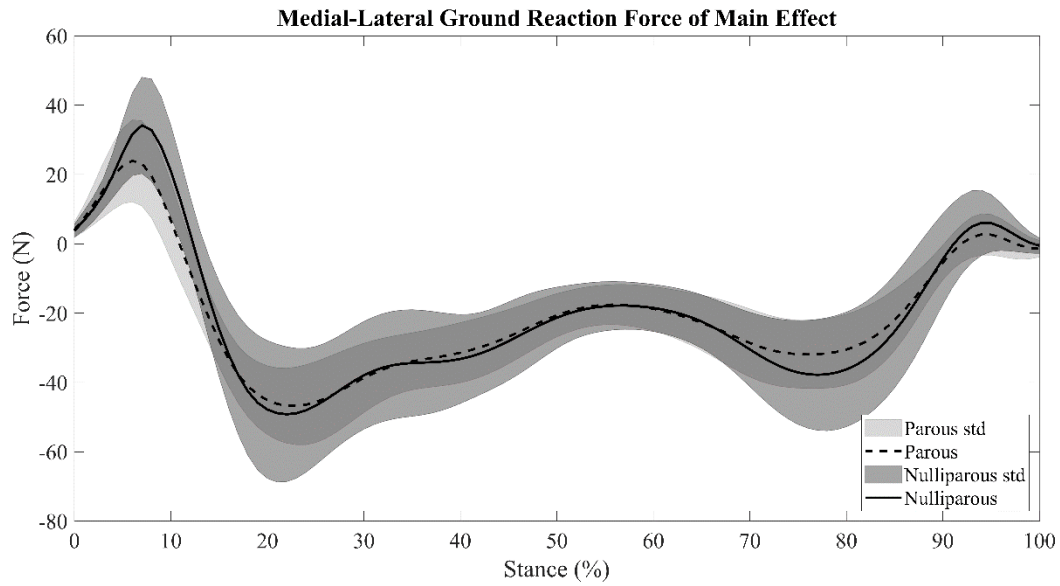


Figure 27: Waveform for main effect of medial-lateral ground reaction forces across stance phase. Positive is lateral, negative is medial.

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