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The Influence of Trunk Inclination on Lower Limb Moments and Muscle Activation Patterns

Wael Ahmed Alghamdi

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Wael Ahmed Alghamdi

School of Health Sciences

University of Salford, Manchester, UK

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List of presentations arising from this thesis

International Conference Posters:

1. Osteoarthritis Research Society International (OARSI 2018), Liverpool UK.

How do lower limb joint moments and muscle activations change when forward trunk lean increases? (Appendix IV)

2. Orthopaedic Research Society (ORS 2019), Austin, USA.

The effect of inter-subject variations in trunk inclination on lower limb moment and muscle activation during walking. (Appendix VIII)

3. Osteoarthritis Research Society International (OARSI 2019), Toronto, Canada.

Could increased trunk flexion underlie alterations in knee muscle activity in people with knee AO? (Appendix IIX)

Abstract

Knee osteoarthritis (OA) is a progressive disease resulting in degeneration of the articular cartilage. Previous studies have shown that people with knee OA walk with altered gait biomechanics, characterised by altered joint moments, increased knee muscle activity and increased muscular co-contraction. Modelling studies suggest that such altered muscle patterns may increase the loading on knee joint, with imaging studies suggesting that co-contraction may accelerate cartilage loss. Interestingly, there is now emerging evidence that people with knee OA walk with an increased flexion of the trunk. It is possible that this alteration in trunk position may underlie some of the previously observed biomechanical differences between people with knee OA and healthy control subjects.

The overall aim of this thesis is to develop new insight into whether alterations in muscle coordination, characteristic of people with knee OA, could result from an increased flexion of the trunk during walking. Five studies were conducted. The first study was designed, similar to previous research, to understand the key biomechanical differences between people with knee OA and healthy controls during walking. This study confirmed that people with knee OA walk with an increased sagittal plane inclination of the trunk and altered muscle patterns, including increased co-contraction. The second study aimed to understand the biomechanical effect of instruction young healthy people to walk with increased trunk flexion and specifically investigated lower limb kinetic patterns and the activation of the quadriceps, hamstrings and gastrocnemius along with muscle co-contraction. This third study also aimed to understand the biomechanical effects of increasing trunk flexion but looked at healthy older people and patients with knee OA. Both the second and third studies utilised a biofeedback approach and demonstrated clear changes in joint moments and increases in hamstring muscle activity and muscle co-contraction as trunk flexion was increased. Building on these two studies, the fourth study was designed to explore whether healthy people who habitually walk with an increased trunk inclination also demonstrate increased muscle activation. Again the data showed a clear association between increased trunk flexion and elevated muscle patterns, supporting the idea that some of the biomechanical characteristics associated with knee OA, may be the result of alterations in trunk inclination during walking and not due to pain and/or knee instability.

The final study was designed to explore a possible biomechanical mechanism which may underlie increased trunk flexion, both in healthy individuals and also in people with knee OA. Specifically, this study investigated the link between hip flexor length and trunk flexion, showing a moderate positive correlation between forward trunk inclination in walking and hip flexor muscle length. Overall, the findings of this Ph.D. demonstrated that patients with knee OA walk with approximately 3^{o} more in sagittal plane inclination of the trunk and exhibit higher hamstrings muscle activations and co-contraction compared to matched healthy controls. The data from studies 2-4 support the idea that idea that these increases in muscle activation may be driven by increases in trunk flexion and the data from study 5 suggests that this increased trunk lean may result from a shortening of the hip flexor muscles. Together these data motivate future study into new interventions for knee OA which could target increased muscle activity by stretching the hip flexor muscles and improving postural control.

Chapter One

Introduction

1.1 Knee osteoarthritis

Osteoarthritis (OA) is defined as the gradual degeneration of the cartilage between the joints. This degeneration can lead to alteration of joint tissue, causing pain, stiffness and disability. OA is a very common medical condition and the third most common musculoskeletal disorder across the world after low back pain and neck pain (Murray et al., 2012). Previous studies have reported that OA has a multifactorial aetiology, including changes in the dynamic processes and the metabolic activity in the joint areas. These changes include the articular cartilage, bone, muscles, synovial membrane and menisci (Alnahdi, Zeni, & Snyder-Mackler, 2012; Felson et al., 2000). Thus, OA characterises a heterogeneous and complex group of disorders that lead to joint degeneration. OA might affect any joints of the human body, for instant knees, hands and hips.

OA most commonly affects the knee (Guccione et al., 1994; Zhang & Jordan, 2010). This condition, known as knee OA, is characterised by pain, stiffness and function limitation (Dixon, Hinman, Creaby, Kemp, & Crossley, 2010; Peat, McCarney, & Croft, 2001). Importantly, pain is the most common cause of disability in patients with knee OA, affecting patients' activity and mood negatively and disturbing their sleep. Clinically, knee OA is characterised by joint pain worsening during activity, tenderness, muscle weakness, and imitated range of motion (Hurley, Scott, Rees, & Newham, 1997; Peat et al., 2001). In addition, objective radiographic characteristics are also used to identify the presence and severity of knee OA. These characteristics include loss of joint space, osteophyte formation, cyst formation and subchondral sclerosis (Peter et al., 2011).

1.2 Incidence and cost of knee OA

According to World Health Organisation (WHO), 10% and 18% of men and women respectively aged 60 and over have symptomatic OA. In the United Kingdom more than eight million people suffer with OA. In 2020, the number of people with knee OA is predicted to jump from four million to five and half million and to six million by 2035. Moreover, more

than four million people in England have OA of the knee and the findings show that women are more affected than men ("Arthritis Research UK," 2016). In addition, Peat et al. (2001) found that 25% of people aged 55 years old suffer from knee pain and one of six people with knee pain visit their doctor.

Numerous studies have attempted to provide insight into the economic cost of OA and have identified direct costs, such as surgery and conservative treatment. Other costs, called indirect costs, are those such as loss of both productivity and working time because of pain and disability (Chen, Gupte, Akhtar, Smith, & Cobb, 2012; Leardini et al., 2004; Ruiz et al., 2013). In the UK, the hospital costs alone for joint replacements were estimated to be approximately £850 million in 2010 (Chen et al., 2012). In the USA, around $14 billion was spent on total knee replacements (Kim, 2008). Moreover, some countries spend 1 % of their gross national product on arthritis care (Hunter, Schofield, & Callander, 2014). Arthritis Research UK has reported that 2.36 million working age people in the UK have required treatment for knee OA; this puts these people at danger of losing their job ("Arthritis Research UK," 2016). These statistics demonstrate that knee OA is a major health burden and is associated with an enormous financial cost. It is therefore imperative that we develop a thorough understanding of the disease, including the different risk factors.

1.3 Biomechanical cause of knee OA

Knee OA is a degenerative disease with multiple risk factors that affect the occurrence and progression of the disease. Previous studies have reported the risk factors for knee OA and have identified systemic risk factors, such as age, race, female gender and hormonal factors. Although these are considered as non-modifiable factors, other factors such as knee alignment, joint injury and heavy physical activity are considered as modifiable risk factors (Heidari, 2011; Silverwood et al., 2015b). Knee OA is a mechanical disease and therefore affected by the amount of knee joint loading (Brandt, Dieppe, & Radin, 2008). It is believed that abnormal increases in knee joint loading will lead to increased demands on the articular cartilage (Litwic, Edwards, Dennison, & Cooper, 2013) which could ultimately impact cartilage health. Therefore, the biomechanical factors (modifiable risk factors) may have a crucial influence characteristics articular cartilage (Cooper et al., 2000; Felson et al., 2000) and could determine the incidence and progression of knee OA.

Knee moments and muscle co-contraction may be used as surrogate measures of knee joint loading. Previous research showed that people with knee OA demonstrate changes in joint moments during walking (Baliunas et al., 2002) and other activities (Childs, Sparto, Fitzgerald, Bizzini, & Irrgang, 2004). For example, studies have found that the knee adduction moment (KAM) is elevated in individuals with knee OA compared to healthy subjects (Baliunas et al., 2002; Mundermann, Dyrby, Hurwitz, Sharma, & Andriacchi, 2004a) and it has been indicated that an increase in KAM may lead to an increase in knee joint loads across the medial compartment (Hinman, Hunt, Simic, & Bennell, 2013). Other researchers have shown that the knee extensor moment may be decreased in patients with knee OA (Astephen, Deluzio, Caldwell, & Dunbar, 2008; Kaufman, Hughes, Morrey, Morrey, & An, 2001) compared to healthy subjects. Taken together, these studies demonstrate that OA patients may walk with alterations in knee moments. It is possible these alterations may affect knee joint stresses, negatively affecting cartilage.

Co-contraction is the simultaneous activity of both the agonist and antagonist muscles around the joint. A number of researchers have reported that people with knee OA walk with greater co-contraction between the quadriceps and hamstring (Childs et al., 2004; Hodges et al., 2016a; Hortobagyi et al., 2005; Zeni, Rudolph, & Higginson, 2010a) and between the quadriceps and gastrocnemius (Childs et al., 2004; Schmitt & Rudolph, 2008; Sritharan et al., 2016) when compared to control healthy subjects. In addition, it has been suggested that increased co-contraction will increase joint loading and lead to long-term joint degeneration (Andriacchi, 1994; Brandon, Miller, Thelen, & Deluzio, 2014; Sritharan et al., 2016). Furthermore, recent evidence has demonstrated that increased co-contraction is associated with the progression of knee OA (Hodges et al., 2016a; Hubley-Kozey, Hatfield, & Stanish, 2013). Overall, biomechanical studies suggest that joint loading may be elevated as a result of increased co-contraction and this could lead to more rapid joint degeneration. Given this evidence of the negative impact of co-contraction, there is the need to understand why people with knee OA walk with increased co-contraction and also identify and test interventions that could lead to reduced co-contraction.

The evidence presented in the paragraph above demonstrates that people with knee OA walk with alterations in knee joint moments and muscle activation patterns when compared with matched healthy subjects. There is also emerging body of evidence to suggest that these

changes actually increase the stress on the articular cartilage. In addition, there is now emerging evidence that people with knee OA walk with an increased flexion of the trunk (Preece, Algarni, & Jones, 2018). This increased flexion may shift the position of the centre of mass relative to the hip, knee and ankle joint, which could in turn lead to changes in muscle activation patterns during walking. However, at present, it is not clear whether these alterations in moments and muscle activity patterns are a direct consequence of the disease, or the result a habitual way of walking. The aim of the work presented in this thesis was to understand the potential effect of trunk flexion on lower limb kinetics, muscle activation and muscle co-contraction during walking. Experimentally, it is not possible to separate out the contribution of pain from other factors (including trunk flexion) which may lead to the altered knee muscle activation observed in people with knee OA. Therefore, this thesis sought to first understand the effect of directly manipulating trunk flexion, both in healthy people and those with knee OA. This work was then developed by exploring differences in muscle patterns between healthy people who habitually walk with different trunk flexion angles. The final aim of the thesis was to explore a possible mechanism which may underlie increased trunk flexion, both in healthy people and those with knee OA. This mechanism focused on the length (not activation) of the hip flexor muscle. Further detail on the five studies presented in this thesis are provided below, in the next section.

1.4 Structure of thesis

This section above presented an overview of the biomechanical characteristic observed in people with knee OA. Despite the large amount of research into knee OA, little is known about the biomechanical factors that may lead to increase co-contraction in people with this condition. In this thesis, a new model to explain the observed alteration in lower limb biomechanics during walking in people with knee OA is proposed. This model is developed around an understanding of the influence of altered sagittal plane trunk inclination (trunk flexion) and the potential effect that has on lower limb kinematics, kinetics and muscle activation patterns during walking in both people with knee OA and healthy people. In the final section, the thesis explores ideas which could explain the previously observed differences in forward lean between healthy people and people with knee OA.

Following the literature review **(Chapter 2)** and a methodology chapter **(Chapter 3)**, a number of experimental studies are developed related to the ideas outlined above. These studies are presented in separate chapters:

Chapter 4: (Study 1), this study sought to compare gait characteristics between three groups: a group of patients with knee OA, an asymptomatic group of aged-matched people and an asymptomatic group of younger people. This study was performed to ensure that the characteristics of the participants studied in this thesis were consistent with previous research.

Chapter 5: (Study 2), this experimental study sought to understand the effect of imposed trunk inclination on lower limb kinematics, kinetics and muscle activation patterns in young healthy subjects during walking. Specifically, this study sought to quantify the effect of small (5°-10°) increases in trunk forward lean on lower limb kinematics, kinetics and muscle activation patterns.

Chapter 6: (Study 3), this study sought to understand the effect of imposed trunk lean inclination on lower limb kinematics, kinetics and muscle activation patterns in OA and healthy older groups during walking. This work was an extension of the research carried out in chapter 5.

Chapter 7: (Study 4), this experimental study sought to identify biomechanical differences which are associated with natural variations in trunk inclination. For this study, healthy subjects were divided into two groups (forward/backward leaners) and lower limb kinematics, kinetics and muscle activation patterns compared between the two groups. The findings were then contrasted to the differences between the OA and the healthy groups reported in chapter 4 (study 1).

Chapter 8: (Study 5), this study aimed to explore the biomechanical mechanisms which could underlie inter-individuals differences in forward lean during walking and standing. Specifically, a hypothesis related to hip flexor muscle length, pelvic position and trunk inclination was explored. A further aim of this study was to quantify possible differences in knee muscle activation between two groups of healthy with distinctly different hip flexor muscle lengths.

Chapter Two

Literature review

2.1 Pathophysiology, clinical symptoms, epidemiology and cost of knee OA

2.1.1 Pathophysiology of knee OA

Hyaline articular cartilage is found at the end of most articulating joints. This tissue functions to protect the subchondral bone by distributing the joint loads and allowing frictionless movement between the movable synovial joint surfaces during activity (Mow, Holmes, & Lai, 1984). The knee joint is an example of a synovial joint in the human body. The structure of the hyaline articular cartilage consists of chondrocytes, collagen, proteoglycans proteins and water. These factors together aim to maintain the organic matrix, permeability effects, viscoelasticity and frictional resistance of cartilages (Nordin & Frankel, 2001; Shah, Kaplan, Meislin, & Bosco III, 2007). The biomechanical properties of the hyaline articular joint components mentioned determine the load bearing and lubrication capacity of joints. However, since articular cartilage is avascular, it has limited capacity to repair (Nordin & Frankel, 2001).

Given the limited capacity for repair, damage to the hyaline articular cartilage may disturb the biomechanical properties and lead to OA in the joint. OA is a degenerative disease of the articular cartilage characterised by increasing chondrocytes synthesis, decreasing proteoglycan proteins, degeneration of collagen fibres and an increase in water content (Buckwalter & Mankin, 1998; Pritzker, 2003). Accordingly, these changes lead to a narrowing of the joint space due to softening of the articular cartilage, osteophyte formation and subchondral sclerosis (Arden & Nevitt, 2006b; Felson, 2006). In addition, other studies have found that these degenerative changes, associated with knee OA, could also affect the connective tissue around joints, such as , muscles, ligaments and synovium (Felson, 2006; Sun, 2010) which may in turn lead to altered joint mechanics (Andriacchi et al., 2004; Sun, 2010). Importantly, one of the hallmarks of knee OA is a change in the structural integrity of the articular cartilage. It is therefore important to review the accepted radiographic methods of characterising Knee.

2.1.2 Kellgren-Lawrence radiographic grades for knee OA

A considerable amount of literature has been published on radiographic knee OA. These studies have reported that the primary investigation of knee OA is conducted through radiography (Dougados et al., 1992; Ledingham, Regan, Jones, & Doherty, 1993) as it can be used to assess the severity and progression of knee OA (Kellgren & Lawrence, 1957). Whilst it has been determined that the early stage of knee OA cannot be identified by radiographic methods (Watt & Doherty, 2003), magnetic resonance imaging can be used to detect the early stage of the disease and progression of knee OA (Hayes et al., 2005). It has been observed radiographically that people with knee OA present with osteophytes, joint space narrowing, subchondral sclerosis and subchondral cysts (Huch, Kuettner, & Dieppe, 1997).

In the 1950s, the first radiographic scale was introduced by Kellgren and Lawrence to measure the severity of OA (Kellgren & Lawrence, 1957). This scale, referred to as the Kellgren-Lawrence (K-L) scale, has become the most commonly used scaling system for assessing radiographic knee OA progression in clinical research (Altman et al., 1986; Bauer et al., 2006). This system uses five levels between 0 and 4: grade 0 = no features (normal); grade 1 = doubtful – doubtful narrowing of joint space and possible osteophyte lipping; grade 2 = mild – definite osteophytes and possible narrowing of joint space; Grade 3 = moderate – multiple osteophytes, definite narrowing of joint space, some sclerosis and possible deformity of bone ends; Grade 4 = severe – large osteophytes, marked narrowing of joint space, severe sclerosis and definite deformity of bone ends (Kellgren & Lawrence, 1957; Miyazaki et al., 2002; Sharma et al., 1998).

2.1.3 Clinical symptoms of knee OA

Knee OA is described, depending on the affected side, as medial, lateral or patella-femoral knee OA. A large and growing body of literature has observed that OA in the medial compartment is more common than in the lateral compartment (Bartel, 1991; Dearborn, Eakin, & Skinner, 1996; Wise et al., 2012). Clinically, knee OA is characterised by joint pain, tenderness, muscle weakness and a limited range of motion. If the patient is experiencing these symptoms, it is known as symptomatic OA (Yang, Saris, Dhert, & Verbout, 2004). The American College of Rheumatology (ACR) established five criteria for the clinical classification

of knee OA. The ACR criteria are based on symptoms and clinical signs findings. These criteria include having knee pain on most days of the previous month and three of the following criteria: crepitus during active movement, morning stiffness for at least 30 minutes, being above 50 years of age, bone tenderness, bone enlargement and no palpable warmth (Altman et al., 1986).

Pain is the most common cause of disability in patients with OA. It has been demonstrated that people with knee OA release more of a nerve growth hormone, which is a protein associated with increased pain in OA (Stoppiello et al., 2014). Consequently, pain can negatively affect patients' activity during daily living, as well as their mood and sleep. Joint pain happens during physical activity and worsens during long periods of weight bearing, for example walking or standing (Dieppe & Lohmander, 2005). Aching or throbbing describes the nature of the pain during rest; furthermore, sharp and stabbing pain is experienced during activities such as walking (Kidd, 2006). Interestingly, it has been widely recognised that there is a weak correlation between radiographic OA (see section 2.1.2 above) and the severity of joint pain (Green, Noble, Ahuero, & Birdsall, 2006; Kidd, 2012). Indeed, a study by Hannan, Felson, and Pincus (2000) reported that out of more than 300 subjects with radiographic knee OA, only 47% experienced pain (Hannan et al., 2000). Given the lack of pain receptors in articular cartilage, pain is more likely to result from damage to innervated tissues such as subchondral bone, synovium, ligaments and joint capsules, which are responsible for nociceptive stimuli. This idea is consistent with the concept that pain in OA is not simply the result of wear of the articular cartilage.

In addition to knee pain, knee stiffness is a common symptom in patients with knee OA. It has been shown that joint stiffness increases during walking in patients with knee OA (Zeni & Higginson, 2009). Previous research has indicated that there was a correlation between increasing knee stiffness and developing osteophytes (Mazzuca et al., 2007), and falls in older people (Foley, Lord, Srikanth, Cooley, & Jones, 2006). Sorensen, Jorgensen, Rasmussen, and Skou (2014) stated that patients with knee OA have knee joint stiffness in the morning for at least 30 minutes (Sorensen et al., 2014). Furthermore, several studies have observed that patients with knee OA walk with a reduction in knee excursion (Al-Zahrani & Bakheit, 2002; Gok, 2003; Ramsey, Snyder-Mackler, Lewek, Newcomb, & Rudolph, 2007) and this may be a

result of increasing joint stiffness. Therefore, clinically, knee stiffness is an important symptom in patients with knee OA.

One of the most important clinical findings in patients with knee OA is muscle weakness. It has conclusively been shown that muscle weakness occurs among patients with knee OA (Hassan, Mockett, & Doherty, 2001; Slemenda et al., 1997). Researchers have shown that muscle weakness is associated with the onset of knee OA (Slemenda et al., 1997) and the progression of radiographic knee OA (Thorstensson, Petersson, Jacobsson, Boegård, & Roos, 2004). Muscle strength in the quadriceps and hamstring are essential for activities of daily living, for example walking and standing (Nordesjö, Nordgren, Wigren, & Kolstad, 1983). Muscle weakness in the quadriceps is widely observed in patients with knee OA (Alnahdi et al., 2012; Slemenda et al., 1997). The possible reason behind muscle weakness in OA people is that these avoid using the affected limb and, instead, try to rely on the pain free limb.

2.1.4 Incidence of knee OA

A study by Vos et al. (2012) reported that, in high-income countries, OA represents 3% of all years lived with disabilities (Vos et al., 2012). The incidence of OA increases with age (Buckwalter, Saltzman, & Brown, 2004). For instance, in the UK, OA represents 15-25% of all musculoskeletal consultations in people aged over 45 and over 75 years respectively (Jordan et al., 2007). In addition, in the England, more than four million people have OA of the knee ("Arthritis Research UK," 2016). Although OA can affect any joints of the human body, for example knees, hands and hips, it has been shown that the knee is the most commonly affected joint (Felson, 1990). Data from Conaghan, Kloppenburg, Schett, Bijlsma, and Comm (2014) reveals that approximately 90% of knee and hip joint replacements are carried out because of OA.

In addition to affecting the UK population, OA is found in the populations of many countries around the world. For example, a recent study in the United States of America (USA) reported that, between 2013 and 2015, more than 50 million people were diagnosed with arthritis and the most common form of arthritis was OA (Barbour, Helmick, Boring, & Brady, 2017). Furthermore, it was expected that 30% of the USA population would have knee OA by 2030 (Lawrence et al., 2008). According to Deshpande et al. (2016), more than 14 million people in

the USA suffer from knee OA and nearly two million people under the age of 45 have symptomatic knee OA, with the annual incidence of knee OA being highest between the ages of 55 and 64 years old (Deshpande et al., 2016). OA is therefore costly to the health care system of any country.

2.1.5 Social and individual cost

The economic cost of OA is a result of direct costs, such as medical expenditures. such as surgery, diagnostic tests, allied health professionals' visits and hospitalisations. The treatment cost of OA is different between countries; in the UK, between 1991 and 2006, more than 20,000 joint replacements were carried out on knees and the estimated mortality-adjusted lifetime risk was 10.8%-8.1% for women and men, respectively (Culliford et al., 2012). The USA also reports very large costs – OA is costing the USA economy more than 100 billion per year (Sandell, 2012). Furthermore, in Canada it was estimated that the economic burden of OA in 2010 was more than $10 billion for direct costs and more than $ 17 billion for indirect costs (Bombardier, Hawker, & Mosher, 2016). Indirect costs relate to a patient's time, health and productivity; for instance, an inability to work because of pain and disability (Leardini et al., 2004; March & Bachmeier, 1997; Ruiz et al., 2013).

Data from several sources have identified that indirect health care costs may be higher than direct costs (Boonen & Severens, 2011; Roos, 2005). Pain in individuals with OA will lead to a reduction in their quality of life and negatively impact their mood, relationships and sleep. In a cross-sectional study, DiBonaventura et al. (2012) stated that workers with OA more frequently reported pain, a negative impact on their life, a reduction in their productivity and frequent use of health care resources. Similarly, many patients with OA reported that the average cost to them of their travel and treatment was more than £450. Moreover, one-third of people with OA retire an average of 8 years early or reduce their hours of working, due to the effects of OA (Arthritis Care, 2017). Therefore, developing effective treatments for OA is needed in order to decrease the social, economic and direct healthcare costs of the disease.

2.2 Risk factors of knee OA and current clinical management approaches

2.2.1 General risk factors

There have been several studies in the literature reporting the risk factors of knee OA. These studies have identified systemic risk factors, such as age, race, female gender and hormonal factors. Studies have found that the prevalence of knee OA increases with increasing age and it is one of the strongest risk factor for OA (Litwic et al., 2013; Neogi & Zhang, 2013). Arden and Nevitt (2006a) reported that people aged 65 have evidence of radiographic knee OA and, in those aged 75 and over, this increases to 80% (Arden & Nevitt, 2006a). In addition, data from meta-analysis reported that knee OA is higher in women than men (Srikanth et al., 2005) mainly due to hormonal changes, especially oestrogen which is important to regulate the bone metabolism (Neogi & Zhang, 2013). Both age and gender are the most important non-modifiable risk factors.

In addition to age and gender, a number of studies have found that ethnic differences and bone mineral density play a role in OA (Felson & Nevitt, 1998; Johnson & Hunter, 2014; Neogi & Zhang, 2013). Previous studies found that radiographic hip OA in female African-American people is more common than in American white women (Nelson et al., 2010) and radiographic knee OA is more common in African-American people generally compared to Caucasians (Nelson et al., 2012). Although these are considered to be non-modifiable factors, other factors such as obesity, deficiency of vitamin D, knee alignment, joint injury and heavy physical activity are considered to be modifiable risk factors (Heidari, 2011; Silverwood et al., 2015b) and these are discussed in subsequent sections.

2.2.2 Modifiable risk factors

In addition to the more general risk factors discussed above, studies have shown that a deficiency of vitamin D, which is known to be important to bone metabolism, may have a negative effect on knee OA (McAlindon et al., 1996; Zhang et al., 2014). Therefore, people with knee OA are often recommended to take Vitamin D supplements (Bennell, Hall, & Hinman, 2016). It is also clear from previous studies that obesity is one of the major risk factors for knee OA disease (Cooper et al., 2000; Felson, Zhang, Anthony, Naimark, & Anderson, 1992; Sowers & Karvonen-Gutierrez, 2010). Obesity is characterized by a body mass index of more than 30 Kg/m^2 (World Health Organization, 2006). Furthermore, the

findings from several studies have demonstrated that there is a link between obesity and the increased risk of knee OA (Hart, Doyle, & Spector, 1999; Spector, Hart, & Doyle, 1994). Data from the Framingham Heart Cohort study found that there was a substantial increase in the risk of knee OA in obese people, with a stronger association in women than in men (Felson, Anderson, Naimark, Walker, & Meenan, 1988). It was reported that obesity may negatively impact on knee articular cartilage (Pottie et al., 2006; Wang et al., 2009).

The biomechanical factors play an important role in knee joint loading and have been associated with disease progression. It has been suggested that OA could be initially precipitated by kinematic and kinetic changes, followed by damage to cartilage that fails to adapt and is unable to distribute the forces between articular surfaces (Andriacchi & Mundermann, 2006; Buckwalter, Mankin, & Grodzinsky, 2005). For example, previous studies reported that alterations in knee adduction moment (KAM) (Bennell et al., 2011; Chang et al., 2015; Chehab, Favre, Erhart-Hledik, & Andriacchi, 2014), knee flexor moment (KFM) (Chehab et al., 2014; Erhart-Hledik, Favre, & Andriacchi, 2015) and joint malalignment (Sharma et al., 2001) are more closely associated with patients with knee OA than with healthy subjects. In addition, elevated levels of lower limb muscle activation patterns and co-contraction between pairs of knee muscles were observed in people with knee OA (Astephen, Deluzio, Caldwell, Dunbar, & Hubley-Kozey, 2008; Heiden, Lloyd, & Ackland, 2009; Hortobagyi et al., 2005; Schmitt & Rudolph, 2008).

These biomechanical changes mentioned above have been shown to negatively affect functional joint loading, which has a crucial effect on joint degeneration. In particular, recent modelling studies demonstrated that alterations in knee muscle patterns, which are characteristic in knee OA patients, may lead to excessive joint loading during walking (Brandon et al., 2014; Sritharan et al., 2016). Moreover, recent evidence from longitudinal studies has demonstrated that there is a link between altered muscle activation (co-contraction) and the progression of knee OA (Hodges et al., 2016a; Hubley-Kozey, Hatfield, et al., 2013) (See section 2.7.1 for more details). Therefore, in order to understand the pathomechanics of the disease, it is essential to comprehensively understand the biomechanical characteristics of the knee joint. These ideas are explored in more detail in sections 2.3 to 2.8. Before the effects of biomechanical factors on knee joint loading are

discussed, it is necessary here to cover important clinical interventions and the treatment of knee OA.

2.2.3 Current clinical management of knee OA

Despite the growing number of people with OA, there is no cure for OA. Therefore, the management of knee OA aims to relieve pain, improve physical function and, where possible, prevent the progression of the disease (Jordan et al., 2003; Zhang et al., 2005). The first line therapy for people with knee OA is pharmacological treatment such as analgesics (paracetamol, opioids and capsaicin), anti-inflammatory drugs (non-steroidal anti-inflammatory drugs –NSAIDs and COX-2 inhibitors) and intraarticular corticosteroids (Michael, Schluter-Brust, & Eysel, 2010; Vad & Dysart, 2012). Although anti-inflammatory or analgesic therapies are widely used in treating people with OA, it has been found that extensive use of these drugs might be linked with an increase in joint forces (Schnitzer, Popovich, Andersson, & Andriacchi, 1993), risk of gastrointestinal problems (Ishihara et al., 2014) and renal failure (Moon et al., 2011). Therefore, care should be taken in the pharmacological treatment.

In addition to pharmacological treatments, non-pharmacological treatments are considered an important component of managing patients with mild and moderate knee OA (Bijlsma & Knahr, 2007; van Raaij, Reijman, Brouwer, Bierma-Zeinstra, & Verhaar, 2010). Physiotherapy treatment is a common conservative treatment to reduce pain and stiffness, and improve the mobility and function for patients with knee OA (Bartholdy et al., 2017; Conaghan, Dickson, & Grant, 2008). In the UK, physiotherapy treatment is recommend by NICE guidelines to reduce the pain in the short term and improve the function in those patients (Conaghan et al., 2008). The physiotherapy treatment approaches in the literature include therapeutic exercises (Bartholdy et al., 2017), manual therapy (Anwer, Alghadir, Zafar, & Brismée, 2018), electrotherapy (Mascarin et al., 2012) and patient education (Lopez-Olivo et al., 2018). In addition, stretching, aerobic and Tai Chi exercises have been found to be beneficial for improving pain and function in people with knee OA (Bennell & Hinman, 2011).

A recent systematic review concluded that manual therapy with exercise may reduce pain, improve function, and physical performance in short-term compared to exercise alone for

patients with knee OA (Anwer et al., 2018). However, many other studies have concluded that there is a lack of clinical effectiveness of current physiotherapy treatments, especially over a longer time period (Deyle et al., 2000; Jansen, Viechtbauer, Lenssen, Hendriks, & de Bie, 2011). Moreover, the effect of exercise on knee OA may not be effective in reducing joint loading (Bennell et al., 2016; Sled, Khoja, Deluzio, Olney, & Culham, 2010) and this may explain why only small improvements in pain and function are typically observed after physiotherapy therapeutic exercises (Anwer et al., 2018; Bennell et al., 2016). Furthermore, in recent systematic review, it was concluded that there was a lack of dosage of the exercise therapy with patients with knee OA (Fransen et al., 2015). Given the lack of effectiveness of current exercise management of knee OA, more focus on other treatments, which have the potential to reduce joint loading, is needed.

A number of researchers have investigated the use of footwear-type interventions, including lateral wedge insoles, which aim to alter knee joint loading. For example, the lateral wedge insole is designed to change the direction of ground reaction force (GRF) vector, reduce the knee adduction moment (KAM) and decrease the load on the medial compartment of the knee. Furthermore, biomechanical studies reported that the use of lateral wedge insole reduces knee joint loading (Barrios, Butler, Crenshaw, Royer, & Davis, 2013; Chapman, Parkes, Forsythe, Felson, & Jones, 2015) and pain (Arazpour et al., 2013), compared to normal shoes without insoles. A number of authors have studied the effect of footwear on knee loading and demonstrated that specially designed footwear could significantly reduce the KAM in people with knee OA (Madden, Kean, Wrigley, Bennell, & Hinman, 2015, 2017). However, some studies did not identify a significant reduction in knee joint loading and pain (Santo, Roper, Dufek, & Mercer, 2012; Sobhani et al., 2013). Although footwear may affect frontal plane moments, it unlikely to affect co-contraction. Given the link between co-contraction and loading, research is needed into other conservative managements which have the potential to change co-contraction and therefore loading.

Surgical interventions in OA include arthroscopy, osteotomy and knee joint replacement; some procedures aim to correct biomechanical abnormalities. Researchers has shown that surgical treatment is effective for reducing the symptoms of the disease and improving patients' functions (Briem, Ramsey, Newcomb, Rudolph, & Snyder-Mackler, 2007; Yang et al., 2004). Furthermore, previous studies reported that arthroplasty procedures, which involve

changing the articulating surface of the joint, could relieve pain, improve function, and increase tolerance for the performance of activities of daily living in patients with knee OA (Aujla & Esler, 2017; da Silva, Santos, Júnior, & Matos, 2014; Khan, Evaniew, Bedi, Ayeni, & Bhandari, 2014). On the other hand, consideration should be given to the side effects of surgical treatment; for example the cost of the treatment is extremely expensive for the National Health Services (NHS) and it negatively impacts on patients with regard to recovery time and functional independency (Buckwalter & Lohmander, 1994; Skou et al., 2015).

Current evidence demonstrates that conservative physiotherapy management of knee OA is only associated short-term reductions in pain which are typically small (Anwer et al., 2018; Conaghan et al., 2008). It is possible that current interventions for managing knee OA are not effective because they do not directly target the knee joint loading. Clearly, a range of biomechanical factors will contribute to the loading at the knee joint surface. Therefore, there is a need to understand the mechanics of gait and joint loading in knee OA and to use this understanding to identify potential new treatment programmers for this disease. In the following section, a short summary of biomechanics of gait is presented, followed by a detailed discussion of the specific biomechanical characteristic of people with knee OA.

2.3 Measuring the biomechanics of gait

2.3.1 Gait analysis

The science of biomechanics aims to understand the structure and function of mechanical aspects of biological systems, including how bones, tendons, ligaments and muscles work together in terms of producing movement (Hatze, 1974; Rau, Disselhorst-Klug, & Schmidt, 2000). To fully understand biomechanical research in knee OA, it is first necessary define gait and the gait cycle. Gait is a form of bipedal locomotion in which there is an alternate movement between two lower limbs to provide support and propulsion (Baker, 2006; Hatze, 1974). Furthermore, the term ‘gait cycle’ refers to the period of time for two steps and is started and measured from the heel strike of one foot to the next heel strike of the same foot (Murray, Drought, & Kory, 1964).

The gait cycle has two major phases (Figure 2-1); the stance phase when the foot is in contact with the ground, which represents approximately 60% of the gait cycle, and the swing phase

when the foot is not in contact with the ground, which represent the remaining 40% of the gait cycle. There are two intervals in the stance determined by whether one or two feet are in contact with the ground: single limb support and double limb support. In addition to the major phases, there are eight sub-phases of the gait cycle, which are initial contact (heel strike), loading response (foot flat), mid stance, terminal stance (heel off), and pre swing (toe off) in the stance phase. The rest of are part of the swing phase - initial swing, mid swing and terminal swing phases (Levine, Richards, Whittle, & Whittle, 2012; Nordin & Frankel, 2001; Perry, 2010).

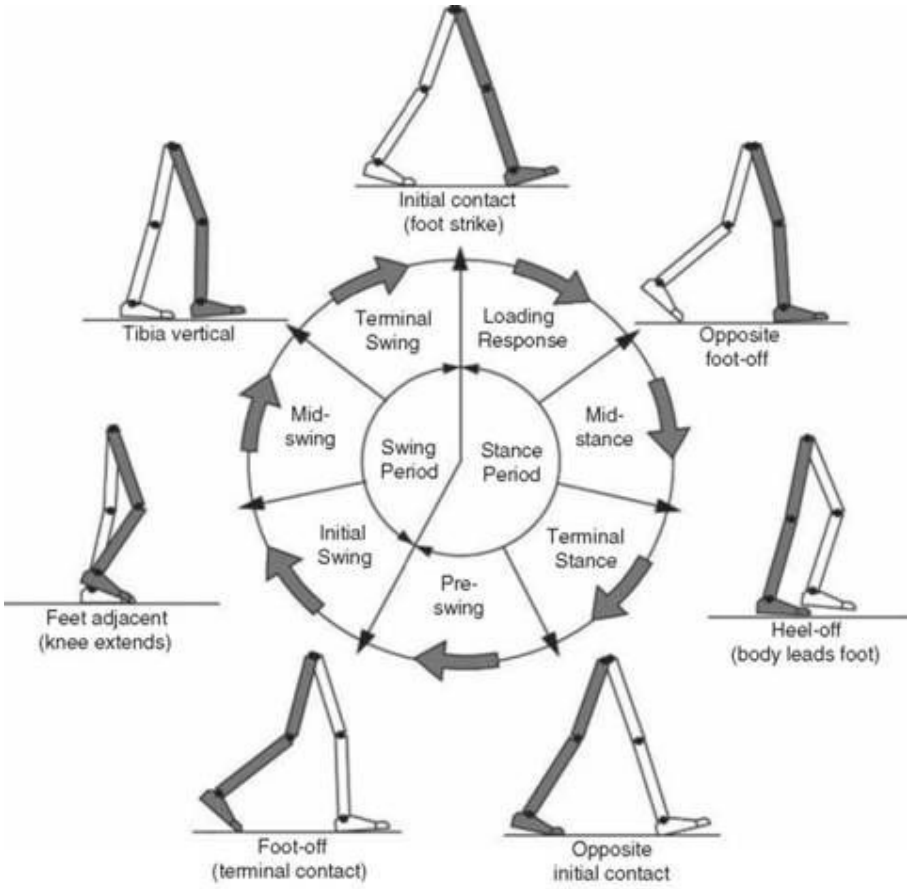


Figure 2-1 Stance and swing phases of the gait cycle adopted from (O'Sullivan, Schmitz, & Fulk, 2013).

2.3.2 Kinematics and kinetics

The term 'Kinematic' provides a basic description of gait that includes distance (spatial), time (temporal) and measurement of joint angles, whereas the term 'kinetic' is used to describe

the forces and moments acting on the body during gait. Moments are the turning forces created by muscles around the joint (internal moment) and the GRF (external moment), and they give some indication of the load experienced by the knee joint. Muscles, tendons and ligaments produce internal moments of force across joints during different periods of the gait cycle, whereas external moments result primarily from the action of gravity, which is understood through the ground reaction force (GRF). For instance, in normal gait at loading response, the line of the GRF passes behind the knee joint, producing an external knee flexion moment. During the same period, contraction of the quadriceps muscles generates an internal knee extensor moment as shown in the Figure 2-2 below (Levine et al., 2012; Nordin & Frankel, 2001; Perry, 2010). For a full understanding of normal gait, we need to know which muscles are active during the different phases of the gait cycle.

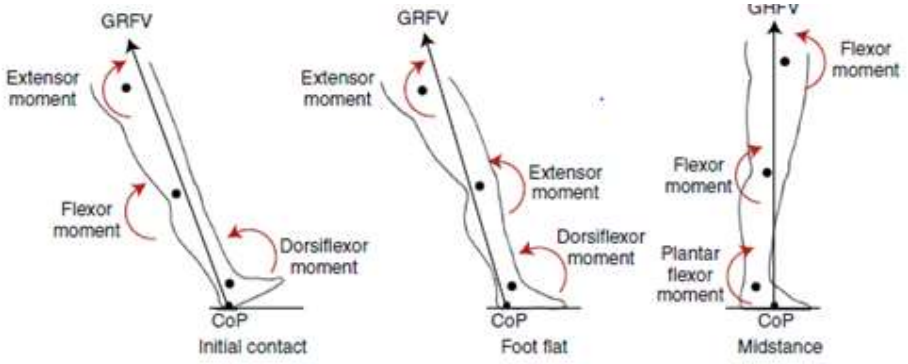


Figure 2-2 Ground reaction force vector during stance phase of gait cycle adopted from (Levine et al., 2012)

2.3.3 Electromyography

Electromyography (EMG) is a technique that is used to study and measure the electrical activity of contracting muscle (Basmajian & De Luca, 1985) and it is widely used in gait analysis. EMG is used in physiological, biomechanical and kinesiological studies and could be indicated for clinical treatment, evaluation, and biofeedback. In gait analysis, EMG can be synchronised with kinematics and/or kinetics to link the muscle activity with another events in the gait cycle. Although EMG measures electrical activity at the surface of the skin, it cannot be used to differentiate between the different types of contractions, such as concentric and eccentric. EMG is often used to provide information about muscle timing, thus it can determine the onset and termination of muscle contraction during activity (Allison, 2003). In

addition, EMG can measure the muscle amplitude, which is important to determine the neuromuscular control of the body (Cram, 1998).

2.4 Gait changes which are associated with knee OA

2.4.1 Altered spatiotemporal variables and knee joint kinematics in people with knee OA

Previous studies have identified the changes in the spatiotemporal gait characteristics of people with knee OA. A number of studies have observed that individuals with knee OA walk with a slow speed, short step lengths and a large double support period (Al-Zahrani & Bakheit, 2002; Kaufman et al., 2001; Messier et al., 2005), reduce cadence, stride length and increase stance time (Al-Zahrani & Bakheit, 2002), compared with healthy control subjects. In addition, it has been found that walking speed is correlated with the severity of radiographic knee OA (Astephen, Deluzio, Caldwell, & Dunbar, 2008) and it has been suggested that the reason behind the reduction in speed is to help decrease the load on the knee's articular surface (Mundermann, Dyrby, Hurwitz, Sharma, & Andriacchi, 2004b).

There are contradictory findings between studies investigating differences spatiotemporal parameters among individuals with knee OA and healthy counterparts (Landry, McKean, Hubley-Kozey, Stanish, & Deluzio, 2007; Mundermann, Dyrby, & Andriacchi, 2005). These different findings could be related to the demographic characteristics of the subjects in the different research studies (Astephen, Deluzio, Caldwell, & Dunbar, 2008) or to the differences in the severity of OA between the different research studies (Rutherford, Baker, Wong, & Stanish, 2017). Although it has been suggested that alterations in spatiotemporal parameters in people with knee OA could increase knee stability while walking, or be a strategy to attempt to decrease knee joint loading (Boyer, Johnson, Banks, Jewell, & Hafer, 2017), this is unlikely unless they are accompanied by other changes in moments and/or muscle patterns. Spatiotemporal give little insight into knee joint loading, therefore there is the need to look to other biomechanical parameters that could give better insight into joint loading.

In addition to alterations in the spatiotemporal parameters in people with knee OA, it has been noticed that knee kinematics change in people with knee OA while walking. Previous research findings into the knee kinematics of people with knee OA and healthy subjects have been inconsistent and contradictory. Some studies have reported that people with knee OA

walk with a greater knee flexion angle during the early stance phase (Baliunas et al., 2002; Childs et al., 2004), however other studies have reported that people with knee OA walk with greater knee extension angles (Rudolph, Schmitt, & Lewek, 2007; Smith, Lloyd, & Wood, 2004). Furthermore, some studies have demonstrated that people with knee OA walk with a reduction in the knee flexion excursion during loading (Childs et al., 2004; Lewek, Rudolph, & Snyder-Mackler, 2004a; Lewek, Scholz, Rudolph, & Snyder-Mackler, 2006; Rudolph et al., 2007), while some researchers reported that OA patients walk with greater knee flexion angle during initial contact (Heiden et al., 2009; Rudolph et al., 2007). These inconsistent results obtained with people with knee OA show varied kinematic responses. Nevertheless, kinematics give little insight into joint loading and do not provide insight into differences in joint loading between people with knee OA and healthy subjects. Therefore, we need to look at measures that might give better insight into joint loading, such as kinetics.

2.4.2 Altered knee kinetics in people with knee OA

Joint moments are the turning forces created by muscles around the joint and the GRF, and they give some indication of the load experienced by the knee joint (Winby, Lloyd, Besier, & Kirk, 2009). As explained earlier the external moment is determined by the magnitude of the GRF and the perpendicular distance from the GRF vector to the joint centre. Previous research showed that people with knee OA demonstrate changes in joint moments during walking (Baliunas et al., 2002) and other activities (Childs et al., 2004) compared to healthy subjects. It is possible that these changes in joint moments could relate to alterations in joint loading in people with knee OA; this has been studied primarily in two planes: the sagittal plane and the frontal plane. The key differences are described in the following paragraphs.

The frontal moment, or the coronal plane moment as it is more commonly known, has received much research attention. The KAM describes the frontal plane moment, which is an indicator of the mechanical load distribution between the medial and lateral compartment of the knee joint (Meyer et al., 2013; Moyer et al., 2015). When walking in the stance phase, the GRF vector passes medial to the knee joint, which produces moment in the frontal plane and leads to adduction of the knee (Schipplein & Andriacchi, 1991). It has been shown that an increase in KAM may lead to an increase in knee joint loads across the medial compartment (Hinman et al., 2013). The relationship between KAM and knee OA has been widely

investigated. For example, an increase in KAM has been linked to knee OA symptoms (Kim, Richards, Jones, & Hegab, 2004; Prodromos, Andriacchi, & Galante, 1985b), severity (Birmingham, Hunt, Jones, Jenkyn, & Giffin, 2007) and progression (Chang et al., 2015). Moreover, many studies have found that the KAM is elevated in individuals with knee OA compared to healthy subjects (Baliunas et al., 2002; Mundermann et al., 2004a). Recent evidence concluded that both sagittal and frontal planes give more insight into knee joint loading than KAM alone (Chehab et al., 2014; Erhart-Hledik et al., 2015; Manal, Gardinier, Buchanan, & Snyder-Mackler, 2015). Therefore, it is important to understand the differences in sagittal knee moment which are characteristics of people with knee OA.

In addition to changes in KAM, knee extensor moment has been shown to be changed in people with knee OA. The knee extensor moment is an indicator of mechanical loading in the sagittal plane and determined by the sagittal plane GRF vector and moment arm that flexes the knee, as shown in Figure 2-3. Recently, studies have highlighted the importance of analysing sagittal plane in people with knee OA, especially the knee extensor moment (Chehab et al., 2014; Edd et al., 2017; Erhart-Hledik et al., 2015). Specifically, several studies have reported that individuals with knee OA walk with a lower knee extensor moment than healthy individuals during the mid-stance phase (Astephen, Deluzio, Caldwell, & Dunbar, 2008; Astephen Wilson, Stanish, & Hubley-Kozey, 2017; Huang et al., 2008; Kaufman et al., 2001; Liu et al., 2014; Manal et al., 2015; Sritharan et al., 2016). In earlier studies have found that people with knee OA walk with increased knee extensor moment (Chehab et al., 2014). These contradictory findings may indicate that alterations in knee joint moment are the result of non-disease related factors, such as upper body position. This idea will be discussed in more depth in subsequent sections.

In addition to changes in knee moment, Liu et al. (2014) and Huang et al. (2008) found that hip extensor moments in patients with bilateral knee OA are increased compared to healthy subjects. In summary, previous studies have demonstrated differences in sagittal plane moments in people with knee OA when compared to healthy individuals. These differences are characterised by increased in the magnitude of hip extensor and plantar flexor moment during early and mid-stance phase during walking. Given that people with knee OA walk with alteration in sagittal lower limb moments, again a possible explanation of this might be the alteration in upper body position. This idea is discussed in more detail in section 2.9 below.

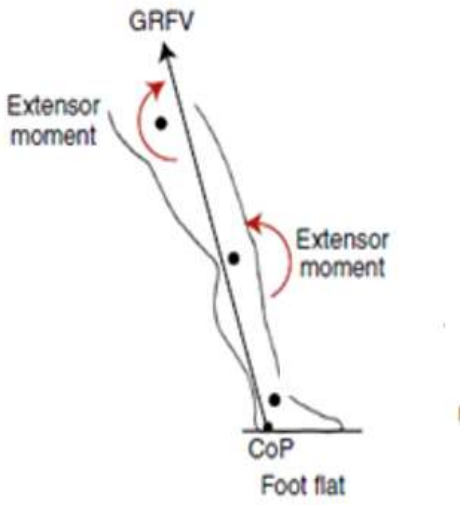


Figure 2-3 Internal hip and knee extensors moment Adopted from (Levine et al., 2012).

2.4.3 Differences in muscle activity between healthy subject and people with knee OA

Previous studies have found differences in muscle activity amplitude while walking for people with knee OA compared to healthy subjects (Astephen, Deluzio, Caldwell, Dunbar, et al., 2008; Hortobagyi et al., 2005; Rutherford, Hubley-Kozey, Stanish, & Dunbar, 2011). For example, a study by (Hubley-Kozey, Deluzio, Landry, McNutt, & Stanish, 2006) aimed to understand whether knee muscle activity during walking in people with knee OA differed from healthy controls. It was concluded that people with knee OA walk with increased muscle activity of the hamstrings, quadriceps and gastrocnemius compared to healthy subjects (Hubley-Kozey et al., 2006). These alterations in neuromuscular are considered to have important clinical implications and may influence course of the disease (Hortobagyi et al., 2005; Rutherford, Hubley-Kozey, & Stanish, 2013).

In addition to alteration in the amplitude of muscle activity, previous studies have reported differences in muscle activity duration between individuals with knee OA and controls (Astephen, Deluzio, Caldwell, & Dunbar, 2008; Childs et al., 2004; Rutherford, Hubley-Kozey, Stanish, et al., 2011). For example, a study by Childs et al. (2004) showed differences between people with knee OA and healthy control subjects in the temporal activation of the

hamstrings, quadriceps and gastrocnemius during walking. The data from this study is illustrated in Figure 2-4 and shows prolonged periods of muscle activity during the stance phase in the OA group. This shows that, muscles in individuals with knee OA turn on sooner and turn off later that the same muscles of healthy subjects (Childs et al., 2004). Furthermore, it has been demonstrated that, in severe OA patients, the hamstring muscle (medial and lateral) had greater activity in the stance phase and the gastrocnemius muscle (medial) also had greater activity in the early stance phase (Astephen, Deluzio, Caldwell, Dunbar, et al., 2008). These findings motivated researchers to study the relationship between the alterations in muscle activity patterns and disease progression/pain.

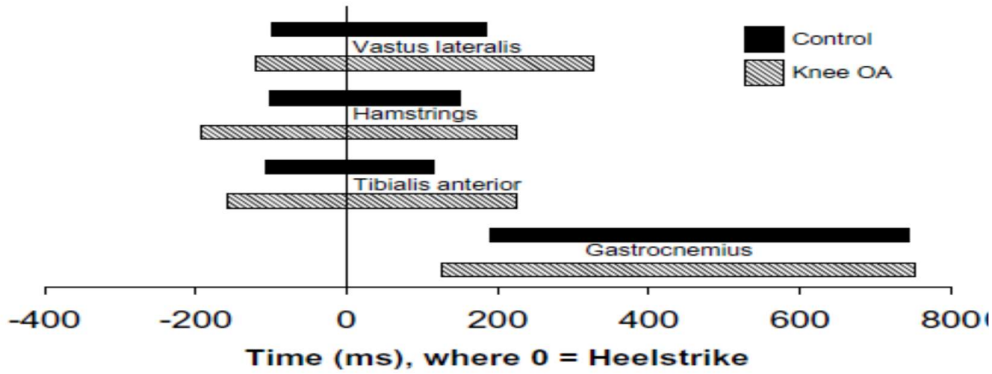


Figure 2-4 Duration of muscle activity in people with knee OA and healthy subjects. Adopted from (Childs et al., 2004).

A study by Wilson, Deluzio, Dunbar, Caldwell, and Hubey-Kozey (2011) was the first attempt to understand the relationship between muscle activity pattern and pain severity. The authors found that higher pain was correlated with a higher medial hamstring muscle activity in the early stance phase (Wilson et al., 2011). In addition, a recent study by Rutherford et al. (2013) investigated the relationship between alterations in knee muscle activity during walking and disease severity (defined by radiographic KL-grades) in people with knee OA. They found that changes in muscle activation patterns were associated with disease, with more advanced knee OA being associated with increased muscle activation (Rutherford et al., 2013). These findings demonstrate that there is an association between alterations in the muscle activity of the lower limb in knee OA patients and disease status/pain. However, it not clear whether

these altered muscle patterns are causative, whether they could be a response to the disease process or whether they may be related to another aspect of gait.

It is interesting to interpret the results of studies which have sought to compared differences in muscle patterns between the affected and unaffected limb in people with knee OA. These studies have typically investigated differences in muscle activity between people with knee OA and healthy subjects as well as comparing between the two limbs of people with knee OA. In general these studies have demonstrated differences in muscle activation patterns between healthy people and those with knee OA but similar patterns between the ipsilateral and contralateral limb in individuals with knee OA (Astephen, Deluzio, Caldwell, Dunbar, et al., 2008; Childs et al., 2004; Gustafso, Anderton, Sowa, Piva, & Farrokhi, 2019; Hortobagyi et al., 2005; Hubley-Kozey et al., 2006; Rutherford et al., 2013). These findings suggest that altered knee muscle activity in people with knee OA may not be simply the result of pain and/or instability and may have another biomechanical driver.

2.5 Muscle co-contraction in people with knee OA

Muscle co-contraction is defined as the simultaneous contraction of both the agonist and antagonist muscles around the joint. Activation of leg muscles can happen selectively and with precise timing which enables the individual interact successfully with the physical world (Doorenbosch, Harlaar, Roebroeck, & Lankhorst, 1994; van Ingen Schenau, Boots, De Groot, Snackers, & Van Woensel, 1992). Alternatively activation can occur more generally as a global co-contraction pattern that could limit joint motion (Zeni et al., 2010a). Although previous authors suggest that co-contraction between the hamstring and quadriceps muscles may improve knee stability (Aagaard et al., 2000; Baratta et al., 1988; Hirokawa, Solomonow, Luo, Lu, & D'ambrosia, 1991), it has also been suggested that prolonged or higher co-contraction may lead to joint degeneration (Andriacchi, 1994; Meyer et al., 2013). It has commonly been reported that patients with knee OA walk with excessive muscular co-contraction (this will be discussed below). However, before this research into co-contraction is discussed, it is important to outline the different approaches to quantifying this phenomenon.

A number of methods have been proposed to characterise knee muscle co-contraction during walking (Heiden et al., 2009; Lewek et al., 2004a; Winby, Gerus, Kirk, & Lloyd, 2013). One

method uses the ratio of peak muscle activity to calculate muscle co-contraction and gives an indication of the relative activity of the opposing muscles, without reference to the total level of activity. Although this method identifies the dominant muscle group, it does not account for the net activity muscle and this might make it inappropriate as a predictor for joint loading. The second method is a separate summing of the medial and lateral muscles activation, an approach which does reflect the overall level of muscle activity (Winby et al., 2013). Interestingly, a recently modelling study by Winby et al. (2013) examined the relationship between predicted joint loading and the two methods of calculating co-contraction. This study showed that simply summing the activity of the agonist and antagonist to measure co-contraction gave the best indication of joint loading.

A considerable amount of literature has been published on muscle co-contraction between the quadriceps and hamstring muscles in people with knee OA (Childs et al., 2004; Hodges et al., 2016a; Hortobagyi et al., 2005; Ramsey, Briem, Axe, & Snyder-Mackler, 2007; Zeni et al., 2010a). For example, Hortobagyi et al. (2005) compared quadriceps to hamstring muscles during activities of daily living (walking, stair ascent and descent). Two groups of individuals, one with knee OA patients and the other with matched healthy subjects, were recruited. The authors used the ratio method to calculate co-contraction and found that people with knee OA had a significant increase in the lateral muscles co-contraction between the quadriceps and hamstring muscle. In addition, Heiden et al. (2009) calculate co-contraction between the quadriceps and hamstring muscles by summing the separate medial and lateral co-contraction EMG curves and found that people with both knee OA walk with higher co-contraction compared with matched healthy subjects (Heiden et al., 2009). Taken together, these studies show that people with knee OA walk with increasing co-contraction between the quadriceps and hamstring muscles when compared to matched healthy subjects.

In addition to co-contraction between the quadriceps and hamstring muscles, some studies have investigated co-contraction between the quadriceps and gastrocnemius muscles (Childs et al., 2004; Schmitt & Rudolph, 2008; Sritharan et al., 2016). For example, Schmitt and Rudolph (2008) used the ratio method to calculate the co-contraction and observed that people with knee OA walk with higher muscle co-contraction between medial quadriceps and medial gastrocnemius (Schmitt & Rudolph, 2008). This finding was replicated in a subsequent study which also showed that people with knee OA walk with greater co-contraction between

these muscles than healthy people (Heiden et al., 2009). Overall, these studies demonstrate that OA patients walk with higher co-contraction between the quadriceps and gastrocnemius muscles.

Although most researcher has examined muscle co-contraction in the affected limb in people with knee OA, there have been a small number of studies which have compared co-contraction between the affected and unaffected limb. For example, Metcalfe et al. (2013) recruited 20 individual with knee OA and studied the EMG of lower limb muscle activity for both limbs during walking. They found that patients with knee OA had a significant increase in hamstring and quadriceps muscle co-contraction bilaterally compared to healthy participants. These findings demonstrate that increased knee muscle co-contraction in people with knee OA is unlikely to be just the result of pain and is likely to have another biomechanical driver.

Taken together, the research studies discussed above show that people with knee OA walk with higher co-contraction between the quadriceps, hamstring and gastrocnemius muscles than healthy subjects. Furthermore, these altered muscle patterns appear to be present in both limbs of people with unilateral OA. It is possible that these higher muscle co-contractions may increase loads at the knee joint surface. This idea is discussed in more detail below.

2.6 Muscle co-contraction and joint loading

The knee joint is required to bear between 2 and 4 times of the body weight during activities such as walking (Morrison, 1970). In addition, the medial compartment of the knee joint, when the knee is loaded, may withstand 70% of the load on the knee joint (Johnson, Leitl, & Waugh, 1980; Morrison, 1970). As stated earlier, knee OA is a mechanical disease affected by the magnitude of the compressive load on the knee joint (Brandt, Dieppe, & Radin, 2009), and repetitive cycle (dynamic) knee joint loading is associated with pathogenesis of knee OA (Andriacchi & Mundermann, 2006; Sharma et al., 1998). Given the large mechanical loads placed on the knee, there is the need to understand how mechanical loading in the knee joint could affect the integrity of anatomical knee structures. This understanding can be achieved through vivo methods (including animals studies) or through non-invasive gait analysis (Pandy & Andriacchi, 2010).

2.6.1 In vivo measures of knee joint loading

Researchers have developed telomerised implants to measure the joint contact forces in vivo in a laboratory setting. With this technique it is possible to precisely quantify the loads the joint surface during functional tasks, such as walking. Data from these implants supports the idea that people with knee OA walk with altered in knee joint mechanics which could lead to increase the rate of knee OA progression (Miyazaki et al., 2002). Interestingly, some studies have shown that these alteration may influence the outcome of knee OA treatment (Prodromos, Andriacchi, & Galante, 1985a; Wang, Kuo, Andriacchi, & Galante, 1990).

Recent research by Trepczynski et al. (2018) used instrumented knee implants to understand the role of muscle co-contraction on vivo knee joint load in nine patients with total knee replacement assessed 26 ± 13 months post-operatively. The authors combined gait analysis with lower limb EMG activity and a musculoskeletal model to measure the in vivo tibia-femoral force during daily living activities (walking, stair ascent and descent). The result showed that higher quadriceps and gastrocnemius muscle co-contraction led to approximately a 50% increase in knee joint contact force in late stance during walking. Authors demonstrated that reducing quadriceps, hamstring and gastrocnemius muscle activity levels should be taken into consideration to reduce such overloading and prevent future joint failure in patients with knee OA.

In addition to in vivo methods, laboratory-based research on animals can be used to understand how cartilage responds to different loading patterns. For example, (Radin et al., 1984) studied the effect of the dynamic loading (repetitive impulse loading) in rabbits. The result showed that the dynamic loading could lead to overload the musculoskeletal tissue within the joint causing stiffening of the articular cartilage and subchondral bone (Radin et al., 1984). In addition, animal modelling study by Chen, Burton-Wurster, Lust, Bank, and Tekoppele (1999) found that sustained compressive load could lead to damage to the articular cartilage. These studies demonstrate that specific loading patterns may lead to OA-related changes in articular cartilage and go some way to explaining OA progression. However, to date there have been no animal studies which have sought to understand how co-contraction could affect cartilage loads. For insight into this phenomenon, it is necessary to look to gait analysis and modelling studies.

2.6.2 Modelling studies used to understand how co-contraction affects knee loading

The knee is a synovial, tricompartmental joint, comprised of the medial and lateral tibiofemoral and the patellofemoral compartment. The knee joint plays an important role in transmitting the load from the upper body to the foot during weight bearing activity, such as walking. Figure 2-5 shows the major muscles that cross the knee joint, which include the quadriceps (vastus medialis and lateralis), hamstrings (biceps femoris and semitendinosus), and the muscles of the calf (medial and lateral gastrocnemius).

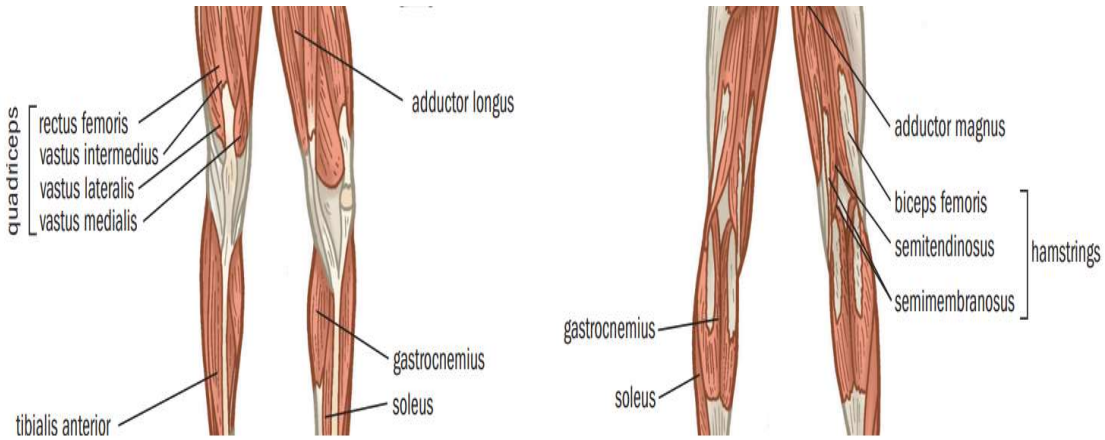


Figure 2-5 knee muscles (front and back view). Adopted from (Hoehn & Marieb, 2007).

The total compressive force in human joints is determined by four primary factors. These are the action of the muscles around the joint (Pandy & Andriacchi, 2010; Sowers & Karvonen-Gutierrez, 2010), the GRF, gravitational force, and the effects of ligament and soft tissue constraints (Pandy & Andriacchi, 2010). It is clear that, in order to fully understand joint loading in knee OA, it is essential to take into account the contributions of the muscles and their contributions to joint loading (Selistre et al., 2017; Winby et al., 2013). In line with this idea, some recent studies have used musculoskeletal modelling to understand how inter-subject variability in muscle activation patterns may lead to differences in joint loading. These studies are discussed in detail below.

A study by Brandon et al. (2014) developed an EMG-based modelling approach to try to understand how alterations in hamstring and quadriceps muscle activity may affect medial and lateral knee joint loading during walking. With their study, control (baseline) joint loading

during normal walking for eight moderate medial knee OA subjects was first computed. Muscle activations were then calculated, while kinematics and kinetics were held constant, and the model used to calculate the corresponding change in joint loading. As can be seen in the Figure 2-6 (A+B), plots show that there are two clear peaks in the medial and lateral load which occur at approximately 10-15% of the gait cycle at around the mid-stance phase. However, Figure C which illustrate the total joint loading shows that increased muscle co-contractions was associated with increased knee joint loading during the first period, with minimum change at the second peak (Brandon et al., 2014). This model clearly shows that alterations in muscle activation, characteristic of people with knee OA (shown in red below (C)), led to increases in contact force in the 10-15% of gait cycle, which corresponds to 15-25% of stance phase.

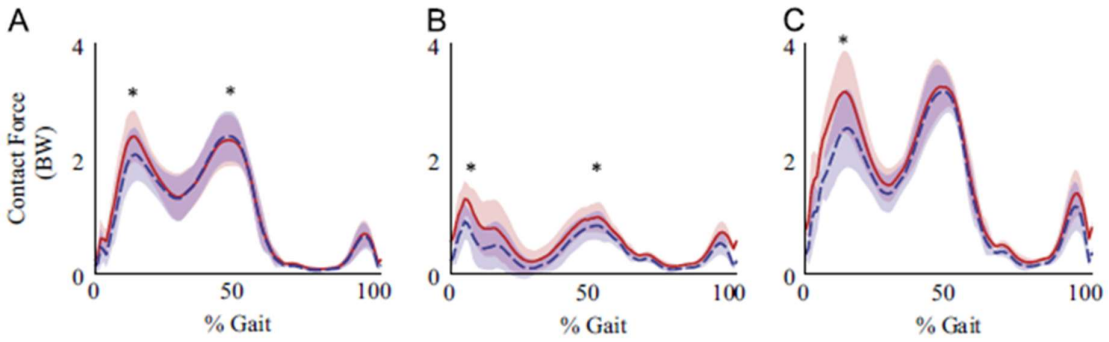


Figure 2-6 (A) medial, (B) lateral and ((C) total joint. The "Baseline" condition (blue, dashed), "OA-type" activation perturbation (red, solid) (Brandon et al., 2014)

Another musculoskeletal model study was used to understand the contribution of muscle and moment to knee joint force (Sritharan et al., 2016). This study aimed to measure the effect of lower limb muscle activation and co-contraction on the tibiofemoral joint in thirty-nine patients with knee OA, compared to fifteen healthy subjects during walking. Kinematic, kinetic and muscle activity were used as input to the model. The authors observed that patients with knee OA walked with higher hamstring and gastrocnemius muscle activity, but lower quadriceps muscle activity compared to healthy subjects at the first peak of the stance phase. In addition, this study showed that the increased duration of hamstring and gastrocnemius muscles contributed to the increased medial knee joint loading. This study clearly illustrate that increased higher hamstring and gastrocnemius in OA patients will lead

to an increase in the medial joint force: especially around the initial peak in loading (15-25% stance).

Both of above studies (Brandon et al., 2014; Sritharan et al., 2016) clearly show that higher in muscle activations will lead to increase knee joint force at the first peak of the stance phase approximately from 15-25% of the stance phase. Consequently, in the following sections different factors are discussed which could influence muscle activation during the early stance period.

2.7 Muscle co-contraction, disease progression and clinical pain

2.7.1 Co-contraction and disease progression

Evidence is beginning to emerge of the potentially damaging effect of increased co-contraction in people who have been diagnosed with knee OA. To date, there have been two longitudinal studies demonstrating a link between co-contraction and the rate of knee OA disease progression (Hodges et al., 2016a; Hubley-Kozey, Hatfield, et al., 2013). For example, in a recent study, Hodges et al. (2016b) investigated the relationship between knee muscle co-contraction (quantified as the duration of simultaneous hamstring-quadriceps co-contraction) and cartilage loss. Fifty patients with moderate knee OA were recruited in this study. Medial knee cartilage thickness and knee muscle activity were measured by MRI and EMG respectively at baseline and again after one year. Interestingly, they found increased co-contraction to be associated with the progression of knee OA. This study found that increased in medial muscle activity was correlated with medial cartilage volume loss in those with knee OA. Although the group size was relatively small, significant relationships were found. However, the results of this study cannot be generalized to all people with knee OA as only people with Kellgren Lawrence grade 2 and 3 were recruited. Nevertheless, these data provide motivation to understand the underlying cause of co-contraction.

In another study that published in a conference Hubley-Kozey, Hatfield, et al. (2013) investigated 50 patients with moderate medial knee OA at baseline and followed them for eight years. WOMAC scores were recorded and knee radiographs were graded using the Kellgren Lawrence grading scale and muscle co-contraction were measured at baseline and again after 7 year. At follow up (mean 7.8 years), 25 of these patients elected to go for total

knee replacement surgery because they were unable to manage their pain. Interestingly, the authors found that hamstring and quadriceps muscle activity were higher in this group at baseline. In addition, the authors showed that co-contraction between the hamstring and quadriceps muscles was associated with an increased likelihood of a knee replacement at 5 years (Hubley-Kozey, Hatfield, et al., 2013). This study demonstrated that increased muscle co-contraction in people with knee OA is associated with the decision to undergo knee replacement. Again, these finding motivate further research to understand the drivers for increase co-contraction.

The two studies discussed above provide compelling evidence that co-contraction in people with knee OA could increase joint loading and accelerate the progression, which may result in patients choosing to go for a knee joint replacement earlier. This could due to increased muscle co-contraction will increase the compressive loads at the knee joint. Therefore, more insight into the possible reasons for increased co-contraction is needed in order to develop biomechanical interventions which could slow the progression of the disease.

2.7.2 Co-contraction and clinical pain

The source of pain in people with knee OA is not well understood. Articular cartilage is devoid of blood vessels and neurologic innervation (Nordin & Frankel, 2001) and therefore nociceptive signals (pain) in knee OA is likely the result from damage to innervated tissues around the articular joint such as subchondral bone, synovium, ligaments and/or muscles (Hunter, McDougall, & Keefe, 2008). For example, increased co-contraction will increased the forces through the knee muscles and this could lead to pain and discomfort around the joint. Another possible source of pain in knee OA are bone marrow lesions (bone bruises), defined as regions of hyperintense marrow signal. Interestingly, these regions have been positively linked with pain in knee OA (Alliston, Hernandez, Findlay, Felson, & Kennedy, 2018; Felson et al., 2001; Yusuf, Kortekaas, Watt, Huizinga, & Kloppenburg, 2011). Evidence is beginning to emerge which suggests that altered joint loading could be linked to the bone marrow lesion and cartilage degeneration (Alliston et al., 2018; Felson et al., 2003). Given that people with knee OA exhibit higher co-contraction, it is possible that this places increased stress on the bones, and is implicated in the development of bone marrow lesions (Alliston et al., 2018), however further research is required to investigate this idea further. In summary, co-

contraction will increase the forces and stress on a range of anatomical structures around the knee joint, such as the muscles, bones and ligaments. Given that there are no pain receptors in the cartilage, it is likely that this increased stress plays a role in OA-related pain.

2.8 Biomechanical and neurological mechanisms underlying muscle co-contraction

Based on the findings described above, it is clear that increase co-contraction will lead to increase knee joint loading (Brandon et al., 2014; Sritharan et al., 2016). In addition, there is evidence that increased co-contraction will accelerate cartilage degeneration (Hodges et al., 2016a) and also increase the likelihood of progression to total knee replacement (Hubley-Kozey, Hatfield, et al., 2013). Therefore, studies are needed to clarify the possible cause of increasing co-contraction in those patients. Several theories on the possible reason of increasing co-contraction have been proposed. In the subsequent paragraphs, the biomechanical and neurological mechanism that could lead to increase muscle co-contraction are discussed in detail.

2.8.1 Current theories to explain biomechanical co-contraction in people with knee OA

A number of biomechanical explanations have been proposed for increased co-contraction in people with knee OA. For example, it is theorised that higher lateral muscle activation may arise in order to counteract the high medial joint loading often found in those with knee OA (Andriacchi, 1994). In addition, it has been suggested that high lateral co-contraction may control the knee adduction moment during walking (Lewek et al., 2004a). However, modelling studies suggest that such increased muscle co-contraction may increase the loading on the joint (Brandon et al., 2014; Sritharan et al., 2016), with imaging studies suggesting that increase co-contraction may accelerate cartilage loss (Hodges et al., 2016a). These studies demonstrate that increased muscle co-contraction will actually increase joint loading and negatively impact on articular joint. Therefore, the suggestion that lateral muscle activation may arise in order to counteract the high medial joint loading does not appear to be supported by scientific data.

Another possible explanation of higher muscle co-contraction is that increasing co-contraction may lead to increase joint stiffness which acts to counteract instability associated with joint space narrowing (Childs et al., 2004). This study determined a relationship between

co-contraction and knee stability in the OA population. Specifically, they reported that increased co-contraction was correlated with better knee stability (Lewek, Ramsey, Snyder-Mackler, & Rudolph, 2005). However, it is not clear whether their laboratory measures of stability translated in better real-world stability of the knee. Furthermore, the authors then concluded that higher co-contraction may lead to joint compression and therefore could exacerbate joint damage.

In line with the results obtained by Lewek et al. (2005), it has been observed that there is a significant increase in muscle co-contraction in people with deficiency of the anterior cruciate ligament (instable knee) compared to healthy people (Doorenbosch & Harlaar, 2003). Moreover, co-contraction around knee muscles was higher in knees with articular cartilage defects (Thoma et al., 2016). This gives further support to the assertions of (Steultjens, Dekker, & van der Esch, 2006) who suggest that increaased co-contraction is a necessary mechanism to provide stability to the damaged (and unstable) knee. In contrast to earlier findings, however, Schmitt and Rudolph (2008) explored the link between the knee joint stability in patients with knee OA and muscle co-contraction. Surprising, the result showed that the higher muscle co-contraction used by patients with unstable knee OA appeared to be an ineffective strategy to stabilize the knee joint (Schmitt & Rudolph, 2008). Therefore, it is not clear whether higher co-contraction functions to improve knee joint stability.

As explained earlier, increasing co-contraction around the knee joint in the OA population may lead to increased joint loading, damage of the articular cartilage and increased progression of the disease. This does not suggest a positive adaption to the disease. Although it has been suggested that increased co-contraction could lead to improved knee stability, the evidence is not unequivocal and such increased stability could be achieved by better coordinated activity of the knee muscles, without the need for co-contraction. Another possible explanation of higher muscle co-contraction in people with knee OA could be through neurological mechanisms. This idea is discussed below in details.

2.8.2 Possible neurological theory of co-contraction.

It is possible that co-contraction could result from some form of neurological deficit. Before the mechanisms underlying neurological co-contraction are discussed, it is necessary here to

cover some background material in neuroscience. To control movement, the central nervous system (CNS) uses information from many receptors which provide information on spinal cord and brain. One important set of receptors are muscle spindle receptors which are in the belly of skeletal muscle and which relay information to the CNS. It has been demonstrated that muscle spindle plays an important role in coordinating muscle contraction (contract agonist and relax the antagonist) (Burke, Hagbarth, Löfstedt, & Wallin, 1976; Gandevia, McCloskey, & Burke, 1992) and provides position of the limb to CNS (proprioception) (Mileusnic, Brown, Lan, & Loeb, 2006). In addition to muscle spindle, reciprocal inhibition plays an important role in controlling muscle contraction.

Reciprocal inhibition is a neuromuscular reflex that inhibits antagonist (opposite) muscle during movement. During agonist contraction, the role of reciprocal inhibition is to prevent the excitation of motor neuron of the antagonist muscle. These processes are controlled by the basal ganglia (connected to the cortex and thalamus) which generates the motor plan by selecting a desire muscle (agonist) and inhibiting the undesired muscle (antagonist) (Hallett, 1993). For example, when knee flexor muscles contract then knee extensor muscles will be inhibited. However, without these mechanisms the contraction of both muscles occurs simultaneously and due to increased excitation and decreased reciprocal inhibition. Through this mechanism, excessive co-contraction can occur (Hallett, 1993; Mink, 1996) in specific neurological condition which are discussed below.

Cerebral palsy (CP) is a condition that affects muscle control and movement caused by damage in the CNS. In patients with CP the normal pattern of neurons pathways from and to CNS are disturbed. It has been observed in patients with CP that some of the primary impairments are abnormal muscle tone and co-contraction (Rosenbaum et al., 2007).

The pathomechanics of this disease involve an involuntary co-contraction of agonist and antagonist muscles which are caused by dysfunction in the CNS (Kyllerman, 1982). Another example of neurological co-contraction is found in Parkinson's disease which is a neurological disease affecting the motor system of the CNS (Hughes, Daniel, Kilford, & Lees, 1992). It has been found that the pathophysiological on the basal ganglia, which plays an important role in organization the muscle co-contraction, has been associated with the movement impairment in patients with Parkinson's disease (Obeso et al., 2000). In these patients the reciprocal

inhibition is absent and this results in altered neuro-activation of the muscle (Mutch, Alberman, Hagberg, Kodama, & Perat, 1992; Obeso et al., 2000; Rosenbaum et al., 2007; van der Stouwe et al., 2015)

Reciprocal inhibition is reduced in some neurological diseases and this leads to a disturbance in the mechanisms of inhibition of the antagonist muscle resulting in increased the co-contraction. However, it is unlikely that people with knee OA have a neurological deficit. Moreover, the evidence for co-contraction as adaptive response to counteract instability is also weak and it is clear that increased muscle activity will increase stress on anatomical structures, increasing the rate of disease progression. It is therefore important to consider other mechanisms, not a direct result to the disease, which could underlie increased co-contraction. In the following section, a new model is proposed to explain increased co-contraction in knee OA.

2.9 A new model to explain increased co-contraction in people with knee OA

2.9.1 The biomechanical effect of increasing forward trunk lean

During walking, balance is required to keep the body upright by maintaining the body's centre of mass within the base of support (Prince, Winter, Stergiou, & Walt, 1994). Muscles and connective tissues produce internal moments which act in an opposite direction to external moments, which result from the GRF. For instance, when the GRF passes anterior to the hip joint, creating an external hip flexion moment, the hamstring muscle (hip extensor muscles) will contract to create an internal hip extensor moment. The external moment is determined by magnitude of the GRF and the perpendicular distance from the GRF vector to the joint centre. As a result, if the perpendicular distance from the GRF vector to the joint centre increases, it will lead to an increase in the moment.

Figure 2-7 illustrates that normally the GRF vector passes behind the knee joint in the loading response phase. However, if an anterior trunk bend (or forward lean is adopted), this brings the GRF vector in front of the knee joint. This change in the position and direction of the GRF will change the moments, and loading, at the hip, knee and ankle joints. These changes will include an increase in hip extensor moment and a decrease in knee extensor moments. Such change in joint moments will be accompanied by corresponding changes in muscle activation

patterns. This mechanism could result in changes in muscle activity, particular hamstring activity, and could explain some of the differences in muscle activation patterns previously observed in people with knee OA. Given the potential for changes in sagittal trunk angle to impact of moments and muscle activation patterns, it is important to review previous work which has examined biomechanical changes which are associated with alterations in trunk inclination. This research is discussed in depth below.

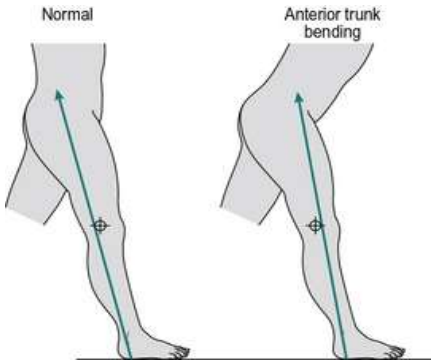


Figure 2-7 The GRF during standing and forward lean. Adopted from (Levine, Richards, Whittle, & Whittle, 2012).

2.9.2 Kinematic and spatiotemporal changes associated with increased trunk flexion

The first investigation of the effect of sagittal trunk flexion on lower limb kinematics in healthy people was performed by Saha, Gard, and Fatone (2008). This study aimed to understand the effect of three imposed of trunk flexion angles on lower limb kinematics. Fourteen healthy subjects were asked to walk in three different conditions: normal, $25^{o} \pm 7^{o}$ and $50^{o} \pm 7^{o}$ of trunk flexion at three different speeds: slow, normal and fast. In order to maintain the posture, biofeedback was used. They found that the average walking speed between various trunk flexion conditions were not significantly different, however, step length was significantly decreased during trunk flexion. In addition, the knee range of motion was higher when walking with trunk flexion compared to normal posture walking. Importantly, they found that increasing trunk flexion significantly increased the hip flexion angle. One limitation of this study is that the participants walked with large changes in trunk flexion. Therefore, although the study demonstrates that walking with trunk flexion is associated with changes in kinematics and spatiotemporal characteristics, it cannot be used to understand the potential impact of normal variation in trunk flexion in healthy people or those with knee OA.

In another study, Lewis and Sahrmann (2015) studied the effect of trunk flexion on lower limb kinematics in three different trunk postures (natural, swayback and forward flex) in fifteen healthy individuals. They observed that walking with a swayback posture resulted in increasing hip extension angle and ankle dorsiflexion compared to natural posture. In addition, walking with swayback posture increased knee extension angle compared to forward flexed posture. However, they reported that walking with forward flexed posture lead to decrease hip extension angle compared to the natural posture. In addition, knee extension and ankle planter flexion were decreased compared to natural posture during walking with forward flexed posture (Lewis & Sahrmann, 2015). Although interesting, this study was limited by a small sample size and no direct control of trunk angle e.g. biofeedback.

Taken together these two studies demonstrate that walking with altered upper body position produces corresponding changes in lower limb kinematics and spatiotemporal characteristics. However, these changes in kinematics do not provide insight into possible changes in lower limb moments or muscle activity change in response to increase in forward trunk lean. Moreover, these studies were carried out by instructing participants to walk with relatively large changes in trunk angle, therefore gives no indication of the magnitude of changes that would be observed with only small trunk flexion angles, more typical of normal variations in trunk lean under real-world conditions.

2.9.3 Kinetics changes associated with increased trunk flexion

Previous research has observed that walking with trunk flexion alters lower limb joint moments. Building on a previous study by Saha et al. (2008) another study investigated the effect of the three imposed trunk flexions on lower limb kinetics in healthy subjects (Kluger, Major, Fatone, & Gard, 2014). Data was collected from fourteen healthy subjects using the same procedure in the previous study (Saha et al., 2008) however, participants were instructed to walk at their self-selected speed with three different conditions: normal, $25^{o} \pm 7^{o}$ and $50^{o} \pm 7^{o}$ of trunk flexion. The results showed that the hip extensor moment and the ankle plantarflexor moment were significantly increased with trunk flexion. In addition, the results showed that the knee extensor moment was decreased but not significantly. This study indicates that there are significant changes in lower limb joint moments with significant

changes in knee and hip moments (Kluger et al., 2014). However, this study is limited by that the trunk angles not being representative of typical normal variation.

A similar study was performed by Lewis and Sahrmann (2015) who investigated the effect of three different trunk posture (natural, swayback and forward flex) on lower limb kinetics in fifteen healthy individuals. The results showed that walking in the swayback posture increased in both the hip flexor moment and also the knee extensor moment in comparison to natural position. In addition, walking in the swayback posture increased the ankle plantarflexion moment compared to forward posture. Moreover, result showed that walking in the forward flexed posture increased in the hip extensor moment and decreased in both the knee extensor moment and the ankle plantarflexor moment. Taken together these results show that changes in upper body positioning have the potential to alter lower limb joint moments. However, the trunk flexion angles were very big, therefore, this offers no insight into the effects of small changes in trunk inclination. In addition, this study was limited by not including EMG data and so provided no insight into the effect of upper body position on muscle activation patterns.

Average natural sagittal trunk inclination during walking varies among subjects. For example the overall range of sagittal trunk reported by Thorstensson, Nilsson, Carlson, and ZOMLEFER (1984) was between 1.5^{o} and 6^{o}, and in another study by Leteneur, Gillet, Sadeghi, Allard, and Barbier (2009) the average natural trunk inclination during walking was found to vary between -1.7^{o} and 2.9^{o}. Recently, researchers have studied the effect of normal variation in trunk inclination on lower limb moments during walking. For example, a study by Sato and Maitland (2008) investigated the relationship between participant's natural trunk inclination and lower limb joint moments during walking in thirteen elderly female subjects. Trunk angle was determined as the angle between a vertical line oriented at the midpoint and a neck-hip line. The results showed that walking with leaning forward lead to increase in the hip extensor moment, the knee extensor moment and the plantarflexor moment. In addition, findings revealed that there was a positive moderate correlation between the leaning forward and the lower limb joint moments during walking. Nevertheless, there was no significant differences observed (Sato & Maitland, 2008). This study indicates that walking with forward natural trunk lean may lead to change the lower limb moments.

In another study (Leteneur et al., 2009), recruited twenty five healthy subjects who were divided into two groups depending on their natural trunk angle: backward leaners with an average of -1.7° of trunk lean (12 subjects) and forward leaners with an average of 2.9° of trunk lean (13 subjects). Trunk angle was expressed as the angle between the line joining the midpoint between the greater trochanters and acromion process. Hip, knee and ankle moments were measured and compared between the two groups. The results demonstrate clear differences in hip moments (Figure 2-8) between the groups, with duration of hip extension moments being increased for the forward lean group. In addition, this study showed that in forward leaners there was a small decrease in the knee moment and a small increase in the ankle plantarflexor moment. However, this study did not include EMG, therefore further research is needed to understand the changes in muscle activity which are associated with small variations in natural trunk inclination.

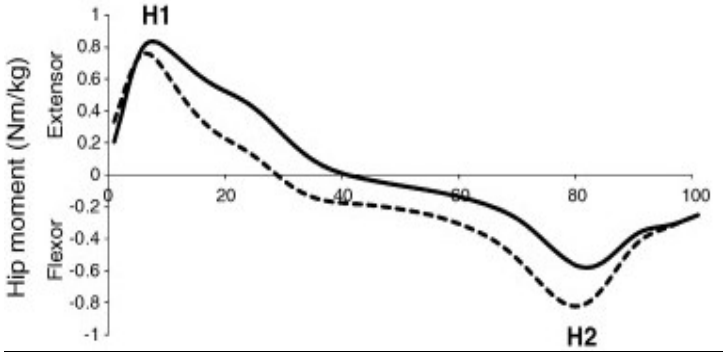


Figure 2-8 Hip moments during backword lean (dotted line) and forward lean (solid line) (Leteneur et al., 2009).

Although there has been some research investigating the effect of trunk inclination on lower limb biomechanics, there are still many unanswered questions. Firstly, angles of trunk inclination were very large in most previous studies (Kluger et al., 2014; Lewis & Sahrmann, 2015; Saha et al., 2008) and this gives no insight into kinetic/kinematic changes which could be associated with normal between-individual differences in trunk inclination. Interestingly, Leteneur et al. (2009) observed differences of between 5-10 degrees between healthy subjects and therefore further research is required to understand the biomechanical changes associated with this magnitude of trunk lean. Secondly, previous studies have not investigated the effect of incremental increases in trunk inclination. As a result, there is no data on the

precise relationship between changes in trunk inclination and changes in lower limb moments. Finally, previous studies did not include EMG data. Therefore, the effect of trunk lean of muscle activation patterns is not clear.

2.9.4 Muscle activity changes associated with increased trunk flexion

To date, there has been only one study investigating the changes of EMG in lower limb muscles during walking with and increased forward lean (Grasso, Zago, & Lacquaniti, 2000). In this study, the authors compared normal walking with two different trunk flexion postures: knee flexed walking and knee and trunk flexed walking. The findings showed that there was an increasing in the muscle activity in the lower limb muscles in walking with bent posture. However, this study has a number of limitations. Firstly, only five healthy participants were tested which is insufficient for statistically analysis. In addition, as the trunk flexion angles were very big, approximately 50 degrees the study offers no insight into the effects of small changes in trunk inclination. Another problem is that they did not study trunk flexion without knee flexion. Nevertheless, the findings suggest that muscle activity in walking with trunk flexion is greater than in normal walking. Given the potential for changes in sagittal trunk angle to impact on muscle activation patterns, this mechanism could result in changes in hamstring muscle activity and could explain some of the differences in muscle activation patterns previously observed in people with knee OA. Therefore, further research is required to more accurately understand the effect of smaller changes in trunk inclination on lower limb muscle activity.

2.9.5 Summary of previous biomechanical changes which are associated with forward trunk lean

In the section above, a summary was presented of previous research which has investigated the effect of trunk flexion on lower limb biomechanics. These studies have identified that variations in trunk inclination in healthy people can affect both muscle activation and also lower limb joint moments. However, further research is required to fully understand the precise effects of increasing trunk lean in healthy people and to determine whether the observed differences between people with knee OA are the result of the disease or can be attributed to altered trunk angle. This next section of thesis outlines previous research that

has sought to investigate differences in trunk inclination between people with knee OA and healthy control subjects.

2.10 Trunk inclination in people with knee OA

Recent evidence found that people with knee OA stand with forward inclination of the trunk (Turcot, Sagawa, Hoffmeyer, Suva, & Armand, 2015). Eighty-seven people with knee OA and twenty-five healthy subjects were recruited and their joint positions while standing position were compared. The findings revealed that people with knee OA (solid line) stand with a more flexed posture in all joints compared to healthy control subjects (dotted line), as shown in Figure 2-9. Furthermore, the results demonstrated that, in OA patients, the trunk was flexed by approximately 2.9° more than the healthy control subjects when standing (Turcot et al., 2015). This study shows that people with knee OA stand with a forward trunk inclination. However, these authors did not study walking and therefore, it is not clear if this posture is maintained when walking.

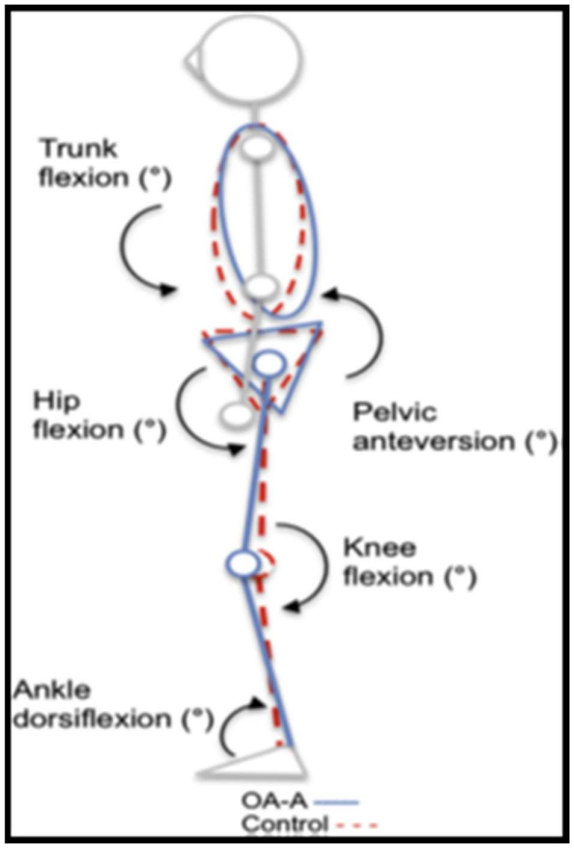


Figure 2-9 Joint positions during standing for OA people (solid line) and healthy subjects (dotted line) (Turcot et al., 2015).

A recent study found that people with knee OA walk with a more flexed trunk. This study Preece et al. (2018) compared the trunk posture of 27 OA patients against that of 19 matched (age and BMI) healthy participants when walking. The result showed that people with knee OA walk with 2.5°- 3º more trunk lean than healthy subjects. It is interesting to note that the magnitude of the change in trunk posture during walking found by (Preece et al., 2018) was similar in magnitude to those observed during standing by (Turcot et al., 2015). Taken together, these two studies demonstrate that individuals with knee OA walk and stand with more trunk flexion when compared to matched healthy subjects.

The trunk accounts for approximately over 50 percent of body mass (Winter, 1990). Thus, small changes in sagittal plane alignment could lead to corresponding changes in hip moments. Although there is no data on the precise relationship between trunk inclination and hip moment, it is interesting to establish if previous research has shown differences in hip moments between people with knee OA and healthy controls. Liu et al. (2014) observed that there was an increase in hip extensor moment at the early mid stance in people with knee OA compared to healthy people during walking, as shown in Figure 2-10. Interestingly, a similar increase in hip moments was observed in healthy subjects who habitually walk with an increased forward inclination of the trunk, as shown in Figure 2-8 (section 2.9.3) above (Leteneur et al., 2009).

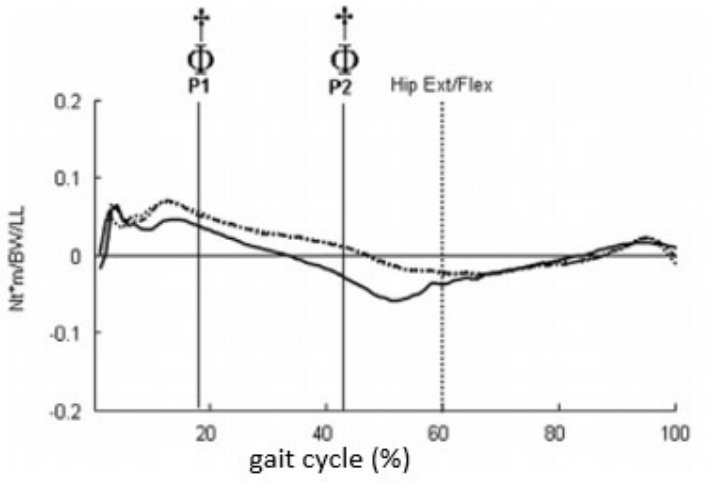


Figure 2-10 Sagittal hip moment for OA people (dotted line) and healthy subjects (solid line) (Liu et al., 2014).

Previous studies on knee OA patients have demonstrated an increase in the hip extensor moment (Liu et al., 2014) which is likely to be linked to increased hamstring muscle activity in patients with knee OA and therefore possible muscle co-contraction. In addition, given that people with knee OA walk (Preece et al., 2018) and stand (Turcot et al., 2015) with an increased trunk inclination, it is possible that the altered muscle activation and joint moments observed in people with knee OA could be the result of altered upper body position and may not just be a localised muscular response. However, to further explore this idea, more research is needed to understand the effect of changing trunk inclination in both healthy people and those with knee OA.

Previous research supports the idea that people with knee OA stand (Turcot et al., 2015) and walk (Preece et al., 2018) with increased trunk lean. It is therefore important to understand potential the biomechanical/physiological drivers for this alteration in upper body position. One idea, explored in this thesis, in that shortened hip flexors muscles may lead to an anterior tilt of the pelvis and a corresponding increase in forward trunk lean. However, to date, there has been minimal research investigating the link between hip flexor muscle length and gait biomechanics in either healthy people or patients with knee OA. Therefore, in the final section of the thesis, ideas are explored which may provide some explanation for the observed differences in forward lean between healthy people and people with knee OA. This is discussed in more detail below.

2.11 Link between muscle shortening and forward lean

Posture is the alignment and maintenance of body segments in certain positions, during standing or sitting. It is widely accepted that standing posture is determined by resting muscle length and the active contraction of muscle. When extended past their resting length, muscles exert a passive tension, which is influenced by the structural properties of the muscle and the surrounding fascia. In static postures, and to some degree during dynamic movement, this passive tension means that the relative position between adjacent body segments is determined by resting muscle length (Czaprowski, Stoliński, Tyrakowski, Kozinoga, & Kotwicki, 2018; Sahrmann, 2002).

Muscle imbalance can occurs if agonist and antagonist muscle lengths change relative to each other (Axelson & Hagbarth, 2001b). An example of this is illustrated in Figure 2-11 , which shows the relationship between the quadriceps/hip flexors and hamstring muscles. In the picture on the left, this relationship is balanced correctly, and the pelvis sits in its neutral position. However, in the picture on the right, it can be seen that the hip flexor muscle has shortened, which has resulted in a relative anterior rotation of the pelvis. Overall, balance of hip flexor and extensor muscles may play an important role in relation to the pelvic tilt in the sagittal plane. Thus, shortening of the hip flexor muscle may affect the sagittal plane orientation of the pelvis on the hip joint and this may lead to changes in the inclination of the trunk or the lumbar lordosis (Axelson & Hagbarth, 2001b).

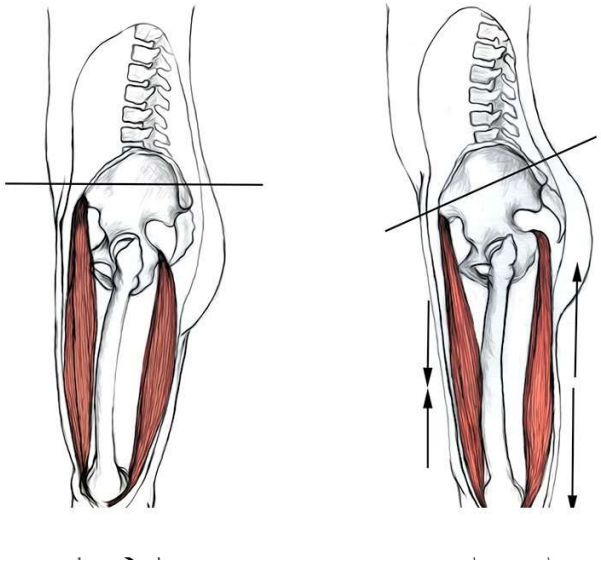


Figure 2-11 Illustrate shows the relationship between the quadriceps/hip flexors and hamstring muscles.

Research has shown that if muscles are frequently maintained at a length that is either shorter or longer than their normal anatomical position, they can adapt to this position (Gossman, Sahrmann, & Rose, 1982; Williams & Goldspink, 1978). Shortening of muscle can be caused by several different factors, related to physiological changes within the muscle. The basic function unit of muscle is the sarcomere which contains the contractile proteins actin and myosin (Lee & Schmid-Schönbein, 1995). The number of sarcomeres in series plays a key role in determining resting muscle length (Williams & Goldspink, 1973, 1978; Ylinen, 2008). Research has shown that when muscles are immobilised at a shorter length, there can be a

reduction in the number of sarcomeres in series (Proske, Morgan, & Gregory, 1993; Williams, 1990; Williams & Goldspink, 1973, 1978) .

Another possible mechanism that may lead to muscle shortening is thixotropy (Knutson & Owens Jr, 2003; Proske & Morgan, 1999). The term thixotropy is a phenomenon in which cross bridges, between actin and myosin, form when a muscle is maintained in a shorten position. This can lead to increased muscle tone (resistance to stretch). This idea has been demonstrated on isolated muscle fibers, for which It has been showed that muscle shortening occurs when the muscle fiber is maintained in a shortened state by immobilization or sustained muscle activity (Proske et al., 1993). At present, it is not clear why these cross bridges (associated with thixotropy form) but, under normal condition, the cross bridges tend to break up on movement or following a passive muscle stretch. However, if a muscle is held in a shortened position for a prolonged time, it is possible that thixotropy leads to structural changes within the muscle which may lead to longer-term muscle shortening.

Given the two mechanisms outlined above, there exits the potential for the hip flexor muscles to become shortened if they are maintained in a short position for prolonged periods of time. Most individuals spend a large proportion of their time sitting (Lee et al., 2015; Sliepen, Mauricio, Lipperts, Grimm, & Rosenbaum, 2018; Wallis, Webster, Levinger, & Taylor, 2013) in which the hip flexor muscles are in a shortened position (Shimada, 1996b). Thus, shortening of hip flexor muscles could result from prolonged sitting.

Through the physiological mechanisms outlined above, shortening of the hip flexor muscles may occur and may influence sagittal plane trunk orientation and/or lumbar lordosis. This idea was investigated in a study by (Kagaya, Ito, Iwami, Obinata, & Shimada, 2003) who simulated human walking with hip flexor contracture (muscle shortening), using computer software. The computer software was used to understand how muscle shortening affected the walking pattern and showed that the trunk tilted forward in proportional to the degree of hip flexion shortening. These results demonstrate the potential impact of hip flexor muscle shortening on the trunk and support the idea that a short hip flexor could create a corresponding anterior shift in the centre of mass relative to the hip joint (Perry, 2010) (Figure 2-12).

There have been only a small number of studies that have examined the effect of short hip flexor muscles on posture and gait patterns. The main study, carried out by Shimada (1996b), aimed to understand how hip flexion contracture (muscle shortening) can lead to alterations in posture and gait patterns in patients with neurological and musculoskeletal disorders during a supine position, or when sitting and standing. They used the Thomas test to determine the degree of muscle shortening and 12 survey demographic variables, for example age, sex, severity of contracture and knee flexion to clarify its contribution to the appearance of gait patterns thought to indicate hip flexion contracture. This study showed that patients with hip flexor contracture were observed to have postures and gait abnormalities, for example exaggerated lumber lordosis or/and trunk forward lean. These data clearly demonstrate the potential for short hip flexors to be associated with alterations in postural alignment, such as sagittal trunk inclination. However, detailed investigations are still needed to examine the association between hip flexor muscle length and trunk inclination during standing and walking, in people with knee OA.

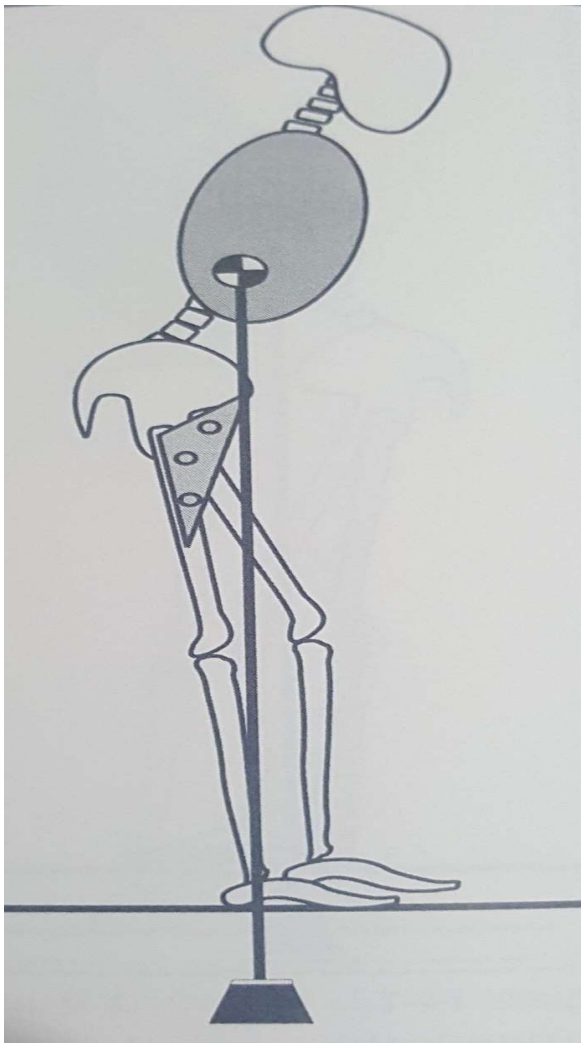


Figure 2-12 Hip flexor muscle shortening lead to lean the trunk forward. Adopted from (Perry & Davids, 1992).

Previous research has shown that people with knee OA walk (Preece et al., 2018) and stand (Turcot et al., 2015) with increased sagittal plane trunk inclination. As explained in previous section, this change in trunk position could be a driver for altered muscle activity in people with knee OA and potentially play a role is disease progression. There is currently minimal research which has investigated the potential link between hip flexor length and sagittal plane trunk inclination, either healthy people of in people with knee OA. Therefore, the final aim of this thesis is to understand the relationship between hip flexor muscle length and trunk inclination (during walking and standing) in both healthy people and individuals with knee OA.

2.12 Overview of the thesis

Recent research has demonstrated that people with knee OA walk with approximately 3^{o} more in sagittal plane inclination of the trunk compared to matched healthy people (Preece et al., 2018). This increased flexion may shift the position of the centre of mass relative to the hip, knee and ankle joint, which could in turn lead to changes in EMG patterns during walking. Interestingly, there has been a large amount of previous research which has demonstrated changes in both lower limb joint moments and muscle activation patterns in people with knee OA. For example, studies have observed decreases in the knee extensor moment and increased in hip extensor moments in patients with knee OA. Studies have also consistently demonstrated increases in hamstrings and changes in quadriceps activity in people with knee OA as well as increased muscle co-contraction during early stance. It has been suggested that such altered muscle patterns may increase the loading on the joint and accelerate cartilage loss. Interestingly, data from healthy subjects suggests a possible link between sagittal plane trunk inclination and lower limb joint moments/muscle patterns. However, further research is fully required to fully explore this concept.

This thesis proposes a new model to understand some of the previously observed biomechanical differences between people with knee OA and heathy subjects. Specifically, ideas are explored around the potential for sagittal plane trunk inclination to be a driver for changes in lower limb moments and lower limb muscle activation patterns during walking. These ideas are explored through a number of different research studies. Firstly, gait characteristics are compared between asymptomatic groups (of young and older adults) and people with knee OA in order to facilitate comparison with previous research (Chapter 4). Secondly, studies were performed to understand the effect (on lower limb kinetics/kinematics and muscles activation) of instructing participants to walk with increased trunk inclination. This investigation was performed in two phases, first in young healthy participants (Chapter 5) with three imposed trunk angles (normal walking +5^{o}, +10^{o} and -5^{o}) and then in older adults and people with knee OA for two imposed trunk angles (normal walking +5^{o} and -5^{o}). The +10^{o} condition was excluded for the older/knee OA as these participants were unable to following this instruction.

In the next study (chapter 7), differences in moments and muscle patterns were explored between groups of healthy subjects who habitually walk with different trunk inclination angles. In the final study (chapter 8), it was aimed to explore the biomechanical mechanisms which relate to forward lean and specifically to understand if hip flexor length has the potential to influence sagittal trunk inclination. A secondary aim of this study was also to understand whether could also be a direct link between hip flexor length and muscle activation patterns during walking.

A summary of the key questions that this doctoral thesis seeks to address are given below:

- What are the key lower limb biomechanical differences between people with knee OA and healthy participants during walking? (**Chapter four**)

- What is the biomechanical effect of instructing young healthy people to walk with increased/decreased trunk inclination? (**Chapter five**)

- What is the biomechanical effect of instructing older healthy people and individuals with knee OA to walk with increased/decreased trunk inclination? (**Chapter six**)

- How do interindividual variations in habitual trunk inclination during walking affect joint moments and muscle activation in healthy people? (**Chapter seven**)

- What is the relationship between hip flexor muscle length and trunk inclination in healthy people and individuals with knee OA? (**Chapter eight**).

Chapter Three

Method

3.1 Overview of data collection procedures

This chapter describes the methods used in this thesis. As explained above, this doctoral thesis sought to explore the potential influence of trunk inclination on lower limb biomechanics through a number of studies (listed above). Three groups: the knee OA subjects (n=20), older healthy subjects (n=20) and young healthy subjects (n=20) across both genders were recruited. All experimental data was collected via a single testing session on each individual participant during which a range of biomechanical data were collected. During data collection subjects were required to walk normally then in three additional trunk flexion conditions: normal -5º, normal +5 º and normal +10 º flexion (note this final condition was only included for the younger group). This provided data on habitual (natural) and imposed trunk lean. In addition, each participant underwent hip flexor muscle length testing.

3.2 Ethical approval and participants

3.2.1 Ethical approval

This study was approved by the Research, Innovation and Academic Engagement Ethical Approval Panel at University of Salford (reference no HSR1617-98) (Appendix I). In addition, this study was approved by the NHS health research authority of the East Midlands, the Derby Research Ethical Committee (reference no IRAS 235079) (Appendix II).

3.2.2 Inclusion for OA participants

The following inclusion criteria were used to define eligibility for people with knee OA:

1. Age range of 40-75 (upper age limit due to the amount of walking involved in the study).

2. Ability to stand and walk independently.

3. Ability to speak and understand English to read the participants information sheet (PIS) (Appendix V) and sign the consent form.

4. Ability to walk without any assistance for at least 100 m (this 100 m is not representative of the real demand of the testing, but it ensures that they can complete the full testing protocol, as if they are unable to walk for 100m they may be fatigued by the protocol and this may affect the results of the study).

5. Knee pain for at least 6 months.

6. Clinical diagnosis of knee OA according to the American College of Rheumatology (ACR) (Altman et al., 1986). People with knee OA (Medial or lateral) were included if they had a clinical diagnosis of knee OA, using the ACR criteria. This is given as clinical presentation of pain in the knee and at least 3 of the following (Altman et al., 1986):

    I. Age 40-75 years.
    II. Stiffness < 30 minutes per day
    III. Crepitus
    IV. Bony Tenderness
    V. Bony enlargement
    VI. Palpable warmth

7. Consent for researcher to access their knee x-ray data.

8. Body mass index (BMI) <35 since as it is not possible to obtain EMG muscle measurements on individuals with excess adipose tissue.

9. All subjects had a confirmed radiological diagnosis of knee OA.

3.2.3 Inclusion for healthy participants

Young healthy participants were aged between 18 and 40 years and older healthy subjects were aged between 40 and 75 years. The following are the inclusion criteria for healthy subjects:

1. Ability to stand and walk independently.

2. Ability to speak and understand English to read the PIS and sign the consent form.

3. Ability to walk without any assistance for at least 100 m.

4. No diagnosis of knee OA.

5. Body mass index (BMI) <35 since as it is not possible to obtain EMG muscle measurements on individuals with excess adipose tissue.

3.2.4 Exclusion criteria

Both the OA and healthy participants were excluded if they had any of the following:

1. Complex pain conditions such as diabetic neuropathic pain or fibromyalgia

2. Had undergone previous surgery to the lower limbs.

3. BMI >35.

4. Lower limb arthroplasty.

5. Any systemic inflammatory disorders, for instance rheumatoid arthritis.

6. Any balance disorders which may lead to fall.

7. Concurrent low back pain (any low back pain within the last 6 months).

8. Patellofemoral OA (for people with knee OA).

3.3 Participant recruitment

OA and healthy individuals were recruited through a number of avenues, listed below:

1. Community posters. Posters were placed around the university campus and in the local community.

2. Email invitations. Emails were sent to staff and students at the University of Salford.

3. Through local GP practices. Participants with knee OA were recruited from local GP practices (within close proximity of the University of Salford). With this approach, a member of the patient's direct healthcare team performed a search of electronic patient records to identify potentially eligible participants. The care team then sent a copy of the PIS and a letter of invitation.

4. Through physiotherapy and musculoskeletal pain outpatient clinics. Patients with knee OA were recruited from outpatient clinics within close proximity of the university. With this approach, physiotherapists/clinicians asked their patients if they were interested in participating in a research study at the university. If interested, patients were given a copy of the PIS along with a letter of invitation.

5. Via poster advert at physiotherapy outpatient clinics and local GP practices. Posters were placed in local GP practices and physiotherapy/musculoskeletal outpatient clinics. Patients who were interested in participating in our knee research were required to text KNEE to a specific number (e.g. 60006) or to send an email to our university generic volunteering email address (e.g. healthcare-volunteering@salford.ac.uk). Note anyone who used the text message service would have their mobile number forwarded to this email account. Potential participants who contacted the university via this route were contacted by the manager of our volunteer database, who sent them the PIS and passed their contact details to the lead researcher. The database manager asked them if they would like to join our volunteer database and if they agreed, they were sent the appropriate documentation.

6. Citizen Scientist. This project is a collaborative venture between GM universities, Salford city council and Salford NHS trust for recruiting participants for research studies. The project has a website (http://www.citizenscientist.org.uk/), used to list all on-going studies.

7. Through the local Rotary Club and the University of the Third Age. Some control participants were recruited through local groups where a significant proportion of the members were over the age of 60. We had identified the University of the Third Age (http://www.u3a.org.uk/) and the Rotary Club http://www.rotary.org/en/Pages/ridefault.aspx. We contacted the leaders of 3-4 local groups and asked them to distribute an email along with a PIS around their group.

With the proposed avenues for recruitment listed above, individuals who were interested in participating in the research were required to contact the researcher directly (or were contacted individually, following an email/text to the university). On making contact with the

researcher, the participants were asked a number of questions to ensure that they met the inclusion/exclusion criteria. Those individuals deemed eligible were then sent the PIS. They were allowed 2-3 days to read this sheet and then they were contacted again. On second contact, the researcher checked that they were happy with the experimental procedure and they made an appointment for them to visit the university laboratory. A minimum period of 24 hours was set between providing the information sheet and determining their decision to take part in the study.

3.4 Sample size calculation

A sample size calculation for each study was performed, using the G-power software, to estimate the number of participants required to answer each separate research question. These calculations are explained below for each study:

3.4.1 Chapter four (Study one)

The primary outcome in this study was trunk inclination during walking and based on previous study by Preece et al. (2018) which examined the sagittal trunk inclination between people with knee OA and healthy control during walking. Their data showed mean (SD) trunk angle for people with knee OA was 4.6^{o} (2.9 ^{o}) and for healthy control 1.6 ^{o} (3 ^{o}). With $\alpha = 0.05$ and a power of 0.8 based on using a two-tailed test, the required minimum sample size for the study is n=13 in each separate group. Thus, 20 participants on each group were recruited.

3.4.2 Chapter five and six (Study two and three)

A previous study by Kluger et al. (2014) investigated the effect of instructing individuals to increase trunk flexion (from 0^{o} to 25^{o}) during walking. Their data showed mean (SD) hip flexor moments for 0^{o} of 11.5 (3.7) N.m/kg and for 25^{o} of 20.4 (7.8) N.m/kg and the effect size was 1.5. With $\alpha = 0.05$ and a power of 0.8 based on using a two-tailed test, the required minimum sample size for this study is n=9 in each group. As the studies in this thesis aimed to understand the effect of small trunk angles (-5^{o}, 5^{o} and 10^{o}), smaller effect sizes were anticipated. Therefore, number of participants was adjusted to 20 in each group.

3.4.3 Chapter seven (Study four)

This primary objective of this study was to investigate biomechanical differences between two groups which were defined by their habitual trunk lean angle (forward leaners and backward leaners). This study was powered based on the outcome hip flexor moment as not previous EMG data available. In a previous study by Leteneur et al. (2009) that shown the differ between a forward leaners group = 39.1% (13.5%) and a backward leaners group = 27.2% (7 %). The G-power software showed that, with an $\alpha = 0.05$ and a power of 0.8 based on using a two-tailed test, the required minimum sample size is 11 participants in each group. Therefore, 14 and 15 participants were included in each group.

3.4.4 Chapter eight (Study five)

No previous studies have been investigating the relationship between the hip flexor muscle length and trunk inclination in walking and standing. Therefore the sample size was based on a correlation of r=0.5, which represents a moderate correlation. With an alpha of 0.5 and a power size of 0.8, the G-power software estimated a sample size of n= 11. Therefore, 20 participants were recruited in each group.

3.5 Overview of data collection procedures

The participants made one visit to the Podiatry Laboratory at the University of Salford. Once the participants had arrived, they had to reread the PIS, sign the consent form to take part in the study and fill in the health history questionnaire (Appendix VII). In addition, individuals with knee OA had to complete the WOMAC clinical questionnaire (Appendix VI) and sign the data access form (to give permission for the researcher to access the medical record of the x-ray of the knee).

During this visit, the participants underwent the hip flexor muscle test (HFMT) and then had a full biomechanical assessment: kinematic, kinetic and EMG data were gathered during normal walking and walking in three (or two if they were unable to complete the third) different trunk flexion positions: normal -5 degrees, normal +5 degrees and normal +10 degrees. Finally, they performed the reference EMG testing. The participants were instructed to change into their shorts and vest during the assessment. Also, they were instructed to walk

barefoot. Data were collected from the most painful limb in the individuals with knee OA and a matched limb in the healthy older control subjects. The following paragraphs will discuss in detail the data collection procedures in the laboratory, as shown in Figure 3-1.

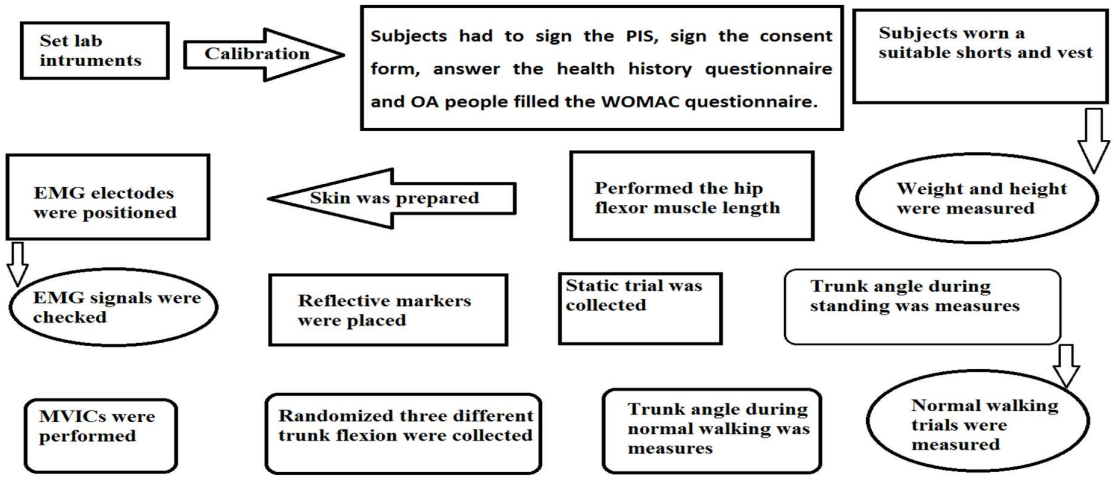


Figure 3-1 Lab protocol steps.

3.5.1 Consent

Upon arrival at the Podiatry Laboratory, the study was explained in full and if the participants had no objections, they completed a consent form. OA individuals were also required to complete the data access form, which was used to provide consent for the researchers to view any previous knee x-rays. These data were reviewed by a qualified radiographer who provided KL grades, see **Error! Reference source not found.** which shows the K-L grading for people with knee OA.

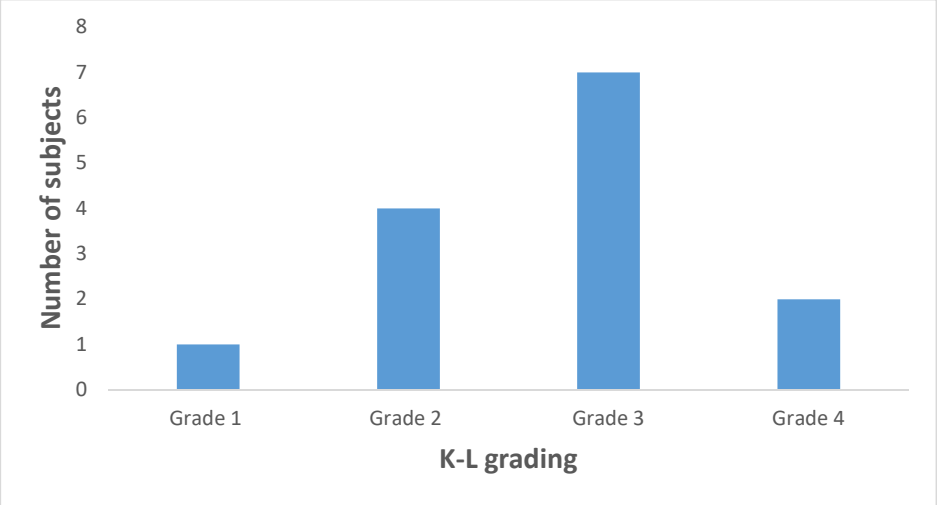


Figure 3-2 The K-L grading for people with knee OA.

3.5.2 Clinical outcomes

Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) is a clinical questionnaire rated on five points in a Likert scale. Before biomechanical data was collected, the participants (with knee OA) filled out the WOMAC questionnaire, which was used to capture pain (5 questions), stiffness (2 questions) and function (17 questions). It is the most commonly used questionnaire for knee OA in previous studies (Bellamy, Buchanan, Goldsmith, Campbell, & Stitt, 1988). The WOMAC questionnaire scores highly for validity, responsiveness and reliability (Chesworth, Mahomed, Bourne, & Davis, 2008; Escobar et al., 2007; Jinks, Jordan, & Croft, 2002; Ryser, Wright, Aeschlimann, Mariacher-Gehler, & Stucki, 1999; Salaffi, Carotti, & Grassi, 2005). In addition, all of the participants completed the health history questionnaire, which was used to help the researchers understand the medical history of the participants. Following this, measurements of height and body mass were then taken and hip flexor muscle length was assessed.

3.5.3 Hip flexor muscle test (HFMT)

The Thomas test is used to measure hip flexor (iliopsoas, rectus femoris and adductor longus) length. The hip flexor muscle originates from lumber vertebrae and/or iliac spine and inserts at the lesser trochanter of the femur and the base of the patella. Thus, in a lying position, a short hip flexor will limit the ability to extend the hip, thereby limiting hip flexion-extension

range of motion. This limitation capture by the Thomas test which provides an easy-to-implement methods for assessing the degree of hip extension on a test plinth. Clearly, this test is not a direct measure of hip flexor length, however, it is widely accepted (Clapis, Davis, & Davis, 2008; Moreside & McGill, 2011)and used in both clinical practice and research. It was therefore chosen to assess hip flexor length for this study.

In order to investigate a possible relationship between hip flexor muscle lengthen and trunk inclination (chapter eight), the HFMT was used (Figure 3-3). For this test, the participant was instructed to lie in a supine position, allowing ischial tuberosity over the edge of the table. The test was performed by asking the subjects to lie backwards on the bed while holding both knees to their chest with their hands. Pelvic tilt was controlled by using a pressure biofeedback device placed under the 'lumbar spine' inflated to 100 mmHg. At that time, the participant's leg was extended passively and slowly. The participant had to keep the pressure at 60 mmHg by slightly moving (flexing or extending) the non-testing leg, a technique used in previous studies (Moreside & McGill, 2011). If the participants felt any restriction in hip extension because of the table, they had to repeat the test procedure. When the participant's leg was fully extended, the angle of the thigh was measured by positioning a ruler on the line between the lateral aspect of the greater trochanteric and lateral femoral epicondyle of the knee joint. Then an Angle Pro (I phone App) was used to measure the thigh angle. This test was performed three times on both sides. If OA participants found this position uncomfortable, they were instructed to place their hands underneath their knees. If this was still uncomfortable, this test was omitted. In order to ensure that the hip flexor muscle length was performed correctly in all the participants, test-retest reliability was obtained (see 3.5.3.1).

Figure 3-3 Hip flexor muscle length procedure.

3.5.3.1 Test-retest reliability of hip flexor muscle test

Aim: This study aimed to examine the test-retest reliability of the HFMT in healthy subjects between days. This is to ensure the quality of this test and the consistency of results separated by one week.

Participants and method: 10 healthy subjects (7 male, 3 female) visited the lab twice with one week between each visit. The participants were recruited from Salford University. Participant inclusion and exclusion criteria in section 3.1.3 and 3.1.4 above were adopted in this study. Intra-class correlation coefficients (ICC) (Model: Two-way mixed effect and type: absolute agreement) (Koo & Li, 2016) were run to determine the test-retest reliability of HFMT between days. Participants underwent the HFMT as described in section 3.5.3 above. The second session (following the first session by an interval of 5-8 days) was performed following an identical protocol.

Results: The mean age, body mass and height (mean ± standard deviation) of the subjects were 25 ± 5.5 years, 63.3 ± 6.1 Kg and 169.8 ± 5.2 cm, respectively. Our result showed an ICC= .97 of the HFMT between days. Standard error of measurement (SEM) was calculated by the following equation:

SEM= pooled SD * √ (1- ICC). As the pooled SD = √ $(SD_1)^2 + (SD_2)^2 / 2$ (Cohen, 1988; Denegar & Ball, 1993). The SEM of the HFMT was 1.16°.

Discussion:

The aims of the current study were to examine the reliability of a hip flexor muscle length test (modified Thomas test) in healthy subjects between days. The rational for this study was to make sure that this test is reliable for measurements repeated by one week. The result showed an excellent (ICC = .97) reliability between days, providing confidence in the measurements for the studies in this thesis. This result is supported by several studies that have also showed this test to have good to excellent reliability (Cejudo, de Baranda, Ayala, & Santonja, 2015; Harvey, 1998; Kim & Ha, 2015; Peeler & Anderson, 2008; Young, Clothier, Otago, Bruce, & Liddell, 2003). Other evidence also demonstrates that the modified Thomas test is a reliable method for measure hip flexor muscle length (Kendall, Kendall, & Wadsworth, 1973; Vigotsky et al., 2016). Given the findings of this study and previous research, the modified Thomas test appears to be an appropriate method for quantifying reliability of the length of the hip flexor muscles between days.

3.5.4 Background to EMG measurement

This thesis aimed to understand the effect of trunk lean on lower limb moments and muscle activation. In order to investigate this idea, electromyography (EMG) was used. Before this technique is discussed in detail, some background information on muscle physiology will be presented along with an explanation of an EMG signal. The function unit of the neuromuscular system is called a muscle unit. A motor unit is composed of an alpha-motor neuron which contains the cell body, neuromuscular junction and muscle fibre. Each muscle fibre contains one motor end plate (neuromuscular junction) (O'Sullivan et al., 2013) (Figure 3-4). Action potential is an electrical impulse that sends signals across the neuron membrane. Once activated by the nervous system, the motor unit transmits the motor unit action potential

(MUAP), which is the summation of the electrical activity of all the muscle fibre, to the axons. When this reaches a synaptic end bulb, it activates a sequence of electrochemical to release the neurotransmitter-acetylcholine (Ach). Then the Ach receptors lead to an influx of sodium ions, creating a membrane depolarisation around the end plate and the negative membrane potential changes into positive.

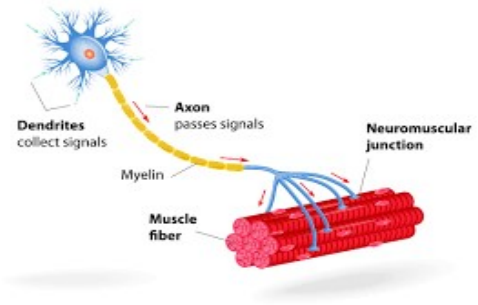


Figure 3-4 Motor unit of muscle

Following this, the potassium gate opens to permit the influx of potassium ions, creating repolarisation. In the resting status, the ionic balance is maintained by an active sodium-potassium pump (Figure 3-5). Thereafter, an action potential is propagated along the muscle fiber and calcium ions are released, which will be attached to the actin myofilaments (contractile elements of skeletal muscle) resulting in contractions of the muscle. Therefore, the EMG signal has the ability to detect the volume of the activity of all active motor units and these signals consist of the amplitude of negative and positive components, which reflects the intensity of the muscle contraction. EMG signals are the electrical summation of active motor units. Two types of methods are used to detect these signals, and these are discussed in the following paragraph.

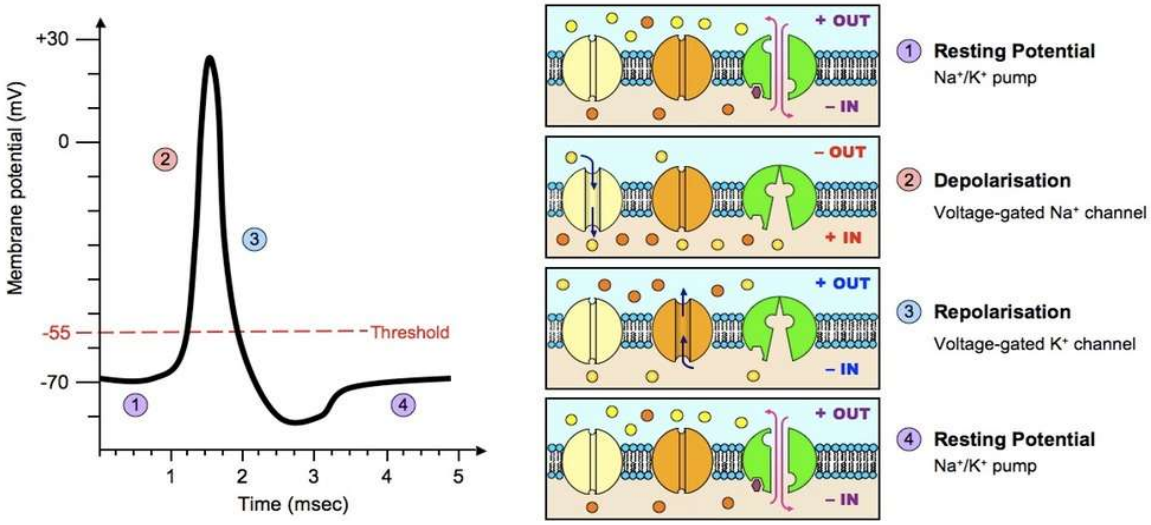


Figure 3-5 Generation an action potential across the neuro membrane. Adopted from http://www.vce.bioninja.com.au

Two types of EMG can be used to detect the signal from muscle: surface electromyography (sEMG) and fine wire electromyography. Surface EMG is one of the common methods used to measure the electrical activity of muscle in gait analysis – it is safe, easy and non-invasive (Kleissen, Buurke, Harlaar, & Zilvold, 1998). However, sEMG is sensitive to extrinsic and intrinsic factors such as tissue type, thickness (Farina & Mesin, 2005), physiological changes, temperature, physiological cross talk (Winter, Fuglevand, & Archer, 1994b), and changes in the geometry between muscle and the electrode site (Delaney, Worsley, Warner, Taylor, & Stokes, 2010). External noise is considered to be an external factor that negatively affects the quality of sEMG and may lead to changes in its shape and characteristics. In contrast to sEMG, fine wire techniques use needle electrodes that are inserted directly into muscles. However, these methods can be uncomfortable and painful (Perry, 2010). Fine wire captures only the localised motor unit not the whole muscle and also the pain from this technique may affect the muscle activation. Although there are some limitations of sEMG, the majority of kinesiological studies have been carried out using sEMG because it is non-invasive, causes minimum discomfort (Jacobson, Gabel, & Brand, 1995) and also provides an overall representation of muscle activity (Soderberg & Knutson, 2000). Therefore, in this thesis sEMG was used to understand muscle activation when walking with different trunk inclination.

3.5.5 EMG equipment setup

Surface EMG was used to measure muscle activity by using a Noraxon Desktop Direct Transmission System (DTS) (Noraxon USA Inc., model 586 Tele Myo DTS Desk Receiver) (www.noraxon.com) at a sampling rate of 1500Hz. DTS receivers can accommodate eight channels of EMG sensors. Six sensors were used in this study, and this system can be integrated with Qualisys at the same time during data collection. The DTS system is provided with sensors (model 542), chargers, sensor cables/leads (542AP), USB leads, DTS Wireless EMG Sensors, double-sided tape, and a desktop USB receiver. In addition, disposable adhesive Ag/AgCl snap EMG electrodes shaped in a figure of eight and measuring 2.2x4cm, with two 1cm diameter conductive circles and 2cm separating each electrode were used – these are the most commonly employed (Hermens, Freriks, Disselhorst-Klug, & Rau, 2000). Six muscle activities were recorded: the two hamstrings (biceps femoris & semitendinosus), the two quadriceps (vastus lateralis & vastus medialis), and the two heads of the gastrocnemius (lateral & medial). In order to obtain a high-quality EMG signal, the skin was prepared beforehand.

3.5.6 Skin preparation

Skin preparation is required to eliminate artefact-causing interference. It also decreases skin impedance and allows the electrode to stick firmly over the skin, and thus enhances the signal quality. Before EMG data was collected, the skin over each muscle was shaved (2-3 cm) (where necessary) by using a disposable razor and then to remove the dead skin, an exfoliating cream (Nuprep Gel) was applied to the electrode site. Afterwards, the skin was cleaned by using an alcohol wipe (70% isopropyl alcohol) and it was left to dry for two minutes. To finish, disposable adhesive Ag/AgCl (20mm interelectrode distance) snap EMG electrodes were applied over the skin parallel to the muscle fiber.

3.5.7 Electrode placement

The most popular guideline for electrode placement is the SENIAM (Surface Electromyography for the Non-Invasive Assessment of Muscles) Guidelines (http://www.seniam.org). This was used to determine the location of the electrodes by defining muscle locations from specific anatomical landmarks. The SENIAM guidelines are

used to limit or avoid the probability of muscle crosstalk, which is defined as the collection of EMG signals from surrounding muscles and not the muscle under study. In addition, the SENIAM guidelines recommend palpating muscles during manual resisted isometric contractions; this locates the belly of the muscle and anatomical landmarks (Hermens et al., 1999), and minimises the crosstalk (Winter, Fuglevand, & Archer, 1994a). In order to determine the correct position of the electrodes, the subject should be positioned in the initial posture – this is described individually for each muscle by the SENIAM guidelines (Hermens et al., 1999). In this study, the SENIAM guidelines were followed to determine the location of the six muscles listed below:

Medial hamstring (semitendinosus)

The subjects lay prone on the testing plinth and their leg slightly medially rotated. The electrode was placed at 50% on the line between the ischial tuberosity and the medial epicondyle of the tibia. Muscle testing was performed by asking the subjects to flex their knee against manual resistance with a small amount of knee flexion and this was used to test if the identified position was over the belly of the muscle.

Lateral hamstring (biceps femoris)

The subjects lay prone on the testing plinth with the leg is slight lateral rotation. The electrode was placed at 50% on the line between the ischial tuberosity and the lateral epicondyle of the tibia. To test if the identified position was over the belly of the muscle, the subjects were asked to flex their knees against fixed resistance as described above.

Gastrocnemius (medial)

For both gastrocnemius muscles, the subjects were asked to lie in a prone position leg extended, with the foot projecting over the end of the testing plinth. The test was performed by asking the subjects to plantarflex their ankles against a fixed resistance. The electrodes were placed on the most prominent bulge of the muscle. To test if the identified position was over the belly of the muscle, the subjects were asked to plantarflex the foot with emphasis on pulling the heel upward more than pushing the forefoot downward.

Gastrocnemius (lateral)

This used the same procedure as described above, however electrodes were placed one-third of the way along the line between the head of the fibula and the heel.

Quadriceps (vastus lateralis)

The subjects were placed in a sitting position on the testing plinth with the knees in slight flexion and the upper body slightly bent backward. The electrodes were placed two-thirds along the line from the anterior spina iliaca superior to the lateral side of the patella. To test if the identified position was over the belly of the muscle, the subjects were asked to extend their knees without rotating the thigh against a fixed resistance.

Quadriceps (vastus medialis)

The same testing procedure as described above was used for this muscle, however the electrodes were placed 80% along the line between the anterior spina iliaca superior and the joint space in front of the anterior border of the medial ligament.

3.5.8 Checking EMG signal quality

Once the electrodes were in place, the electrodes were connected to transmitters which were adhered to the skin next to the corresponding electrode. Signals of muscle activity were then checked using myoRESEARCH® MR3.10 software (Noraxon, USA). The subjects were asked to relax and contract the muscle isometrically. In both phases (relax and contract), the signal-to-noise ratio was tested. Usually, a good signal-to-noise ratio averaged between 10 to 20 times resting level. As the complete noise-free recording impossible, a small amplitude spikes may be visible, which should be not excessed 10-20 microvolt (Konrad, Gad, & Tilp, 2015; Konrad, 2005). The signal of from each muscle was then inspected visually during walking to ensure there was signal to noise ratio of at least 10-20 times resting level. If the signal was poor during either the isometric or the walking tests then the electrodes were repositioned. Finally, the recording electrode transmitters were covered by a crepe bandage (4cm x 2.5m) to minimise the possibility of any movement artefact.

The initial plan had been to measure the two limbs as this would have provided considerable further insight into the effect of trunk inclination on muscle activation patterns. However, the

lab protocol was very complex protocol and, although it was piloted with two limbs (12 EMG electrodes), it was found to be too long and deemed to be too much for participants to tolerate. Therefore, EMG data was only collected on the (most) affected limb for knee OA participants and a matched limb for the control healthy subjects.

3.5.9 Kinematic and kinetic data collection procedure

Kinematic and kinetic data were collected by a motion capture system synchronised with integrated force platforms. Motion capture systems work by emitting infra-red light, which is reflected from the markers to the camera. This allows each camera to obtain a 2D image of the markers. Then for each marker, the three-dimensional position is calculated by combining the two-dimensional images with knowledge (from the calibration) of the relative position of each camera (Kaufman & Sutherland, 2006). In order to identify a unique 3D marker trajectory, at least two cameras are required (Cappozzo, Della Croce, Leardini, & Chiari, 2005). However, for this protocol, at least three cameras could capture data on each marker for all points in the data collection volume.

An Oqus Qualisys motion analysis system (Qualisys ™, Gothenburg, Sweden, 2003) was obtained to calculate the kinematic data operating at 100Hz, which is acceptable in walking (Hori et al., 2009). Fourteen infra-red cameras (Qualisys, Sweden) were used to capture the 3-dimensional positions of the retro-reflective markers attached to each subject's skin over bony landmarks in the lower limbs and trunk. In addition to collecting kinematic data, kinetic data were collected from two AMTI force platforms (model BP400600, AMTI (AMTI: Advanced Mechanical Technology Incorporation), Watertown, MA, USA) at 1500Hz (Figure 3-6). To ensure the accuracy of the marker placement, the location of anatomical markers were palpated first. Then retro-reflective markers (14.5 mm) were placed over bony landmarks using hypo-allergenic adhesive tape. Individual markers were attached on anterior superior iliac spines (ASISs), posterior superior iliac spines (PSISs), iliac crests, right greater trochanter, left greater trochanter, lateral femoral epicondyles, medial femoral epicondyles, lateral malleoli, medial malleoli, the 1st, 2nd and 5th metatarsal heads, and calcaneal tubercle. In addition to these marker placements, markers were placed on the right and left acromion process, sternum, seventh of cervical vertebral, second and eighth of thoracic vertebral. Also, cluster pads with four markers were placed on the shank, thigh, and pelvis using

FabiofoamSuperwrap bandages to reduce the movement of these clusters (Figure 3-7). Further details about how the different markers were used to either define the coordination system or track motions of the individual segments is given in section 3.6.

Figure 3-6 Podiatry gait lab with 16 infra-red cameras and four force plates.

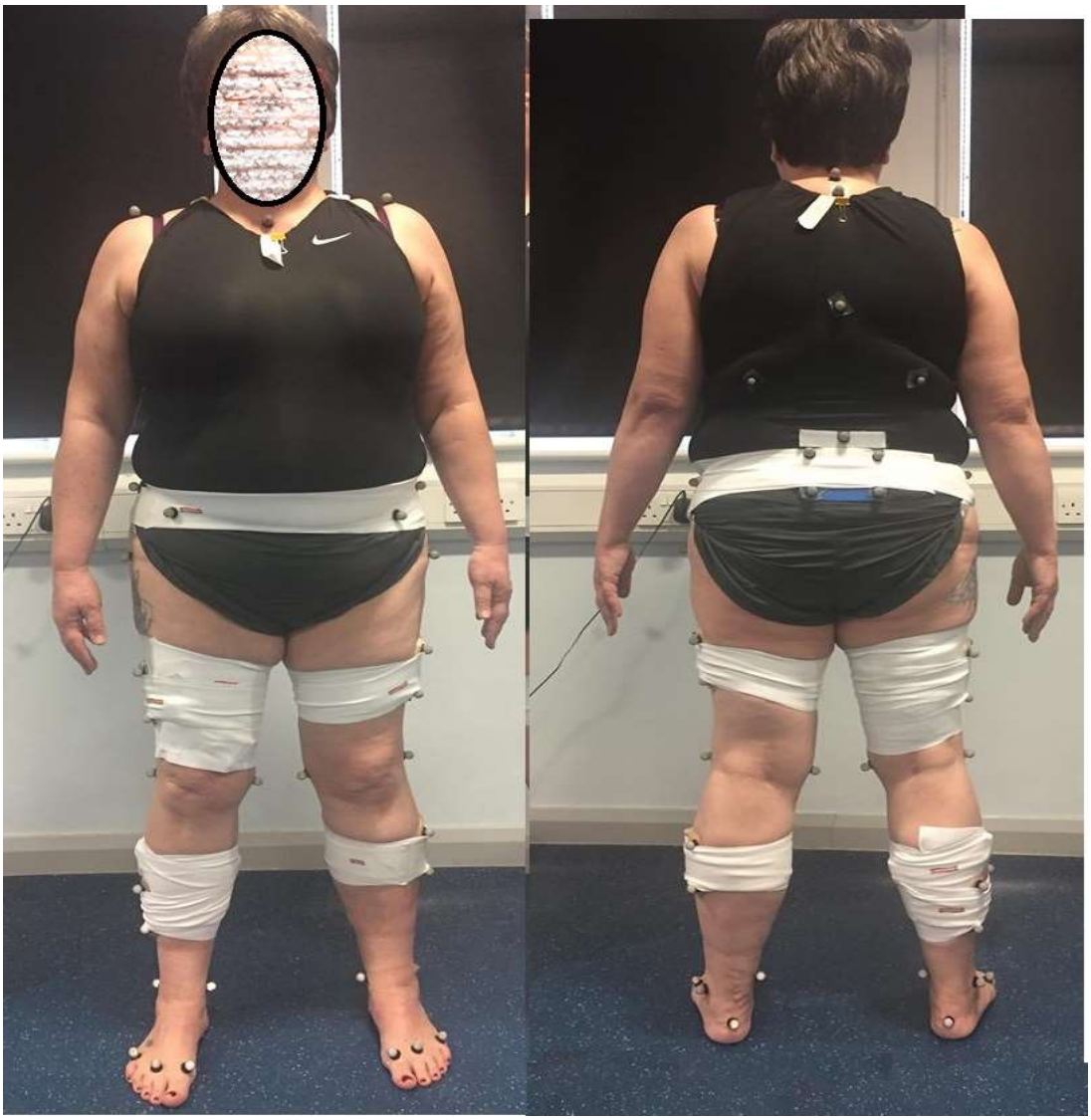


Figure 3-7 Retro-reflective markers (Anterior and posterior views)

3.5.9.1 System calibration

Prior to data collection, the camera system was calibrated. This calibration is essential to produce a laboratory coordinate system (static), identify the camera position and orientation, and identify the lens focal length (dynamic). Calibration tools consist of two parts: the reference L- shaped metal frame (static) and a calibration wand (dynamic) (Figure 3-8). The reference L-frame with its markers was placed on the first force plate parallel to the X and Y axis. The reference L-frame used to identify the origin and orientation of the laboratory and coordinate system with the X (medial/lateral) axis, Y (anterior/posterior) axis, and Z (the vertical) axis. In addition, a wand with two markers was moved inside the measurement volume in all directions for 60 seconds and the reference L-frame was kept still on the force

plates (Payton & Burden, 2017). Once calibration was completed, the calibration result for both cameras and the standard deviation (residual) of the wand length were less than 1 mm. Generally, the lower the residual the more accurate the calibration.

Figure 3-8 The wand equipped with two markers and the L-shaped metal frame.

3.5.9.2 Static test

Once all of the electrodes and reflective markers were in place, the participants were asked to perform a static trial, by taking a step forward to the first and second force plate (each foot on a force plate) and they were instructed to stand in a stationary position for 10 seconds while the image data from the cameras were recorded. Following this, the static trial was labelled and exported to a C3D file. Then the subject's normal trunk angle during standing was calculated.

3.5.10 Collection of biomechanical data during normal walking

Following the collection of the static trial, the walking data was collected. Participants were instructed to walk through the lab from a specific starting position which was chosen to

ensure that they made contact with the force plates. However, they were not told about the existence of the force plates. For these experiments, it was important to control walking speed to ensure that variations in speed did not affect the results. Therefore, to monitor the walking speed, the subject was timed using optical timing gates for each trial. A normal walking speed was first established across five practice walks and only trials within ± 5 % of the subject's self-selected walking speed accepted. The participants were instructed to walk normally at a self-selected speed for along a 6-metre walkway. The trial was accepted if the whole foot made contact with the force platform in the middle and the speed was within the predetermined range (see above); otherwise the trial was rejected. All walking testing was carried out barefoot. A minimum of 5 successful trials were captured for each participant.

3.5.11 Biofeedback and collection of walking data at different trunk lean angles

One of the aims of this project was to understand the effect of independently manipulating trunk inclination during walking. To achieve this aim, it was necessary to provide subjects with precise feedback on their trunk angle during the walking trials for which they were required to adopt an unnatural trunk inclination. Extensive piloting work was performed to develop a robust experimental protocol which could be used to collect walking data at different trunk lean angles. This pilots is described first before the full protocol is described.

3.5.11.1 Pilot work

The initial plan was to develop biofeedback software which could provide participants with instantaneous feedback on their trunk ankle during the walking trial. Although this type of feedback can be delivered with the visual 3D software, participants were not able to respond sufficiently quickly and therefore it was not possible to use this approach to guide participants to change their trunk angle during walking. Therefore, the decision was made to provide feedback on trunk inclination after each walking trial immediately after the trial. This information was not shown to the participant but was used by the experimenter to provide verbal instruction to either maintain, increase or decrease their trunk angle for the subsequent walking trial. However, to facilitate this learning we also used real-time feedback on standing trunk angle (see section below) to help participants get a feel for the target trunk positions. By practicing 3-4 times in standing and then 5-10 times during walking, participants

learned what it felt like to decrease/increase their trunk angle by 5/10°. Once participants were able to achieve the target trunk at the target flexion angle, then a set of trials were recorded. However, subjects did not always achieved the target angles and therefore only trials which were within +/-2 degrees of the target flexion angle were used. A total of 5-10 successful trials were recorded at each trunk flexion angle (see below) and this typically required a minimum of 15-20 actual trials. .

3.5.11.2 Final experimental protocol

As explain above, feedback was provided via a two-stage process. Firstly, feedback was given in real-time in a standing position to enable the participant to develop a clear kinaesthetic sense of a 5/10°increase in trunk inclination (forward lean) and a 5° decrease in trunk inclination (backwards lean). This feedback was provided by first calculating the standing angle of trunk inclination from the static trial (section 3.3.9.2) and then using this as input to a custom MATLAB programme (see below). For details on how trunk angle was calculated see section 3.6.4.

The MATLAB script, which provided real-time feedback on trunk standing trunk angle, communicated directly with the V3D server and applied the visual3D model. Trunk angle data was calculated by the V3D server, streamed into MATLAB and plotted as shown in as in Figure 3-9 below. This biofeedback system was shown on a big screen in front of the participant. By practicing 5-10 times, participants learned what it felt like to decrease/increase their trunk angle by 5/10°.

Figure 3-9 Biofeedback during standing.

The next stage of the biofeedback was applied during walking. Pilot testing showed that subjects could not respond quickly enough to real-time feedback during walking and therefore feedback on trunk inclination after each walking trial was provided immediately after the trial. This was achieved by using the V3D server with another piece of custom MATLAB code to provide a rapid visualisation of trunk angle across the gait cycle, see Figure 3-10 below. This information was not shown to the participant but was used by the experimenter to provide verbal instruction to either maintain, increase or decrease their trunk angle for the subsequent walking trial.

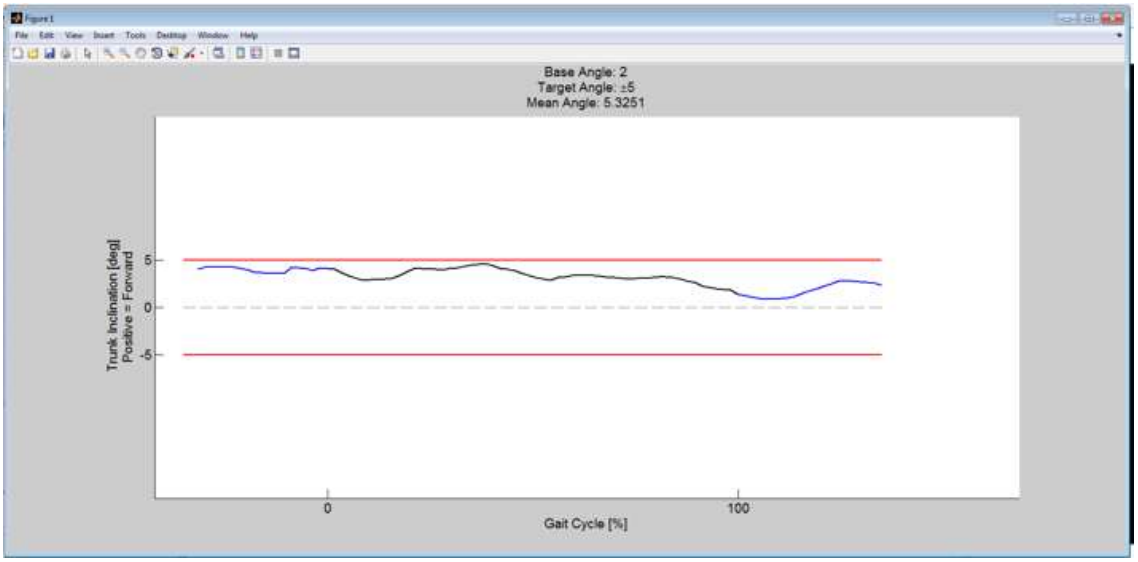


Figure 3-10 Biofeedback for trunk angle during walking.

Once the participant could walk consistently with a trunk angle 5° higher than their natural trunk position, then collection of the different trunk lean conditions was performed. Specifically, a minimum of five successful trials (within +/-2 degrees of the target flexion angle) were collected in each of the following conditions:

1. Condition 1: normal trunk angle -5° flexion
2. Condition 2: normal trunk angle +5° flexion
3. Condition 3: normal trunk angle +10° flexion

The order of the conditions above was randomly selected by a MATLAB custom code. Following each trial, feedback on trunk inclination was provided as explained above. Again,

trials were only included if they were within +/-2 degrees of the target flexion angle. Pilot testing demonstrated that people with knee OA, and the majority of healthy older subjects, could not consistently perform condition 3, therefore this condition was omitted for these two groups.

3.5.12 Reference contractions

EMG signals collected during walking are dependent on electrode position (Jensen, Vasseljen, & Westgaard, 1993), temperature (Winkel & Jørgensen, 1991), muscle fatigue (HANSSON et al., 1992) and fat thickness (McGill, 1991). Therefore, in order to be able to compare the EMG activity across different individuals or across different muscles, the EMG signals should be normalised. The term EMG normalisation has come to be used to refer to the process by which EMG signals of a task are expressed as a percentage of that muscle's activity during a reference contraction (Lehman & McGill, 1999). Normalisation of EMG signals is usually achieved by dividing the EMG signals during activity by a reference EMG value of the same muscle. There are many different ways for normalisation to produce reference EMGs.

Numerous methods have been proposed to normalise EMG activity (Halaki & Gi, 2012), including isometric, isokinetic and dynamic muscle actions (peak and mean dynamic) (Burden, Trew, & Baltzopoulos, 2003; Lehman & McGill, 1999). With the peak dynamic approach, the EMG signals are normalised using the peak activation of the muscle over the movement (walking). This results in a signal that normalized by peak and which varies between 0 and 1 (corresponding to the peak activation). Some authors recommend using the mean and dynamic methods in order to reduce inter-subject variability (Burden, 2010); however, as the mean/dynamic methods involve normalisation using the peak activation, this will remove important information from EMG signals related to the overall level of muscle activation. As an alternative to dynamic methods, a maximal voluntary isometric contraction (MVIC) is often used to normalize EMG activity. With this method, a maximum contraction is used as the reference for each muscle (Arsenault, Winter, Marteniuk, & Hayes, 1986; Yang & Winter, 1983).

The MVIC method is suggested by SENIAM's and Kinesiology's guidelines and within the literature, there have been studies aiming to identify which exercises to use for research

aimed at comparing muscle patterns with knee OA to healthy subjects (Heiden et al., 2009; Hubley-Kozey, Robbins, Rutherford, & Stanish, 2013). Although, MVICs can be affected by pain (Benoit, Lamontagne, Cerulli, & Liti, 2003) which is a common symptom in OA patients, this method is widely used in the majority of the literature studies in people with knee OA (Heiden et al., 2009; Hortobagyi et al., 2005; Lewek, Rudolph, & Snyder-Mackler, 2004b). Many studies have demonstrated good and acceptable EMG reliability between days (Hsu, Tang, & Jan, 2002) and weeks (Ball & Scurr, 2010), respectively. In addition, good reliability was reported for MVIC in patients with knee OA (Hubley-Kozey, Robbins, et al., 2013). Moreover, the majority of the 14 studies described in a systematic review and meta-analysis of muscle activation in knee OA (Mills, Hunt, Leigh, & Ferber, 2013) used the MVIC technique to normalize the EMG data. This is mainly because it gives the best indication of how active the muscle is. Given the widespread use of this approach, MVIC normalisation was use in this thesis.

3.5.12.1 Maximal voluntary isometric contractions procedure (MVIC)

Following the walking tests and the removal of markers, EMG calibration data were collected during a reference contraction for each muscle group. A separate test was performed for each muscle, designed to elicit a MVIC. Three trials, each lasting for 5 seconds, were completed with 60 seconds rest between each muscle contraction to eliminate the effect of fatigue. Verbal encouragement, which is recommended to give maximal effort (McNair, Depledge, Brettkelly, & Stanley, 1996) for each test, was provided (see below for details of the individual tests). A goniometer was used to determine the required angles (Error = +/- 1 degree). In addition, a padded 10 cm wide non-extendable resistance mobilisation strap was used for resistance. Using a mobilisation strap has become common for measuring MVICs (Boren et al., 2011; Nyland, Kuzemchek, Parks, & Caborn, 2004). In addition, it should be noted that a fixed resistance strap was firmly secured and it did not rely on human strength (Silvers & Dolny, 2011).

3.5.12.2 Positioning for MVICs

There is variability in the recommended positions for exercises used for normalisation testing. However, some studies do not report the joint position during MVIC procedures (Lewek et al., 2004a; Ramsey, Briem, et al., 2007) and others use a single standard exercise for each muscle

(Childs et al., 2004; Zeni, Rudolph, & Higginson, 2010b). This inconsistency has motivated researchers to understand the effect of different normalisation exercise protocols in healthy subjects and people with knee OA (Rutherford, Hubley-Kozey, & Stanish, 2011b). Rutherford, Hubley-Kozey, et al. (2011b) have examined maximum contractions for quadriceps, hamstring and gastrocnemius muscles in different exercises for sixty-eight knee OA patients and sixty-eight matched healthy subjects. They concluded that the maximum activations for gastrocnemius muscle were during standing plantarflexion and maximum activations for the quadriceps muscles were during 15 degree of knee extension. In addition, the greatest hamstrings muscle activation was during 55 degree of knee flexion (Rutherford, Hubley-Kozey, et al., 2011b). Therefore, in this thesis, these recommendations were followed.

Hamstring muscles

The subjects were required to lie prone on the bed and a mobilisation strap was applied to the waist and distal femur and attached to the bed to provide constant conditions. Then the knee joint of the tested leg was positioned into 55 degree flexion by a goniometer. Another mobilisation strap, which was attached to the bed, was secured around the distal tibia of the tested leg for resistance. The subjects were asked to try to flex their knee as hard as they could against a resistance belt without changing their position.

Gastrocnemius muscles

In order to measure the references for gastrocnemius muscles, the subjects were required to be in a standing position. Then they were asked to bend the non-testing leg and to try to stand on the toes of the testing leg. They were then asked to push as hard as they could onto their tip toes.

Quadriceps muscles

Lastly, the subjects were seated with their tested knee positioned at 15 degree of flexion, measured using a goniometer. A mobilisation strap, which was attached to the bed, was secured around the distal tibia of the tested leg for resistance. Then the subjects were asked to extend the knee of the tested leg as hard as they could against a resistance belt without changing their position.

As explained above, data were collected, from each muscle group during three separate trials, each lasting for 5 seconds. A 60 seconds rest was provided between each contraction. This data was collected using the Noraxon MR3 software operating on a laptop computer.

3.6 Data processing

3.6.1 Derivation of joint angles and moments from the raw marker data

A variety of marker sets have been used in clinical gait analysis. One of the most commonly used in gait analysis is the conventional gait model (CGM) (Davis, Ounpuu, Tyburski, & Gage, 1991; Kadaba, Ramakrishnan, & Wootten, 1990). The CGM markers set only allows three rotational degree of freedom for hip and knee and two for ankle. Importantly, a minimum number of markers and a large distance between the markers make the CGM very sensitive and affected by skin movement (Cereatti, Camomilla, Vannozzi, & Cappozzo, 2007). In addition, in CGM, it is impossible to identify the position and orientation of segments independently of other segments because in the CGM, only two tracking markers are used to provide each segment (Cereatti et al., 2007; Schwartz, Trost, & Wervey, 2004). An alternative to the CGM model is the six degrees of freedom (6DOF) proposed by (Cappozzo, Catani, Leardini, Benedetti, & Della Croce, 1996).

With this approach, each segment is tracked independently of the others by four retro-reflective markers attached these to the two shanks, two thighs and pelvis. The data are used to calculate three rotational orientations (sagittal, transvers and frontal) and three translational positions (vertical, medial/lateral and anterior/posterior) for each segment and these data subsequently used to derived joint angles, between the different segments. With this approach, each segment is measured independently and leads to lower error and less effect of skin motion artefact. The calibration anatomical systems technique (CAST) was used to define the 6DOF movement of each segment during the dynamic tasks. Indeed, it has been shown that 6DOF results in fewer errors than CGM (Cappozzo et al., 1996; Cereatti et al., 2007). Therefore, we used a 6DOF model to derive kinematics and joint moments for this thesis.

3.6.2 Kinematic and kinetic data processing

All kinematic and kinetic collected data were processed using Qualisys Track Manager (QTM) software (Version 2.17) and Visulal3D (V3D) software (Version 6 x64). To begin with, each marker was labelled for the standing and walking trials (normal walking and walking with different trunk leans) using the QTM software. Then all trials were exported to C3D format. The V3D software was then used derived joint angles and obtain joint moments. Firstly, the C3D files were imported into V3D and raw marker data and force data were interpolated and low pass filtered to minimise the noise and remove the high frequency. A 6Hz cut off point for kinematics data (Winter, 2009) and 25Hz for kinetics (Schneider & Chao, 1983) was applied to filter the data using a Butterworth fourth-order filter. A 6DOF model was created in V3D which consisted of two feet segments, two shank segments, two thigh segments, a pelvic and a thorax. This is described in more detail below.

3.6.3 Defining and tracking the lower limb and pelvic segments.

With the 6DOF model created in V3D, all joints were modelled (comprising 8 rigid segments: the thorax, pelvis, right and left thigh, right and left shank and right and left foot) to determine the proximal and distal joint and tracking markers as illustrated in Table 3-1. In order to derive segmental positions and orientations, each segment was tracked with at least three (and sometimes four) non-collinear markers. The Cartesian Optoelectronic Dynamic Anthropometer (CODA) refers to a pelvis segment model which was recommended by (Reize, Müller, Motzny, & Wülker, 2006). The pelvis segment was defined using anatomical locations of the ASIS and PSIS and was tracked by three tracking markers (Pelvis cluster pad). In addition, thigh, shank and foot segments were determined with distal, proximal and tracking markers as descripted in the following table 3-2. As trunk represents a significant portion of total body (Winter & Yack, 1987), thus in gait analysis the thoracic segment should be considered carefully.

3.6.4 Defining and tracking the thoracic segment

To date, several different kinematics model of the thorax have been suggested. For example, Davis, Õunpuu, Tyburski, and Gage (1991) defined the thorax by only two markers: one of them between the right and left clavicle and another one over the Cervical 7 (C7) (Davis,

Õunpuu, et al., 1991). Furthermore, Gutierrez, Bartonek, Haglund-Åkerlind, and Saraste (2003) and Nguyen and Baker (2004) define the thorax by C7, the manubrium sternal or mid-point between clavicles, the sternum (and T10 for (Gutierrez et al., 2003)). The International Society of Biomechanics recommended placing the markers over the incisura jugularis (IJ), process of xyphoid (XP), C7 and Thoracic 8 (T8). However, some of these models have been found to lead to problems, for example the C7 marker was found to be highly responsive to movements of the head (Armand, Sangeux, & Baker, 2014). Recently, it was identified that the optimal marker set with minimum error was that used by (Armand et al., 2014). In this comparison study, researchers investigated two families of markers: one of which was collected by two anterior and one posterior marker (IJ, XP and T8) and another collected by two posterior and one anterior marker (IJ, T2 and T8 or T10). This study found that the former family was not sufficiently adequate to measure the frontal plane and the latter family was shown to give a better measure of range of movement. In addition, this study found a good reliability for the latter family of markers. They concluded that the optimal and minimal markers to define the movement of the thorax should be IJ, T2 and T8 or T10 (Armand et al., 2014). Given the potential utility of using these markers to define the thorax, it was chosen in this thesis.

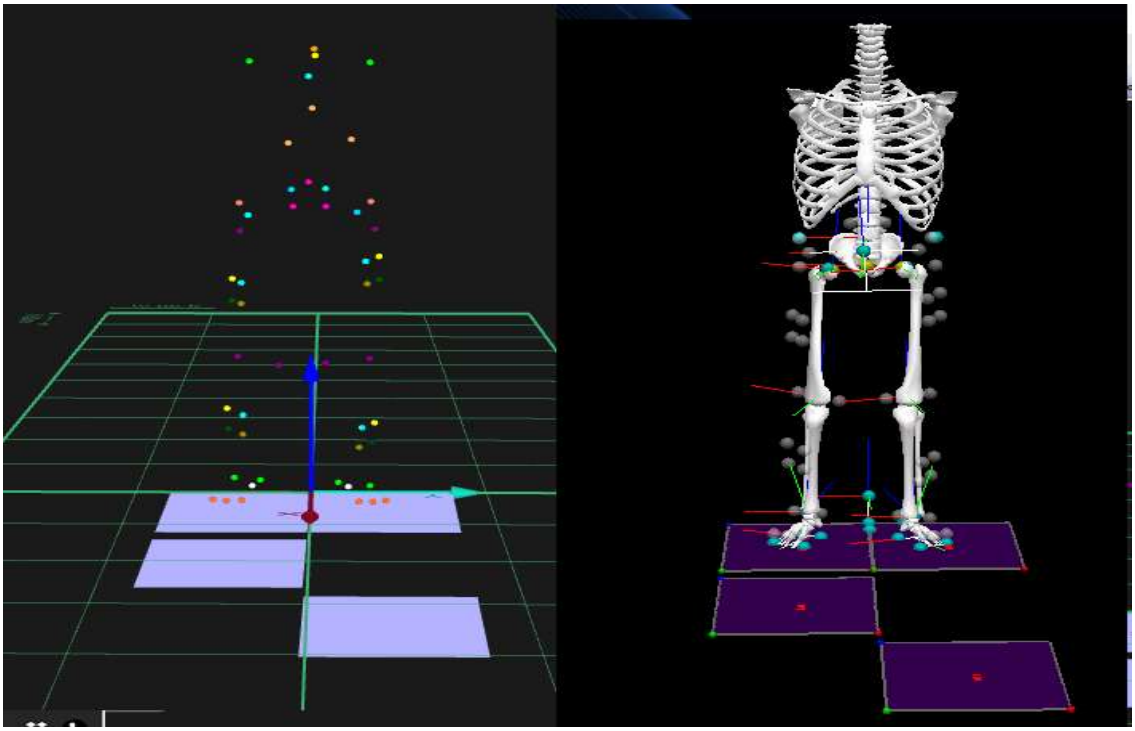


Figure 3-11 Qualisys Track Manager and Visual 3D Model

Table 3-1 Segments, proximal & distal joint and tracking markers

SEGMENT	PROXIMAL JOINT	DISTAL JOINT	TRACKING MARKERS
THORAX	Right and left greater trochanter	Right and left acromion	Jugular notch, 2nd and 8th thoracic vertebra
CODA PELVIS	Right and left anterior superior iliac spine	Right and left posterior superior iliac spine	Pelvis cluster pad (3 tracking markers)
THIGH	Right and left greater trochanter	Medial and lateral femoral condyle	Thigh cluster pad (4 tracking markers)
SHANK	Medial and lateral femoral condyle	Medial and lateral malleolus	Shank cluster pad (4 tracking markers)
FOOT	Medial and lateral malleolus	1st and 5th metatarsal heads	1st, 2nd and 5th metatarsal heads Right and left heel calcaneus

3.6.5 Deriving joint angles and joint moments

After the raw data was filtered, visual 3D (V3D) was used to calculate joint kinematics and kinetics. Specifically, joints kinematics were obtained by applying Cardan/Euler angle calculations to determine 3D joint angles between each set of adjacent segments (Cole, Nigg, Ronsky, & Yeadon, 1993). In addition, force data was the used as part of inverse dynamic calculations, to derive joint moments at the hip, knee and ankle. The subject's mass (in kilograms) were entered into the V3D to normalise the joint moment data. Kinetic data was normalised to 100% of a stance phase, while the kinematic data were normalised to 100% of

a gait cycle. Then gait curves were exported as spreadsheets into Microsoft Excel 2016 to conduct the statistical analysis and construct a graphical plots of the data. This was done for each participant's trial and each trunk lean condition, creating a database containing all data. Using these data, the ensemble averages from the kinematics and kinetics data were graphed for the full gait cycle and stance phases, respectively for each subject and each trunk lean condition. Finally, from these curves, specific outcomes were calculated for each of the separate studies.

3.6.6 EMG data processing

The EMG data for each walking trial was collected using the QTM software and therefore synchronised with the gait data. This EMG was exported in C3D format which could be ready by a custom written Matlab programme (developed by my supervisor). This programme was used as an alternative to the commercially available Noraxon software to reduce cost. EMG data processing was performed via a three-stage process: the first step was to use a high pass filter (20Hz) to remove movement artefacts and noise. The second step was rectification, which makes the signal positive. The final step was to apply a low pass filter to create a linear envelope. A cut frequency of 6Hz was selected as this has been used widely in knee OA research (Heiden et al., 2009; Hubley-Kozey et al., 2006; Hubley-Kozey, Hatfield, Wilson, & Dunbar, 2010; Winter & Yack, 1987). All three steps were performed in custom Matlab software. The software also allowed export of EMG data normalised to stance phase (heel strike to toe off) to Microsoft Excel 2016. The ensemble average curves were produced for each muscle, trunk lean condition and participant. The software also enabled the export of ensemble average EMG data normalised from -29 to 129 of stance phase so that muscle activation immediately prior to foot contact could be analysed.

Figure 3-12 Screenshot from the MATLAB software. It shows the EMG processing steps which are: high pass filter, rectification and low pass filter.

MVIC data processing

As discussed earlier in section (3.5.12), to be able to compare the EMG activity across different individuals or across different muscles, the EMG signals should be normalised. For this work, an MVIC methods of normalisation was used. Three trials of MVIC data (for each muscle group), each lasting for five seconds, were recorded with minimum a minute rest period between each contraction (See section 3.5.12.1 above) . Once data was collected, the MVIC was exported into another custom written MATLAB programme which facilitated processing of the MVIC data in order to derive the maximum EMG amplitude. For this calculation, the same high pass and linear envelope filtering as with the gait EMG signals (see above) were applied to the raw EMG data. A moving average filter (window = 0.1 s) was then used to create a smooth signal, following previous studies (Hubley-Kozey et al., 2006; Preece, Jones, Brown, Cacciatore, & Jones, 2016). Finally, the peak of this smooth signal was taken as the MVIC value. As there were three trials for each muscle group, the final MVIC normalisation factor was selected based on the largest of the three values for each trial. This processed was repeated for each muscle and each participant. Finally, gait EMG signals were divided by the appropriate MVIC normalisation factor.

3.7 Overview of outcomes measurement

The analysis described above explains how kinematic, kinetic and EMG signals were derived over the gait cycle or stance phase. However, to investigate the key ideas in this thesis, it was necessary to produce specific outcomes from this continuous gait data. Based on the modelling studies (Section 2.6.2), a specific window corresponding to peak loading was selected during which kinematic, kinetic and EMG data was analysed. Specifically, the window 15-25% of stance phase was calculated and used for kinematics and kinetic data. However, a window 10-20% of stance phase was used for EMG data in order to account for electromechanical delay (adjusted backwards to account for a 40 ms) (EMD). See below for further justification of this specific time delay. Figure 3-13 below shows differences in contact force between two patterns of muscle coordination: high co-contraction (red) and low co-contraction (blue) (Brandon et al., 2014). It can be seen that contact force associated with the increased co-contraction pattern (red) is noticeably, compared to that associated with the low co-contraction pattern (blue) during the first period, which is centred on 12.5% of the full gait cycle, with minimum differences in contact force at the second peak. Assuming that the stance phase is approximately 62% of full gait cycle, then a first peak occurring at 12.5% of full stance phase corresponds to 20% of stance. Consequently, all subsequent analysis related to kinematics and kinetics in this thesis were focused on the time period 15-25% of stance, chosen to be centred on 20% of stance. As discussed earlier this widow was adjusted backwards for the EMG data and this is explained in more detail below.

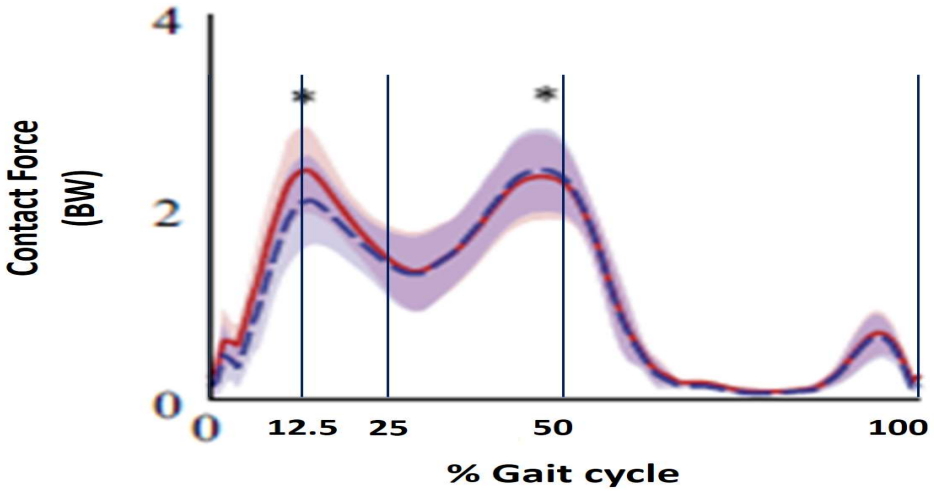


Figure 3-13 The contact knee force across the full gait cycle during walking with (low co-contraction (blue) and high co-contraction (red). Adopted from (Brandon et al., 2014)

3.7.1 Electromechanical delay (EMD)

The electromechanical delay (EMD) is the time delay between the onset of the muscle signal and the initiation of muscle force. This period of time should be determined in order to precisely relate EMG and motion in selected research studies (Zhou, Lawson, Morrison, & Fairweather, 1995). Previous studies have determined EMD for muscle activation, for example, voluntary knee extension (EMD=40 ms) (Perry, 1998). In addition, Houston, Norman, and Froese (1988) found that the EMD was 43.3 ms. Nevertheless, it was suggested by Perry (1998) that the average EMD during gait is no more than 40 ms. Therefore, in this thesis, the EMD was set as 40 ms. To implement this the mean stance time across all participants were calculated and found to range from 600-700 ms. Thus 40ms was taken to be equivalent to 5% of stance and the 15-25% window adjusted accordingly to 10 to 20% of stance.

3.8 Test-retest reliability of gait

The main aim of this thesis was to understand the effect of trunk inclination on lower limb biomechanics. As clinical gait analysis, like any clinical test, is subjected to measurement error, it is important to ascertain that all measurements have good reliability. To confirm this, a reliability study was performed to assess the test-retest reliability of gait kinetics and muscle activation in healthy subjects across two days. This study is described in detail in Appendix IX and summarised here in abstract form. The mean age, mass and height (mean ± SD) of subjects was 26.5 ± 3.9 years, weight was 63 ± 7.9 kg and height was 1.70 ± 0.5 cm. Our result shows that the walking speed did not change significantly ($p > 0.05$) between days. ICC and SEM results for trunk angle, joint moment and muscle activation normalised by maximum isometric contraction (MVIC) are presented below. Table 3-3-2 shows that the reliability results for the trunk angle and the sagittal hip, knee and ankle moments during normal walking. The results showed good reproducibility of the trunk angle during normal walking (ICC = .68 & SEM = .84) and good/excellent reproducibility of the sagittal hip, knee and ankle moments during normal walking and across the different trunk lean conditions. In addition, Table 3-3-2 shows the ICC and SEM results of lower limb muscle activations (normalised by MVIC) averaged between 10-20% of stance phase during normal walking. As can be seen from

Error! Reference source not found., most of the ICC result showed that the reliability was excellent to good. For more details see the (Appendix IX).

Table 3-3-2 The ICC and standard error of measurement (SEM) results of the trunk angle (over the full gait cycle), lower limb joint moment (averaged between 15-25% of stance phase) and muscle activation (averaged between 10-20% of stance phase) during normal walking. (Trunk angle = °, Joint Moment (Nm/Kg) and muscle activation (normalized by MVIC).

	MEAN (SD) 1ST VISIT	MEAN (SD) 2ND VISIT	ICC	SEM
TRUNK ANGLE	4.9 (1.8)	3.1 (1.1)	.68	0.848
HIP MOMENT	.3 (.2)	.2 (.2)	.91	0.012
KNEE MOMENT	.6 (.2)	.7 (.3)	.97	0.043
ANKLE MOMENT	.0 (.1)	0 (.1)	.96	0.02
MEDIAL GASTROCNEMIUS (MG)	.09 (.06)	.09 (.03)	.70	0.025
LATERAL GASTROCNEMIUS (LG)	.07 (.04)	.07 (.02)	.90	0.009
VASTUS MEDIALIS (VMO)	.17 (.11)	.17 (.10)	.92	0.029
VASTUS LATERALIS (VLO)	.17 (.11)	.20 (.11)	.96	0.020
SEMITENDINOSUS (ST)	.08 (.03)	.07 (.03)	.97	0.005
BICEPS FEMORIS (BF)	.08 (.04)	.07 (.02)	.86	0.011

Chapter Four

(Study One)

What are the key lower limb biomechanical differences between people with knee osteoarthritis and healthy participants during walking?

4.1 Introduction

In the previous chapter, data were presented demonstrating the reliability of kinetic and muscle activation measurements, performed using the proposed protocol. These measurements are key to understanding the biomechanical differences between people with knee OA and healthy participants. This first study aimed to fully characterise differences in sagittal plane trunk inclination and other biomechanical variables between people with knee OA and healthy controls. Before these biomechanical differences are explored, it is important to briefly review the previous literature investigating these differences.

Previous studies have reported that people with knee OA walk with alterations in lower limb biomechanics. For example, it was noted that people with knee OA walk with an increase in the hip extensor moment (Huang et al., 2008; Liu et al., 2014). Furthermore, people with knee OA walk with a significant increase in the magnitude of their hamstring muscle activity compared to matched healthy participants (Rutherford et al., 2013; Wilson et al., 2011) along with increased hamstring-quadriceps co-contraction. Interestingly, some research has identified a reduction in the knee extensor moment in people with knee OA (Astephen, Deluzio, Caldwell, & Dunbar, 2008), however other studies have found no differences (Baliunas et al., 2002; Duffell, Jordan, Cobb, & McGregor, 2017). These conflicting findings motivate further research comparing the biomechanical characteristics of people with knee OA and healthy participants. Recently, it was shown that people with knee OA walk with an increased forward lean compared to matched-age subjects (Preece et al., 2018). Given the

central focus of this thesis on the potential impact of forward lean, it is important that the finding of this research is fully confirmed via follow-on studies.

This chapter seeks to repeat research which has been carried out previously that characterises the differences between people with knee OA and matched healthy controls. The purpose of this is to fully validate the findings of previous research and create a clearly defined set of biomechanical characteristics which are typical of people with knee OA. These data will be used to interpret the results of subsequent chapters which seek to explore the effect of increased trunk lean in healthy people (Chapter 5 & 7) and people with knee OA (Chapter 6). A secondary aim of this study was to quantify biomechanical differences between a healthy young people and an older group, matched (from BMI & age) to the group with knee OA. This comparison was performed to facilitate interpretation of the data on the young healthy people (Chapter 5 & 7).

4.2 Research questions and hypotheses

The aim of this study was to characterise biomechanical differences during walking between three groups of subjects: young healthy people, older healthy people, and people with knee OA. These differences were characterised for six separate aspects of walking:

Q 1 What is the differences in sagittal trunk angle between people with knee OA, old and young healthy people during walking?

H 1 People with knee OA will walk with increased sagittal trunk angle compared to healthy groups.

Q 2 What are the difference in the sagittal hip, knee and ankle moments between people with knee OA, old and young healthy people during walking?

H 2 People with knee OA will walk with increased hip extensor moment and ankle plantarflexor moment compared to healthy groups.

Q 3 What are the differences in gastrocnemius, quadriceps and hamstring muscle activities in people with knee OA, old and young healthy people during walking?

H 3 People with knee OA will walk with increased gastrocnemius, quadriceps and hamstring muscle activities compared to healthy groups.

Q 4 What are the differences in gastrocnemius, quadriceps and hamstring muscles co-contraction people with knee OA, old and young healthy people during walking?

H 4 People with knee OA will walk with increased gastrocnemius, quadriceps and hamstring muscle co-contraction compared to healthy groups.

Q 5 What are the differences in sagittal hip, knee and ankle angles people with knee OA, old and young healthy people during walking?

H 5 There will be no differences in lower limb kinematics between people with knee OA compared to healthy groups.

Q 6 What are the differences in the spatiotemporal (speed and step length) people with knee OA, old and young healthy people during walking?

H 6 There will be no differences in the spatiotemporal parameters between people with knee OA compared to healthy groups.

4.3 Methods

A full description of the method for all studies in this thesis, including this study, was provided in the method chapter (see Chapter Three). In this study only, normal walking data were used for the OA, older and young participants. In the following paragraphs, the sample characteristics and primary outcome measures used to analyse the data and the appropriate statistical tests for this study are presented. As explained in the methods section, EMG data was collected from only one limb as, experimentally, it too demanding for the participants to measure both limbs given that this would have required 12 EMG electrodes.

4.3.1 Sample and population

All testing was carried out in the Podiatry lab in the University of Salford. Three groups: the knee OA subjects (n = 20); older healthy subjects (n = 20), and young healthy subjects (n = 20) across both genders were recruited. Participants' characteristics are presented in Table 4-1.

The inclusion/exclusion criteria have been outlined above (see section 3.2). Subjects in the young control group had lower BMI values than the subjects in the OA and older groups (P = .002). It can be noted from Table 4-1 that people with knee OA had high BMI which is typical of this group of patients as increased BMI has been identified as a risk factor for this disease (Silverwood et al., 2015a). Therefore, the older group were purposely selected to include people with higher BMI in order to match the group with knee OA. No differences in age, height, weight or BMI were observed between the OA and older groups.

Table 4-1 Participant characteristics for all groups: people with knee OA, older healthy and young healthy subjects. Values are the mean ± Standard Deviation (SD).

Variables	Knee OA	Older healthy	Young healthy
No. of subjects	20	20	20
Age (Years)	56.0 (8.7)	57.2 (8.7)	26.1 (6.8)
Height (M)	1.7 (.07)	1.7 (.06)	1.7 (.07)
Mass (Kg)	83.1 (14.4)	80 (11.3)	66.1 (8)
BMI (kg/m2)	28.7 (4.9)	27.4 (3.9)	22.2 (2.5)
Gender (M/F)	5/15	7/13	11/9
WOMAC score (Total: pain, function and stiffness)	33.7 (12.2)	----	----

During data collection subjects were required to walk at a self-selected speed. Each trial was accepted only if the whole foot made contact with the force platform and the speed was within ± 5% of the subject's self-selected walking speed. All walking testing was carried out barefoot. See section 3.3.1. for more information.

4.3.2 Derivation of outcome measures

This study was designed to determine differences in biomechanical characteristics between knee OA patients, older and young healthy people during normal walking. In the following paragraphs, the primary outcome measures relevant for each of the separate aspects of walking are explained in detail.

Trunk angle

Trunk angle was tracked using markers on the jugular notch, the second and the eighth of thoracic vertebral (Armand et al., 2014). For more detail, see the methods chapter (3.6.4). Once data was processed, the ensemble average of trunk angle over stance phase was calculated during walking for each participant. Trunk angle was then averaged over the stance phase for all trials and for each of the participants. This final outcome, mean trunk angle over stance phase, was used as the outcome measure.

Sagittal lower limb moments

For each subject, the ensemble average curves for sagittal hip, knee and ankle moment were calculated during normal walking. Next, mean over the specific window of stance phase (15–25%) was calculated; this window has been shown to correspond to the peak of knee contact force (Brandon et al., 2014; Sritharan et al., 2016). See methods section 3.7 for further justification of this choice of time window.

Muscle activity

As explained in the method section, a smoothed linear envelop signal which was normalised by the MVIC signal was created for each walking trial. MVIC was used in order to be able to compare the muscle activity between subjects (more details in section 3.5.12). Then for each subject, the ensemble average curves for each of the two hamstrings, quadriceps and gastrocnemius muscles were calculated during normal walking. Following this, the means of the biceps femoris, semitendinosus, vastus medialis, vastus lateralis and medial and lateral gastrocnemius muscle activity over the specific period of stance phase (10–20%) were calculated. Note that a window occurring slightly earlier than the 15-25% window (used for

the moment data) was chosen to account for electromechanical delay (see section 3.7.1 for more details).

Muscle co-contraction

In order to calculate knee muscle co-contraction during walking, a method that involved a separate summing of medial and lateral muscle activation was used (for more details see 2.5). Specifically, co-contraction was calculated as:

1. The sum of the quadriceps and the hamstring separately for medial/lateral muscles.
2. The sum of the quadriceps and the gastrocnemius separately for medial/lateral muscles.

An ensemble average curve was created for each of the four measures of co-contraction and this was then averaged across the 10–20% period of stance phase to give the final co-contraction outcome.

Sagittal lower limb angles

For each subject, the ensemble average curves for sagittal hip, knee and ankle angles were calculated during normal walking. Then the mean over the specific period of stance phase (15–25%) was then derived.

Spatiotemporal parameters

Speed and step length were calculated to check that the fundamental characteristics of walking were similar between the different groups.

4.3.3 Statistical analysis

A one-way analysis of variance (ANOVA) was used to determine if there any statistically significant differences between the means of the independent groups (knee OA, older and young) for each dependent outcome variable. In order to run this test, the data should be normally distributed. This was assessed by Shapiro-Wilk's test of normality ($P > .05$). There were no significant outliers in any tested variables, as checked by inspection of a box and whiskers boxplot. In addition, homogeneity of variances between groups was assessed using

Levene's test (P > .05). If the homogeneity of variances was violated, a modified version of the ANOVA (Welch ANOVA) was used.

If the ANOVA test showed a significant result, then a post hoc test, with a Tukey correction, was conducted to determine pairwise differences between each group. The Tukey post hoc test is an appropriate (Westfall, Tobias, & Wolfinger, 2011) and recommended (Field, 2013; Kirk, 2013) test for the purpose of testing pairwise combinations of levels of between-subject factors. If Welch ANOVA was used and the results of this test were statistically significant, a post hoc test with Games-Howell was conducted. If the data was not normally distributed or the assumption was violated, a Kruskal–Wallis test was conducted to determine whether there were statistically significant differences between any of the groups. Furthermore, if a Kruskal–Wallis test showed a significant result and the distributions of scores were similar for all groups, as assessed by visual inspection of a boxplot, then a post hoc test was conducted to determine pairwise differences between each group. Effect sizes were determined using the partial eta squared, for which.01, .06 and .14 are defined as small, medium and large respectively (Cohen, 1992; Richardson, 2011).

4.4 Results

4.4.1 Trunk angle

Figure 4-1 illustrates the differences in trunk angle during normal walking over the stance phase for all groups. As shown in Figure 4-1, people with knee OA walked with 3° more trunk inclination than both healthy groups. Table 4-2 displays the differences in trunk angle between groups during normal walking. The ANOVA result showed that there were significant differences between groups (P = .005). In addition, the post hoc test revealed that there was a statistically significant increase in trunk lean angle during walking in OA people compared to the older (P = .04) and young healthy subjects (P = .002) but no significant difference between the older and young healthy groups (P = .37).

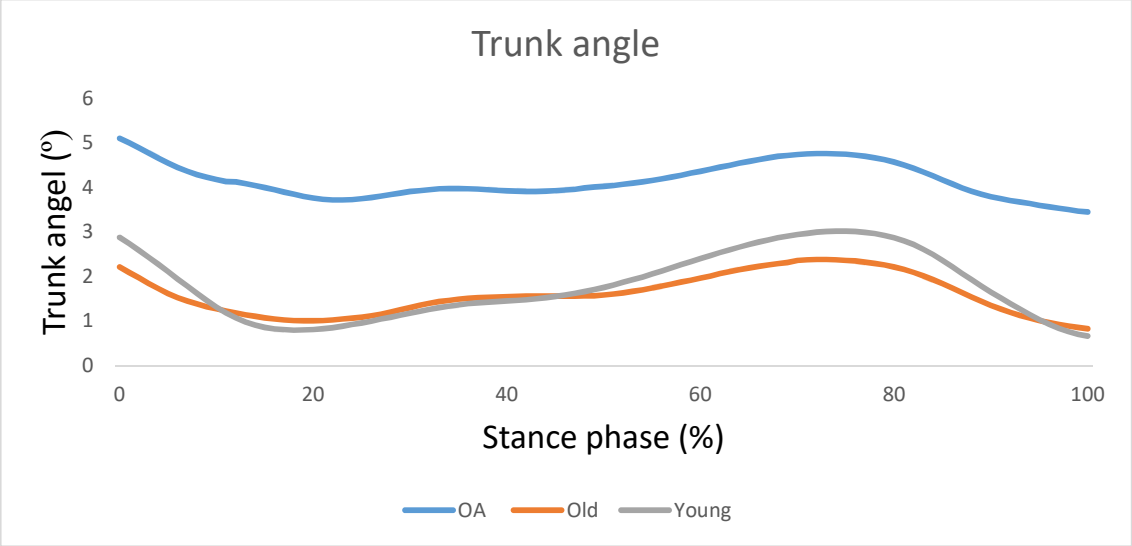


Figure 4-1 The ensemble average of the trunk angle for normal walking in people with knee OA, older and young healthy subjects.

Table 4-2 The mean and standard deviation (SD) of the trunk angle (°) during normal walking over the stance phase for all groups.

Variable	Knee OA	Older healthy	Young healthy	P-Value	ES
Trunk angle	4.2 (1.9)	2.4 (2.8)	1.8 (2.1)	.005	.17

4.4.2 Sagittal moment

Hip moment

Figure 4-2 illustrates the sagittal hip moment in people with knee OA, the older and young healthy subjects during walking across the stance phase. Looking at Figure 4-2, it is apparent that there are minimal differences in sagittal hip moment among groups. The Welch ANOVA analysis across the average of the 15–25% stance phase confirmed that there were no significant differences (P = .15) between the knee OA people, older and young healthy participants (Table 4-3).

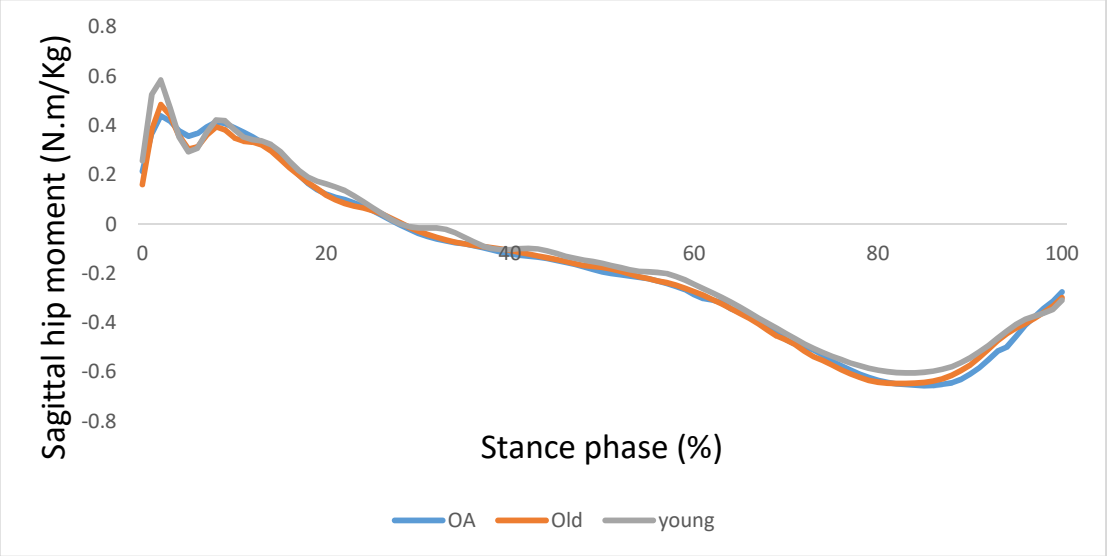


Figure 4-2 Ensemble average of sagittal hip moments during walking for knee OA, older and young healthy groups.

Knee moment

The sagittal knee moment ensemble average is plotted in Figure 4-3, which shows the differences between knee OA and healthy groups across stance phase. What stands out in the plots is that there appeared to be an increase in the peak of knee moment in the young healthy subjects compared to both other groups (Figure 4-3). However, this difference was not significant and there no significant differences (P = .20) in the average knee moment (across 15–25% stance phase) among groups (Table 4-3).

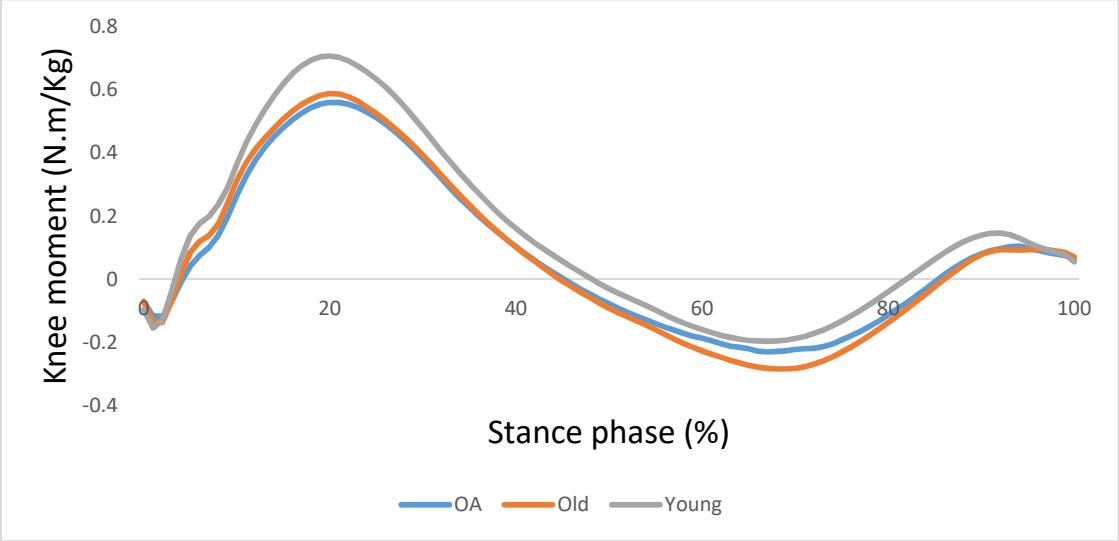


Figure 4-3 Ensemble average of sagittal knee moments during walking for knee OA, older and young healthy groups.

Ankle moment

Figure 4-4 shows sagittal ankle moment during walking in knee OA patients, older and young healthy subjects across stance phase. The plots show some evidence of an increase in plantarflexor moment in knee OA patients compared to the older healthy subjects in later stance phase (Figure 4-4). However, analysis of the 15–25% period of stance showed no significant differences (P = .13) in the sagittal ankle moment, as reported in Table 4-3.

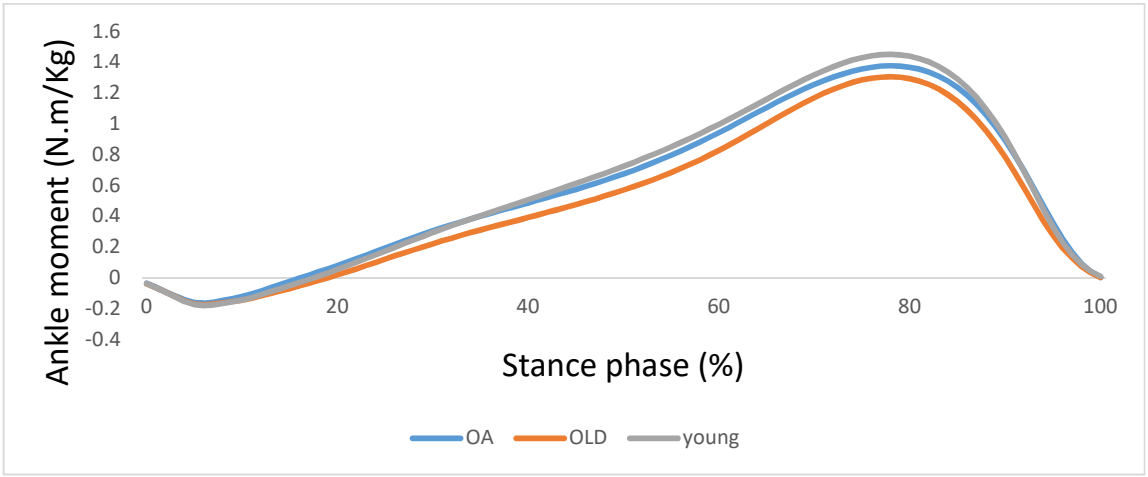


Figure 4-4 Ensemble average of sagittal ankle moments during walking for knee OA, older and young healthy groups.

Table 4-3 Mean (SD) p-values and effect sizes of the sagittal moment (Nm/kg) of hip, knee and ankle during walking across the average (15–25%) stance phase for individuals with knee OA, and older and young healthy subjects.

Variable	Group	Normal walking		
		Mean (SD) (Nm/kg)	p-value	Effect size
Hip moment	Knee OA	.15 (.19)	.15	.05
	Old	.14 (.17)		
	Young	.22 (.10)		
Knee moment	Knee OA	.52 (.27)	.20	.05
	Old	.55 (.21)		
	Young	.66 (.25)		
Ankle moment	Knee OA	.06 (.11)	.21	.06
	Old	.005 (.09)		
	Young	.03 (.06)		

4.4.3 Muscle activation

Medial gastrocnemius (MG)

Ensemble average profiles for MG muscle activity for all groups across the stance phase are set out in Figure 4-5. These plots clearly illustrate that MG activity was increased in both healthy groups during the 20–70% period. However, there appeared to be minimal differences over the early stance (Figure 4-5). In the period of interest (10–20% stance), there was no significant difference in muscle activity between all groups (P = .73, Table 4-4).

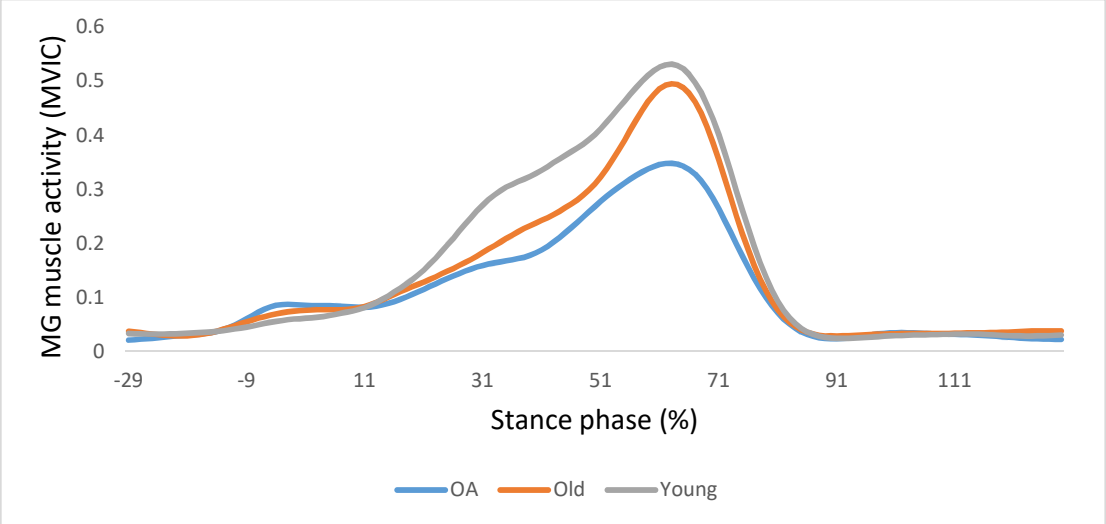


Figure 4-5 The ensemble mean average curves of MG muscle activity during walking across the stance phase for healthy (older and young) and knee OA groups.

Lateral gastrocnemius (LG)

From the graph below, we can see the ensemble average of the normalised LG muscle activity for people with knee OA, older and young healthy subjects across the stance phase (Figure 4-6). LG activity was higher in the knee OA group over the early- and mid-stance phases. Furthermore, the pattern of activity was increased from 60 to 90% of stances in both healthy groups compared to the knee OA group. In the period of interest (10–20% of stance), subjects in the knee OA group were observed to walk with ~25% higher muscle activity than the healthy group, however the Welch ANOVA test showed that the difference was not significant (P = .15, Table 4-4).

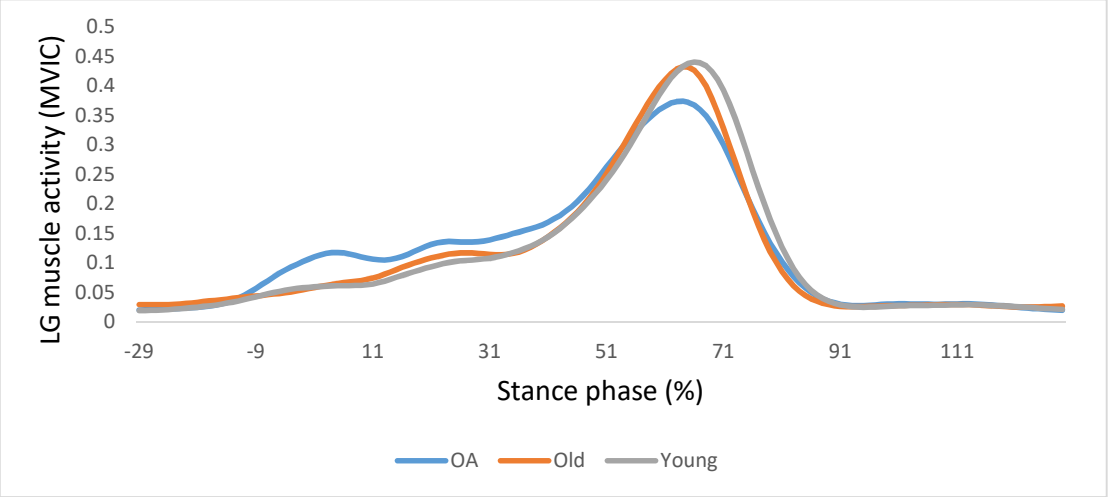


Figure 4-6 The ensemble mean average curves of LG muscle activity during walking across the stance phase for healthy (older and young) and knee OA groups.

Vastus medialis oblique (VMO)

The patterns of VMO muscle activity during walking in people with knee OA, older and young healthy subjects are presented in Figure 4-7. It is clear that VMO muscle activity increased from 10 to 90% of stances in people with knee OA compared to healthy groups. The ANOVA test showed that there were no differences (P = .07) between groups across the period of interest (10–20%). However, the difference was almost significant with a moderate effect size = .09, as shown in Table 4-4.

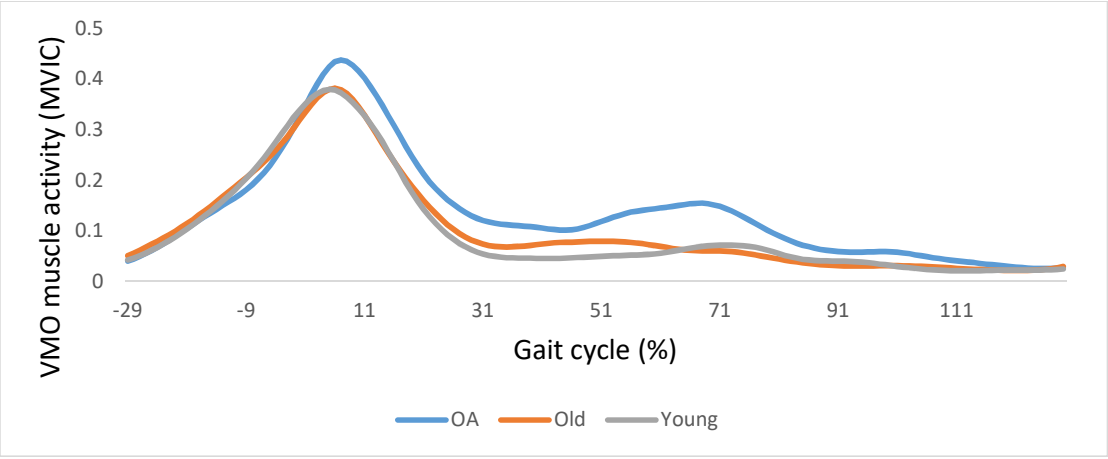


Figure 4-7 The ensemble mean average curves of VMO muscle activity during walking across the stance phase for healthy (older and young) and knee OA groups.

Vastus lateralis oblique (VLO)

The ensemble average curves in Figure 4-8 illustrate the characteristic pattern of VLO across the stance phase of walking. The plot illustrates the differences between the groups, with an overall higher activity in the knee OA group. However, these differences were more pronounced around early-stance and mid-stance. The analysis showed that although there was a difference between the groups across 10–20% stance, this did not reach significance (P = .10) (effect size = .08), as shown in Table 4-4.

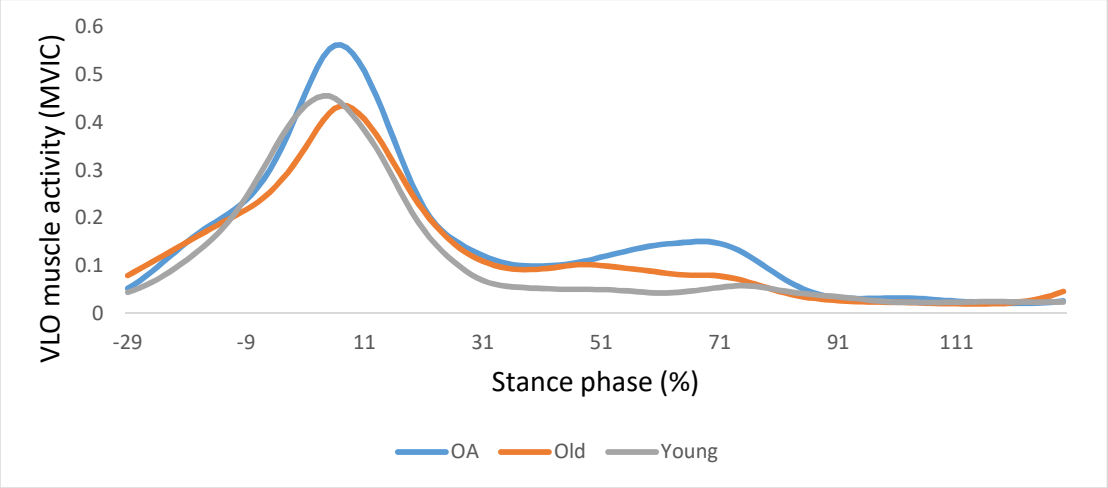


Figure 4-8 The ensemble mean average curves of VLO muscle activity during walking across the stance phase for healthy (older and young) and knee OA groups.

Semitendinosus (ST)

Figure 4-9 illustrates the differences in ST muscle activity in people with knee OA, older and young healthy subjects during walking across the stance phase. It can be observed from the plots below that the ST muscle activity was prolonged across the early-stance phase in people with knee OA compared to both healthy groups (Figure 4-9). Although there was an increase of approximately 50% in ST muscle activity across 10–20% of stance in OA patients compared to healthy subjects, the Kruskal–Wallis test revealed that these differences did not reach significance (P = .15). This result is presented in Table 4-4.

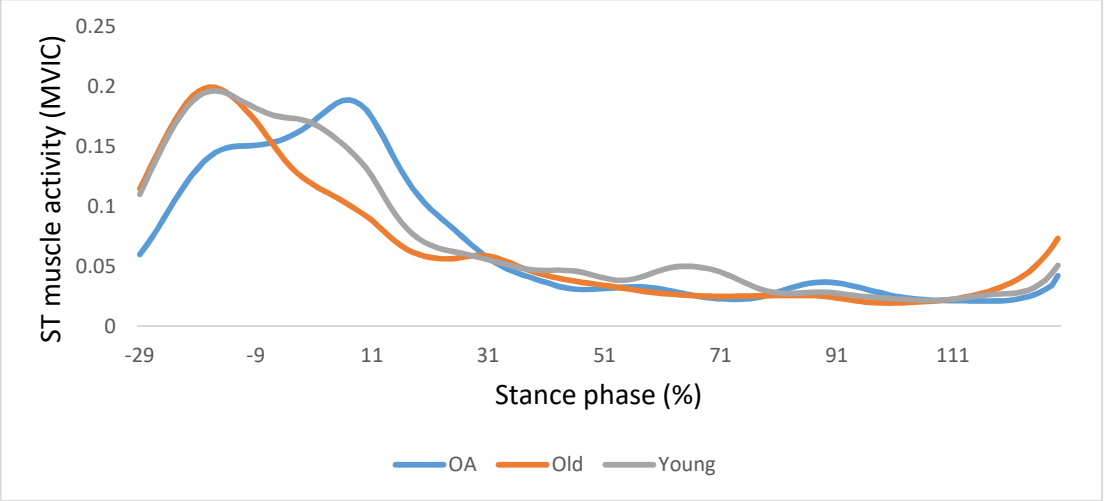


Figure 4-9 The ensemble mean average curves of ST muscle activity during walking across the stance phase for healthy (older and young) and knee OA groups.

Biceps femoris (BF)

The plots below show the ensemble average of the normalised BF muscle activity for people with knee OA, older and young healthy subjects across the stance phase (Figure 4-10). This plot illustrates clearly that BF activity was higher in the knee OA group for almost all of the stance phase. Nevertheless, the pattern of activity was similar between the groups from 60–100% of gait. Statistically, in the period of interest at 10–20% of stance, the Kruskal–Wallis test revealed that there was a significant difference between the groups (P = .008). In addition, the post hoc test revealed that there was a statistically significant increase in BF muscle activity in the knee OA group compared to the older healthy and young healthy groups (P = .01 and 0.02, respectively). This result is illustrated in Table 4-4.

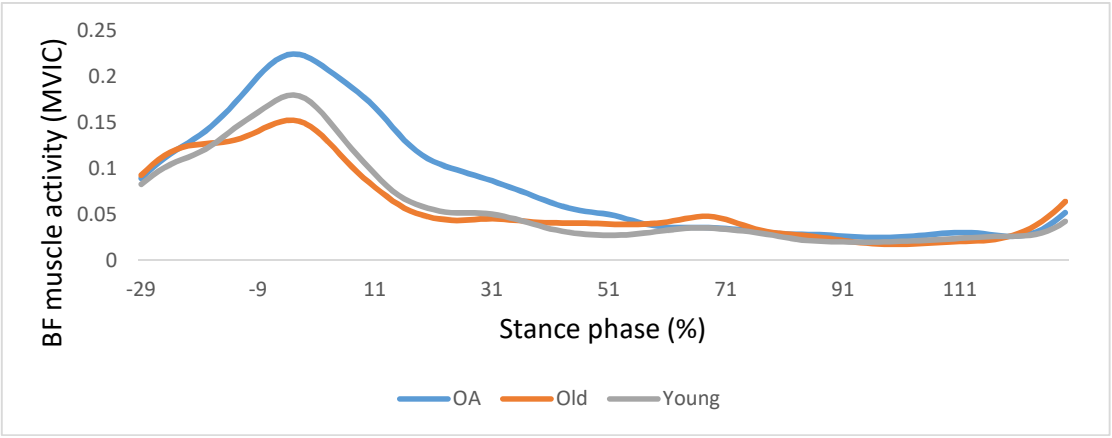


Figure 4-10 The ensemble mean average curves of BF muscle activity during walking across the stance phase for healthy (older and young) and knee OA groups.

*Table 4-4 Means, (SD), p-values and effect sizes of gastrocnemius, quadricep and hamstring activations during walking across the average (10–20%) stance phase for individuals with knee OA, older and young healthy subjects. (MVIC: proportion of the MVIC) * indicates a significant result.*

VARIABLE	GROUP	NORMAL WALKING		
		Mean (SD) (MVIC)	p-value	Effect size
MG	Knee OA	.08 (.06)	**.73**	**.01**
	Old	.10 (.08)		
	Young	.10 (.05)		
LG	Knee OA	.10 (.06)	**.12**	**.06**
	Old	.08 (.05)		
	Young	.07 (.03)		
VMO	Knee OA	.33 (.15)	**.07**	**.09**
	Old	.22 (.13)		
	Young	.26 (.16)		
VLO	Knee OA	.40 (.20)	**.10**	**.08**
	Old	.29 (.14)		
	Young	.30 (.18)		
ST	Knee OA	.16 (.13)	**.15**	**.12**
	Old	.08 (.06)		
	Young	.09 (.05)		
BF	Knee OA	.14 (.10)	**.008***	**.23**
	Old	.06 (.04)		
	Young	.06 (.03)		

4.4.4 Muscle co-contraction

Biceps femoris and vastus lateralis oblique (BFVLO) co-contraction

As Table 4-5 shows, there was a significant difference (P = .01) between the groups over the 10–20% stance. In addition, the post hoc test showed that there was a significant increase of BFVLO co-contraction between OA and the older group, and between OA and the young group (P = .02 and .03, respectively).

Lateral gastrocnemius and vastus lateralis oblique (LGVLO) co-contraction

Even though there was trend of an increase of approximately 25% in LGVLO co-contraction in the knee OA group in comparison with healthy groups, ANOVA analysis indicated that there were no significant differences among groups (P = .05), as can be seen in Table 4-5.

Semitendinosus and vastus medialis oblique (STVMO) co-contraction

The ANOVA test showed that there were significant differences (P = .01) in the STVMO co-contraction between groups. Specifically, a Tukey post hoc test revealed that there was a significant difference (P = .01) between the knee OA and older healthy groups. In addition, results showed that there was a significant increase in the STVMO co-contraction between the knee OA and young healthy subjects (P = .04, Table 4-5).

Medial gastrocnemius and vastus medialis oblique (MGVMO) co-contraction

The results of the MGVMO co-contraction between all groups are illustrated in Table 4-5. A non-parametric test revealed that there were no significant differences in MGVMO co-contraction between groups (Table 4-5).

*Table 4-5 Means, (SD), p-values and effect sizes of gastrocnemius, quadricep and hamstring co-contraction during walking across the average (10–20%) stance phase for individuals with knee OA, the older and young healthy subjects. (MVIC: proportion of the MVIC) * indicates a significant result.*

VARIABLES	GROUP	NORMAL WALKING		
		Mean (SD) (MVIC)	p-value	Effect size
BFVLO	Knee OA	.54 (.27)	**.01***	**.15**
	Old	.35 (.18)		
	Young	.37 (.18)		
LGVLO	Knee OA	.51 (.21)	**.05**	**.098**
	Old	.37 (.17)		
	Young	.38 (.19)		
STVMO	Knee OA	.50 (.24)	**.01***	**.15**
	Old	.31 (.15)		
	Young	.35 (.16)		
MGVMO	Knee OA	.41 (.17)	**.14**	**.06**
	Old	.32 (.15)		
	Young	.36 (.15)		

4.4.5 Sagittal kinematics

Sagittal hip angle

Figure 4-11 shows the sagittal hip angle in people with knee OA, older and young healthy subjects across the gait cycle. Looking at Figure 4-11, it is apparent that there are no differences in hip angle among groups. No significant difference between the three groups was evident over the 15–25% period (P = .19), as reported in Table 4-6.

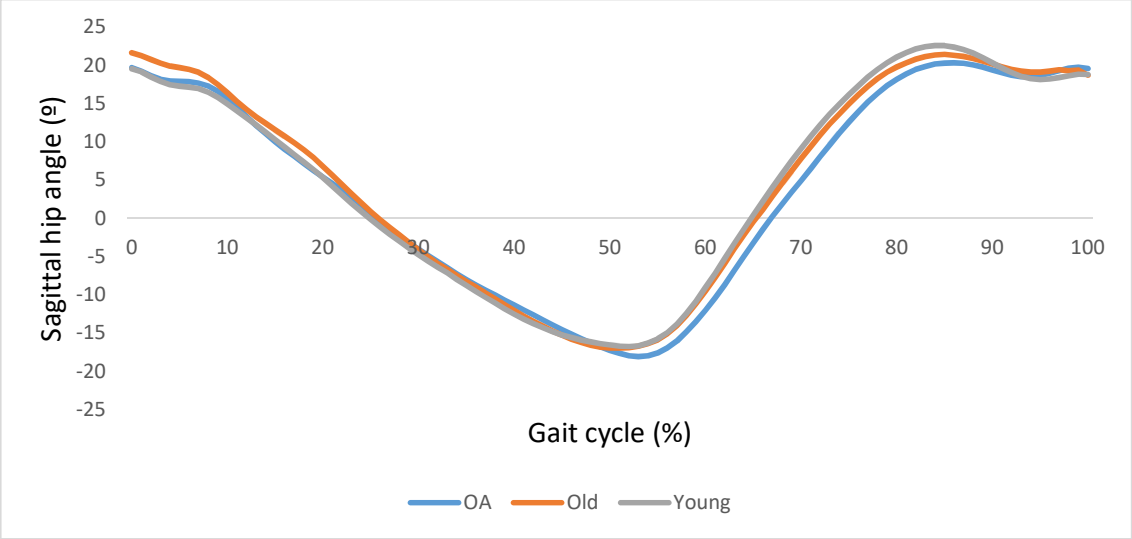


Figure 4-11 The ensemble average of sagittal hip angle for normal walking in people with knee OA, older and young healthy subjects.

Sagittal knee angle

The plots below in Figure 4-12 illustrate the ensemble average of the sagittal knee angle for the three groups during gait cycle. In general, the data shows that there is an increase in the magnitude of the knee flexion angle across the whole gait cycle in the young healthy group compared to the other groups (Figure 4-12). In the period of interest, a non-parametric test showed that there were significant differences between groups (P = .01) (Table 4-6). In addition, a post hoc comparison revealed that there was a significant increase in knee flexion angle in the healthy young group compared to knee OA (P = .01, Table 4-6).

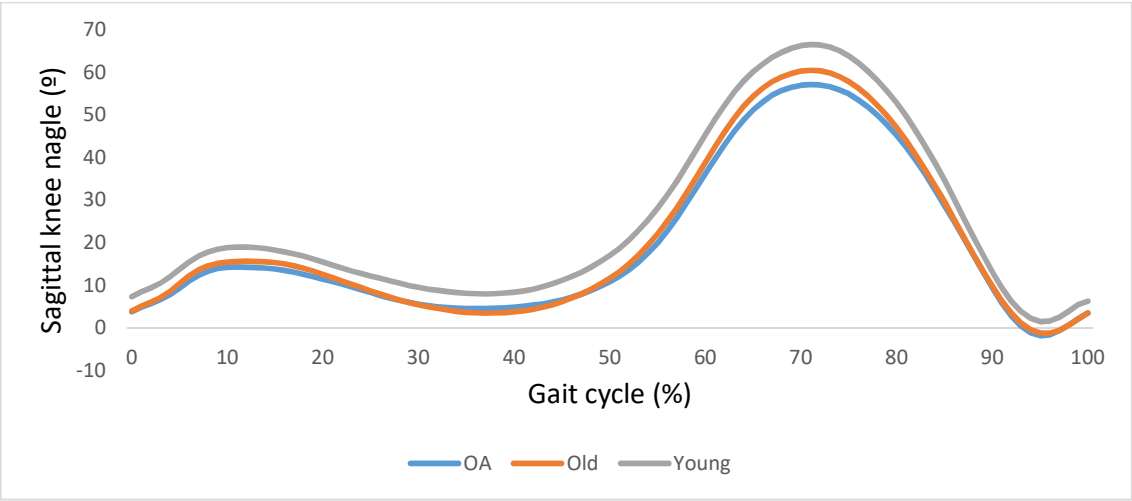


Figure 4-12 The ensemble average of sagittal knee angle for normal walking in people with knee OA, the older and young healthy subjects.

Sagittal ankle angle

Figure 4-13 shows the sagittal ankle angle in people with knee OA, older and young healthy subjects across the gait cycle during walking. Descriptive results for the time period 15–25% stance phase are presented in Table 4-6, and confirm no significant difference between groups over this period, though the p-value was almost significant (P = .051).

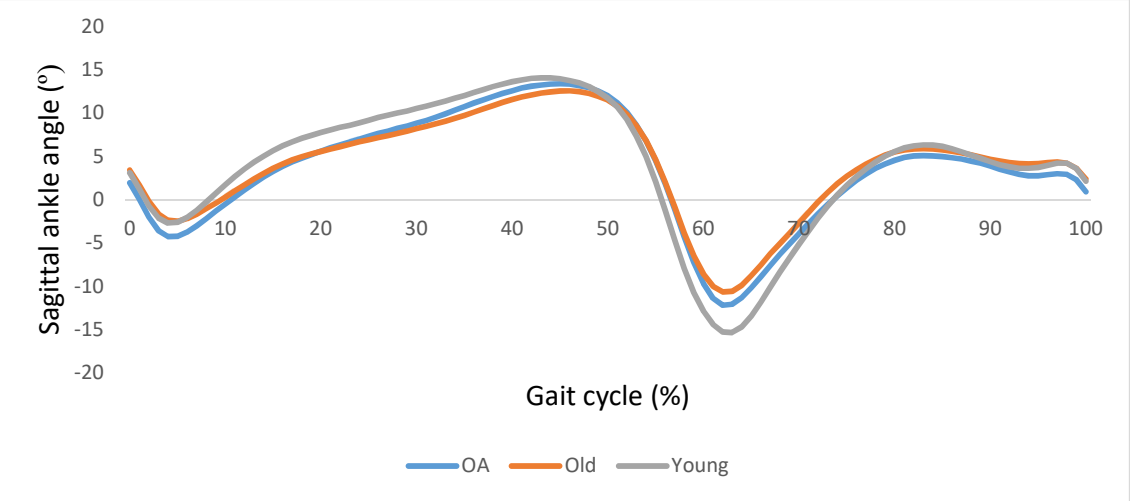


Figure 4-13 The ensemble average of sagittal ankle angle for normal walking in people with knee OA, older and young healthy subjects.

*Table 4-6 Means, (SD), p-values and effect sizes of the sagittal hip, knee and ankle angles (°) during walking across the average (15–25%) stance phase for individuals with knee OA, older and young healthy subjects. * indicates a significant result.*

VARIABLES	GROUP	NORMAL WALKING		
		Mean (SD) (°)	p-value	Effect size
HIP ANGLE	Knee OA	4.1 (4.5)	**.19**	**.06**
	Old	6.6 (3.8)		
	Young	5.4 (4.3)		
KNEE ANGLE	Knee OA	11.5 (5.8)	**.01***	**.097**
	Old	12.3 (4.8)		
	Young	15.4 (5.0)		
ANKLE ANGLE	Knee OA	5.6 (3.6)	**.051**	**.12**
	Old	5.5 (2.8)		
	Young	7.7 (2.0)		

4.4.6 Spatiotemporal parameter

The data in Table 4-7 indicates the differences in spatiotemporal parameters in individuals with knee OA, older and young healthy subjects during walking. A non-parametric test showed that there were no significant differences in speed and step length among the three groups, as presented in Table 4-7.

Table 4-7 Summary result of step length (m) and speed (m/s) during walking for individuals with knee OA, older and young healthy subjects.

VARIABLES	GROUP	NORMAL WALKING	
		Mean (SD)	p-value
SPEED	Knee OA	1.18 (.11)	**.58**
	Old	1.20 (.16)	
	Young	1.21 (.09)	
STEP LENGTH	Knee OA	1.28 (.08)	**.65**
	Old	1.29 (.12)	
	Young	1.32 (.10)	

4.5 Discussion

4.5.1 Overview

The first study in this thesis was designed to characterise differences in kinematics, kinetics, muscle activation and muscle co-contraction between individuals with knee OA, healthy older and healthy young people during walking. The 15–25% period of stance phase was selected for the kinematics/kinetic data, and the 10–20% period for EMG data, as this corresponds to the period of peak knee joint loading during walking (see section 3.7). The data showed the knee OA group to walk with an increase in trunk inclination of approximately 3° more than healthy participants (both the older and young groups). In addition, people with knee OA walked with higher BF muscle activity, BFVLO co-contraction and STVMO co-contraction in comparison with both healthy groups. Moreover, the results showed that there was a trend towards a statistical difference in ST muscle activity, and that of both quadriceps. Furthermore, it was observed that the young healthy subjects walked with more knee flexion angle than the knee OA group. However, importantly there were no other differences between the young and the older healthy groups. Furthermore, no differences were observed in lower limb joint moments or spatiotemporal parameters between the three groups. The discussion will contrast the findings of the current study to those reported in other research

comparing people with knee OA to healthy controls. Then the conclusions and limitations of this study will be discussed.

4.5.2 Kinematics and spatiotemporal parameters

These results in sagittal trunk angle during walking in the OA and older groups are consistent with a previous study which found that OA patients walk with 2.5-3° more forward lean than matched healthy controls (Preece et al., 2018). This current study result also showed that patients with knee OA tend to walk with approximately 3° more lean than the healthy groups but no difference between younger and older participants. To my knowledge, there have been no previous studies which have investigated differences in sagittal trunk angle during walking between younger and older groups. Taken together, these findings from this and the previous study, demonstrate that patients with knee OA walk with increased sagittal trunk inclination. In subsequent chapter, the potential biomechanical effect of this increased trunk lean is explored.

The results of this current study showed minimal differences in lower limb kinematics between knee OA patients and asymptomatic groups. However, reduced knee flexion compared to young healthy people was observed, a finding which is in line with previous studies (Astephen, Deluzio, Caldwell, & Dunbar, 2008). Interestingly, other studies have reported that people with knee OA walk with a greater knee flexion angle compared to matched healthy controls (Baliunas et al., 2002; Childs et al., 2004), whereas others found no differences in the sagittal knee angle between both healthy groups (Duffell et al., 2017; Schloemer, Thompson, Silder, Thelen, & Siston, 2017). These different findings indicate that biomechanical alterations with knee OA are not consistently characterised by altered kinematic patterns.

Our results found that there was no difference in the sagittal hip and ankle angles across all groups. The finding on hip angle is in agreement with (Duffell et al., 2017; Mezghani et al., 2017), who studied the differences in kinematics between knee OA and matched healthy people and with (Duffell et al., 2017; Schloemer et al., 2017), who found no differences in the sagittal hip angle between young and older healthy people when walking. Furthermore, a previous study supports our results for sagittal ankle angle with no differences among all

groups (Duffell et al., 2017). Together, these results again indicate minimal differences in lower limb biomechanics between knee OA and healthy people.

In addition to kinematics findings, our result demonstrated no differences in spatiotemporal parameters between the groups. Although these results are in line with other studies (Landry et al., 2007; Mundermann et al., 2005) which found no differences in self-selected walking speeds among individuals with knee OA and healthy counterparts, there are other studies which have shown that individuals with knee OA walk with a slow speed and a shorter step length (Al-Zahrani & Bakheit, 2002; Kaufman et al., 2001; Messier et al., 2005). It is possible that the difference between these findings and those reported in this chapter are the result of differences in severity of OA between the different research studies.

4.5.3 Kinetics

These results showed a small, but not significant differences, increase in sagittal hip moment in the knee OA group compared to the older healthy group. This is consistent with (Duffell et al., 2017) who studied the early mid-stance phase. However, a study by Astephen, Deluzio, Caldwell, and Dunbar (2008) reported that hip extensor moment was reduced at the early stance in a group with severe OA compared to healthy subjects. In contrast to this finding, previous research found that people with bilateral knee OA walk with higher hip-extensor moment in the early mid-stance phase (Huang et al., 2008; Liu et al., 2014), a finding which is, to some degree, in in with the findings of this current study. However, it is not clear whether these inconsistent findings in sagittal hip moment are the result of differences in the severity of OA between the different research studies.

This current study did not show differences in sagittal knee moment between groups. This is consistent with previous studies (Baliunas et al., 2002) which found no differences in sagittal knee moment between OA and healthy matched groups. In addition, Duffell et al. (2017) showed no differences in sagittal knee moment among young, older and OA participants (Duffell et al., 2017). However, some studies have shown a significant reduction in the first peak of the sagittal knee moment in people with knee OA (Astephen, Deluzio, Caldwell, & Dunbar, 2008; Huang et al., 2008; Kaufman et al., 2001). Nevertheless, these studies were focused on the peak knee moment, and not the mean knee moments across the early mid-stance phase which were used in this thesis and this could explain the difference in findings.

The result from this study showed there was a trend for a difference in the sagittal ankle moment across the 15–25% period compared to the older healthy group; however this was not statistically significant. These results agree with the findings of other studies that indicated no significant change in sagittal ankle moment between moderate knee OA and matched healthy groups (Astephen, Deluzio, Caldwell, & Dunbar, 2008). However, other authors have found ankle moment to be higher in a severely affected group when compared to a healthy group (Liu et al., 2014). In general the results of this current study, and other studies, show larger ankle moments in individuals with knee OA later in the gait cycle but no difference during the period of interest (10–20%).

4.5.4 Muscle activation

The results, presented in the section above, show indicate alterations in neuromuscular in individuals with knee OA in the quadriceps, gastrocnemius and hamstrings muscles but no differences between the healthy groups. Specifically, there was a non-significant increase in quadriceps muscle activation in people with knee OA over 10–20% of stance phase, with reasonably large effect sizes. Although non-significant, this result is consistent with previous studies reported that the quadriceps muscles were greater in the knee OA group compared to asymptomatic young and older healthy people over the mid-stance phase (Rutherford et al., 2017). It is also consistent with studies showing larger quadriceps activity in people with knee OA when compared to age-matched controls (Hodges et al., 2016a; Hubley-Kozey et al., 2006). Nevertheless, the magnitude of the larger activity in the quadriceps appeared to be less than in the other muscles, investigated in this study, and this may explain the lack of statistical significance.

The findings in this study indicate that the biceps femoris muscle activity was significantly larger in people with knee OA over the 10–20% stance period when compared to both groups of healthy subjects. There was also a trend for semitendinosus muscle activity to be larger in people with knee OA by 50%, but the result was not significant. This finding is in agreement with previous studies that reported people with knee OA walked with increased biceps femoris muscle activity compared to matched healthy subjects (Heiden et al., 2009; Hubley-Kozey et al., 2006; Rutherford, Hubley-Kozey, Stanish, et al., 2011) and to the young healthy subjects (Rutherford et al., 2017). Moreover, both muscles were prolonged, with a rapid

decrease in the older and young healthy groups through the early- and mid-stance phases, which matches observations from earlier studies (Hubley-Kozey et al., 2006; Rutherford et al., 2017). Taken together, these findings support the idea that increased hamstring activity is a clear gait characteristic of people with knee OA.

For the gastrocnemius muscles, there were no significant differences between the groups across the 10–20% stance period. The present findings seem to be consistent with other research which found no differences in gastrocnemius amplitude between moderate knee OA and older healthy subjects (Hubley-Kozey et al., 2006; Rutherford et al., 2013). In addition, these results are consistent with those of other studies which indicate no differences in gastrocnemius muscle between older and young healthy people (Schmitz, Silder, Heiderscheit, Mahoney, & Thelen, 2009). However, there was a trend for lateral gastrocnemius to be larger in people with knee OA (Table 4-4). This is consistent with, other research which has shown larger gastrocnemius activity in people with knee OA when compared to matched healthy subjects (Sritharan et al., 2016). Similar to the quadriceps muscles, these findings, along with the data presented here, suggest that, during early stance, there could be small differences in gastrocnemius activity which are characteristic of knee OA but which may not lead to statistical significance.

4.5.5 Muscle co-contraction

In this study the co-contraction between the BFVLO, STVMO, MGVMO and LGVLO was compared between the groups across the period 10–20% stance phase. All pairs involving hamstrings and quadriceps saw a significant increase in the OA patients compared to both healthy groups. Furthermore, there was a tendency for the pairs involving gastrocnemius and quadriceps to be increased in knee OA group, although this was not significant. In addition, our results showed no differences in muscle co-contraction among the healthy groups, which is consistent with the data of (Rudolph et al., 2007). Furthermore, the findings for BFVLO co-contraction were in line with other studies which showed that knee OA patients exhibited greater lateral (BFVLO) co-contraction during the mid-stance phase compared with a matched healthy cohort (Heiden et al., 2009; Hortobagyi et al., 2005; Hubley-Kozey, Hill, Rutherford, Dunbar, & Stanish, 2009b). The result for STVMO co-contraction also agrees with other studies, showing that OA people exhibit a tendency towards greater STVMO co-contraction

during early- and mid-stance compared to healthy people (Hubley-Kozey, Hill, Rutherford, Dunbar, & Stanish, 2009a; Lewek et al., 2004a; Schmitt & Rudolph, 2008). Taken together, previous research along with the data presented in this thesis indicates that people with knee OA walk with increased co-contraction in the hamstrings and quadriceps.

4.5.6 Limitation

There are two main limitations in this study, which should be acknowledged. Firstly, the technique of using MVICs has been questioned. Although EMG normalisation is essential to make comparisons between groups, it has been proposed that the ability to perform MVIC in injured patients can be affected by pain (Benoit et al., 2003). Given that people with knee OA had higher pain levels and lower physical function (Kidd, 2006; Peat et al., 2001), then, these patients might not be able to produce a representative MVIC. However, in this study, verbal encouragement was provided and a series of muscle contractions utilised in an attempt to elicit maximum activation (Rutherford, Hubley-Kozey, & Stanish, 2011a) and ensure that these patients could recruit their muscles to similar percentages of maximum as the healthy controls. In addition, the majority of the previous studies on knee OA patients followed an MVIC approach (Heiden et al., 2009; Hortobagyi et al., 2005; Lewek et al., 2004b), and good reliability was reported for MVIC in those patients (Hubley-Kozey, Robbins, et al., 2013). Therefore, in order to be consistent with previous research, these procedures were adopted to ensure validity and provided confidence in the MVIC data.

Another limitation of the current study was that effect of severity of knee OA was not investigated. Only one group of subjects with knee OA were used in this study, rather than separating subjects by severity. It is therefore not clear whether the observed differences would be the same in severe knee OA patients. Nevertheless, previous research has investigated differences between patients with OA and different severity levels. These data have been discussed above and is generally consistent with our observations of increased muscle activation patterns in people with knee OA.

4.5.7 Conclusion and clinical relevance

This study set out to determine the effect of age and knee OA on knee muscle activation patterns, co-contraction and knee-joint biomechanics during walking. The main findings in this study were:

- Knee OA patients walk with an increased sagittal plane inclination of the trunk in comparison with healthy older and young participants.
- Knee OA patients had minimal differences in lower limb kinematics and kinetics when compared to age-matched and young healthy groups.
- Knee OA patients walked with higher hamstrings muscle activations.
- Medial and lateral hamstring-quadriceps co-contraction was increased in knee OA patients.
- There were minimal differences in lower limb biomechanics between the young and older groups.

The results of this study clearly demonstrates that people with knee OA walk with an increased sagittal inclination of the trunk, or forward lean, an increase in hamstring muscle activity along with a corresponding increase in hamstring-quadriceps co-contraction. Also it is possible that these muscle coordination changes are a direct response to the disease, and it is possible that they are, in some way, linked to altered sagittal plane trunk inclination. Current conservative management for individuals with knee OA aims to reduce pain and stiffness and improve muscle strength and range of motion (Bartholdy et al., 2017; Bennell et al., 2016; Fransen et al., 2015). However, given the link between co-contraction and loading, research is needed into other conservative managements which have the potential to change co-contraction and therefore loading in those with knee OA. In the two following chapters, the potential impact of increased trunk lean is investigated using a within subject interventional-type study, first in healthy individuals (Chapter 5) and then in people with knee OA (Chapter 6).

Chapter Five

(Study two)

What is the biomechanical effect of instructing young healthy people to walk with increased/decreased trunk inclination?

5.1 Introduction

Data presented in the previous chapter showed that people with knee OA walk with an increased flexion of the trunk. These findings are consistent with other recent research which has demonstrated that people with knee OA walk with an increase in the sagittal plane inclination of the trunk (Preece et al., 2018). Increased trunk inclination, or forward lean, has the potential to alter the position of the GFR vector relative to lower limb joint centres and this, in turn, may lead to change in lower limb moments and/or muscle activation patterns during walking. Interestingly, previous research has shown small differences in habitual trunk lean to be associated with differences in the characteristics of the hip extensor moment during walking (Leteneur et al., 2009). However, there is minimal knowledge about how alterations in forward lean could affect muscle activation patterns during walking.

Data in the previous chapter also showed the people with knee OA walk with increases hamstring activation and increased co-contraction during the early phase of stance. Again, this research corroborates the findings of other previous research, which has demonstrated changes in muscle activation patterns in people with knee OA (Astephen, Deluzio, Caldwell, & Dunbar, 2008; Chang et al., 2015; Hubley-Kozey et al., 2009a; Lewek et al., 2004a). To date, numerous explanations have been put forward to explain these altered muscle patterns, such as to increase stability (Childs et al., 2004) or as a localised muscular response to unload the medial knee joint (Andriacchi, 1994) see section 2.8.1 in the literature review. However, as explained in the literature review (Section 2.9), it is possible that these altered muscle patterns are, to some degree, the result of an increased forward lean of the trunk. To understand this idea further, it is useful to explore the effects of instructing young healthy

people to walk with an increase in trunk forward lean. As these people have no disease, or any age-related impairments, then the observed muscular effects can be attributed directly to the change in upper body position rather than a response to the disease or to changes which are associated with older age.

5.2 Research questions and hypotheses

The main objective of this study was to understand the biomechanical effect of instructing young healthy people to walk with increased/decreased trunk forward lean.

This objective was achieved through five separate research questions:

Q 1 What is the effect of increasing/decreasing trunk inclination on hip, knee and ankle moments in young healthy people during walking?

H 1 The hip extensor moment and ankle planter flexor moment will be increased and decreased when walking with increased and decreased trunk inclination, respectively.

Q 2 What is the effect of increasing/decreasing trunk inclination on gastrocnemius, quadriceps and hamstring muscle activities in young healthy people during walking?

H 2 The gastrocnemius, quadriceps and hamstring muscle activities will be increased and decreased when walking with increased and decreased trunk inclination, respectively.

Q 3 What is the effect of increasing/decreasing trunk inclination on gastrocnemius, quadriceps and hamstring muscles co-contraction in young healthy people during walking?

H 3 The gastrocnemius, quadriceps and hamstring co-contraction will be increased and decreased when walking with increased and decreased trunk inclination, respectively.

Q 4 What is the effect of increasing/decreasing trunk inclination on hip, knee and ankle angles in young healthy people during walking?

H 4 The hip flexion angle and ankle plantarflexion angle will be increased and decreased when walking with increased and decreased trunk inclination, respectively.

Q 5 What is the effect of increasing/decreasing trunk inclination on spatiotemporal in young healthy people during walking?

H 5 There will be no differences in spatiotemporal parameters when walking with increased or decreased trunk inclination.

5.3 Method

The aim of this study was to investigate the effect of imposed trunk lean (+5^{o}, +10^{o} and -5^{o}) on lower limb kinetics, kinematics muscle activation and co-contraction in young healthy participants during walking. A full description of the protocol for this measurement was provided in the method chapter and is only summarised here briefly. The right or left leg were randomly selected by a MATLAB custom programme. Following the collection of the normal walking trials, trunk angle was calculated and used as a guide for the subsequent trunk angles. Using the biofeedback approach described in section 3.5.11, participants were instructed to walk with (+5^{o}, +10^{o} and -5^{o}) of their normal trunk position. A minim of five successful trials (within +/-2^{o} of the target flexion angle) were collected at self-selected speed. In the following paragraphs, sample characteristic, derivation of outcome measures and the appropriate statistical tests for this study are presented.

5.3.1 Sample and participants

A total of 20 young healthy individuals ranging from 18-40 years were recruited to take a part in the study. The criteria for inclusion and exclusion for the subjects in this study are given in Section 3.2. The participants were required to attend the gait laboratory for one session. The subject's characteristics are presented in Table 5-1.

Table 5-1 Participants' characteristics for the young healthy people. Values are the mean ± Standard Deviation (SD).

Variables	Young healthy people
No. of participants	20
Age (Years)	26.1 (6.8)
Height (M)	1.7 (.07)
Mass (Kg)	66.1 (8)
Body mass index (kg/m2)	22.2 (2.5)
Tested leg	12 Right and 8 left
Gender (M/F)	11/9

5.3.2 Derivation of outcome measures

For each participant, the ensemble average curves for sagittal hip, knee and ankle kinematics and kinetic were calculated during normal walking and in the different trunk lean conditions (+5^{o}, +10^{o} and -5^{o}). Then mean over the specific window of stance phase (15–25%) was calculated for each kinematic/kinetic trajectory as this window has been shown to correspond to the peak of knee contact force (Brandon et al., 2014; Sritharan et al., 2016). In addition, for each participant, the ensemble average curves for each of the two hamstrings, quadriceps and gastrocnemius muscles were calculated during normal walking and in the different trunk lean conditions. For these muscle data, the mean over the 10–20% period of stance phase was calculated. This window was chosen to account for electromechanical delay (see section 3.7.1 for more details). In order to calculate knee muscle co-contraction during walking and in the different trunk lean conditions, a separate summing of medial and lateral muscle activation was used (for more details see 2.5) then averaged across the 10–20% period of stance phase to give the final co-contraction outcome. More details on these calculations

along with the processing of the raw EMG, motion and force data were explained in detailed in the method chapter, Section 3.6.

5.3.3 Statistical methods

In order to answer each of the separate research questions, the following statistical tests were performed:

One-way repeated ANOVA and the Friedman test

A one-way repeated measures ANOVA was conducted to determine whether there were statistically significant differences between any of the different trunk lean conditions. In addition, if the ANOVA test showed a significant result, then a post hoc test, with a Bonferroni correction, was conducted to determine pairwise differences between trunk lean conditions. The Bonferroni test is a good (Maxwell, 1980) and recommended test (Bailey, 2005; Field, 2013) for the purpose of testing pairwise comparison for one-way repeated measures ANOVA. To check data was appropriate for an ANOVA test, the normally distributed and assumption of sphericity were tested by Shapiro-Wilk test ($P > .05$) and Mauchly's test of sphericity test ($P > .05$), respectively. In addition, in order to use the one way repeated ANOVA test there should be no significant outliers in any level of the within-subjects factor as assessed by inspection of a boxplot. However, if the data was not normally distributed or the assumption was violated, a Friedman test was conducted to determine whether there were statistically significant differences between any of the different trunk lean conditions. Furthermore, if Friedman test showed a significant result, then a post hoc test was conducted to determine pairwise differences between trunk lean conditions.

Linear mixed model (LMM)

Linear mixed effects analysis was performed using the lme4 package (Bates, Mächler, Bolker, & Walker, 2014) in R (Team, 2017) to determine the effect of trunk angle on each variable. Trunk angle was defined as a fixed effect, where a fixed effect is one that is expected to have an effect on the outcome variable. The model also included Subject as a random effect. Random intercepts were assumed for each subject thus resolving the non-independence problem, of multiple measures from the same subject, and accounting for baseline

differences in trunk angle. In addition, the model assumes by-subject random slopes for the effect of the outcome variable; meaning the model can expect the effect of the outcome variable to be different for different subjects. The linear mixed model is thus defined as follows:

outcome variable ~ Trunk Angle + (1 + Trunk Angle | Subject)

where trunk angle is the fixed effect, the | Subject implies random intercepts and the 1 + Trunk Angle dictates the by-subject random slopes.

No obvious deviations from homoscedasticity or normality were identified by visual inspection of the residual plots. To test the significance of the fixed effect (i.e. that outcome variable would be linearly dependent on trunk angle) a Likelihood Ratio Test was used to compare two models, one with the fixed effect of interest (full model) and one without the fixed effect of interest (reduced model). This was done using ANOVA (reduced model, full model), where a significant result implies the missing fixed effect (Trunk Angle) has a significant effect on the outcome variable.

Full model:

outcome ~ Trunk Angle + (1 + Trunk Angle | Subject)

Reduced model:

outcome ~ 1 + (1 + Trunk Angle | Subject)

where trunk angle is the fixed effect, the | Subject implies random intercepts and the 1 + Trunk Angle dictates the by-subject random slopes.

5.4 Result

5.4.1 Trunk angle

Figure 5-1 illustrates the differences in trunk angle during normal walking and with different trunk inclinations: normal -5^{o}, normal +5^{o} and normal +10^{o} flexion (each different trunk angle is shown in a different colour) over the stance phase for 20 subjects. The bold lines represent the mean across the 20 subjects and the shaded area provide an indication of the standard

deviation across all 20 subjects. As explained in method chapter, trials were only accepted if they were within +/-2 degrees of the target flexion angle. These data demonstrate that it was possible, using the biofeedback approach described in the methods section, to ensure that all trunk lean conditions were tightly constrained.

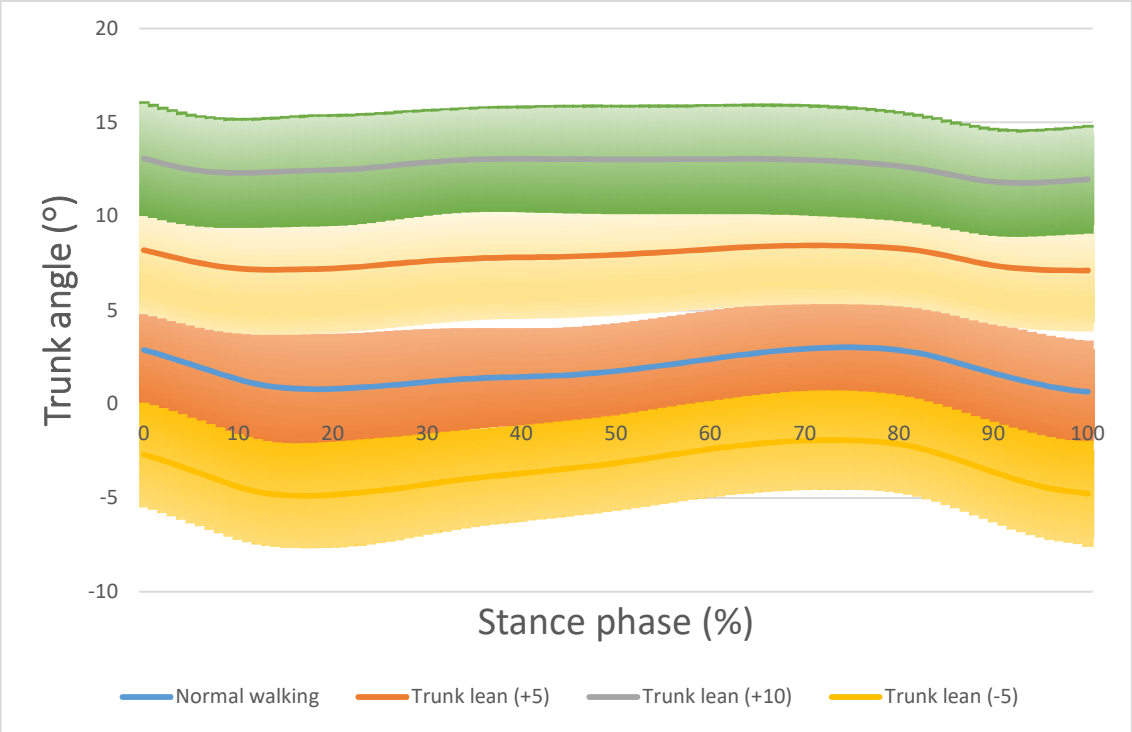


Figure 5-1 The ensemble average, across all 20 subjects, of the mean and standard deviation of the sagittal trunk angle for normal walking and the three different trunk lean conditions.

5.4.2 Sagittal moment

Hip moment

The data showed that as the trunk angle was increased, there was a clear rise in the magnitude of the hip extensor moment across the stance phase and a corresponding decrease in the hip extensor moment as trunk lean was decreased (Figure 5-2). Figure 5-3 and Table 5-2 show data on the hip extensor moment, averaged between 15-25% of the stance phase, for each of the trunk lean conditions. The ANOVA test showed that there was a significant change in the hip moment as trunk lean was changed ($p < .005$). Pairwise analysis (shown in Figure 5-3) demonstrated that the hip moment increased significantly by 70% when trunk lean was increased by 5° and decreased significantly by 50% when trunk lean was

decreased by 5°. The LMM also showed that increasing trunk lean had significant effect on hip moment $p < 0.005$, specifically, when trunk lean was increased by one degree, the hip extensor moment increased by 0.03 Nm/kg, with a standard error = 0.003 Nm/kg.

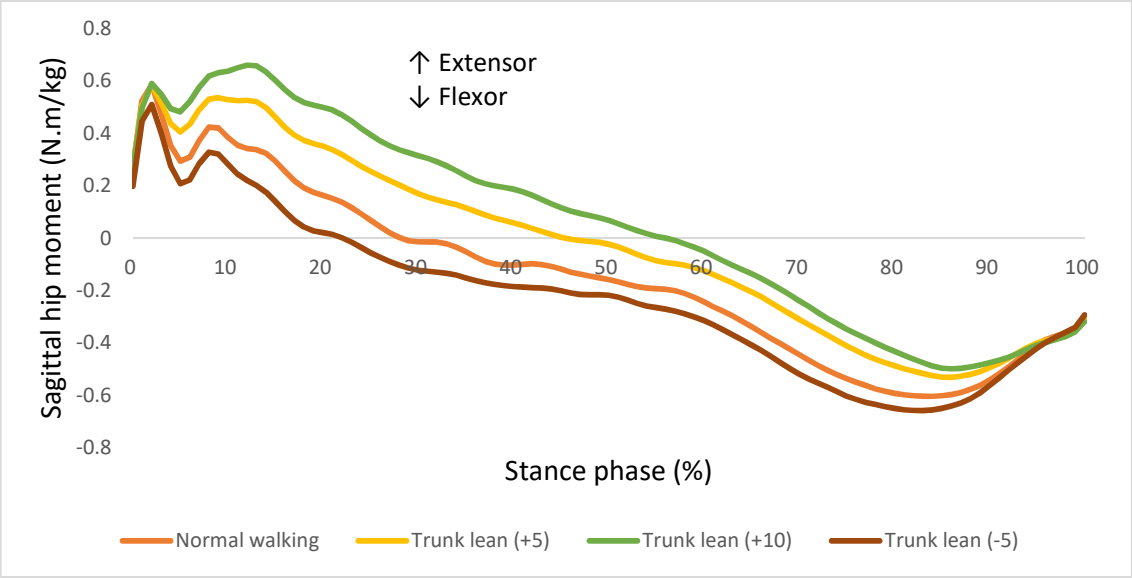


Figure 5-2 The ensemble average, across all 20 subjects, of the sagittal hip moment for normal walking and the three different trunk lean conditions.

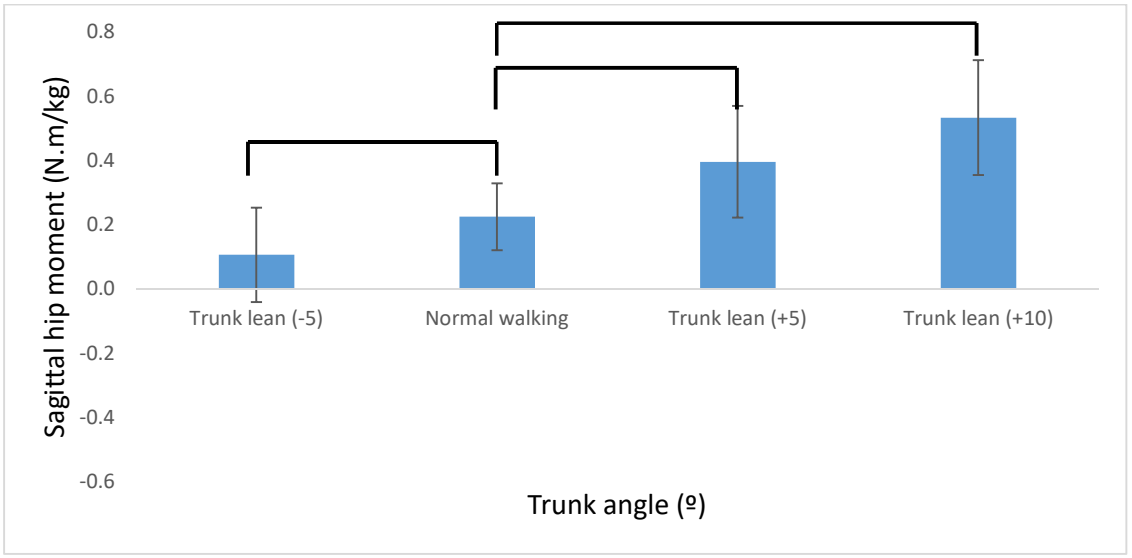


Figure 5-3 The sagittal of hip moment during walking and with different trunk lean, averaged across the period of 15-25% of stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation. Horizontal lines indicate significant ($p<0.05$) pairwise differences. Note for clarity only the pairwise comparisons to normal walking have been shown.

Table 5-2 The mean and standard deviation of the hip moment (Nm/kg) during normal walking and with different trunk lean in the period of 15-25% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	0.11	0.23	0.40	0.53	.000
SD	0.15	0.10	0.17	0.18	

Knee moment

Figure 5-4 shows that as the trunk angle was increased, there was a slight decrease in the magnitude of the knee extensor moment across the stance phase. However, this decrease was relatively small in magnitude. Figure 5-5 and Table 5-3 show data on the knee extensor moment, averaged between 15-25% of the stance phase, for each of the trunk lean conditions. The ANOVA test showed no statistically significant differences in the knee moment during walking with different trunk lean conditions (P = .13). As the ANOVA test did not show a difference, the LMM was omitted.

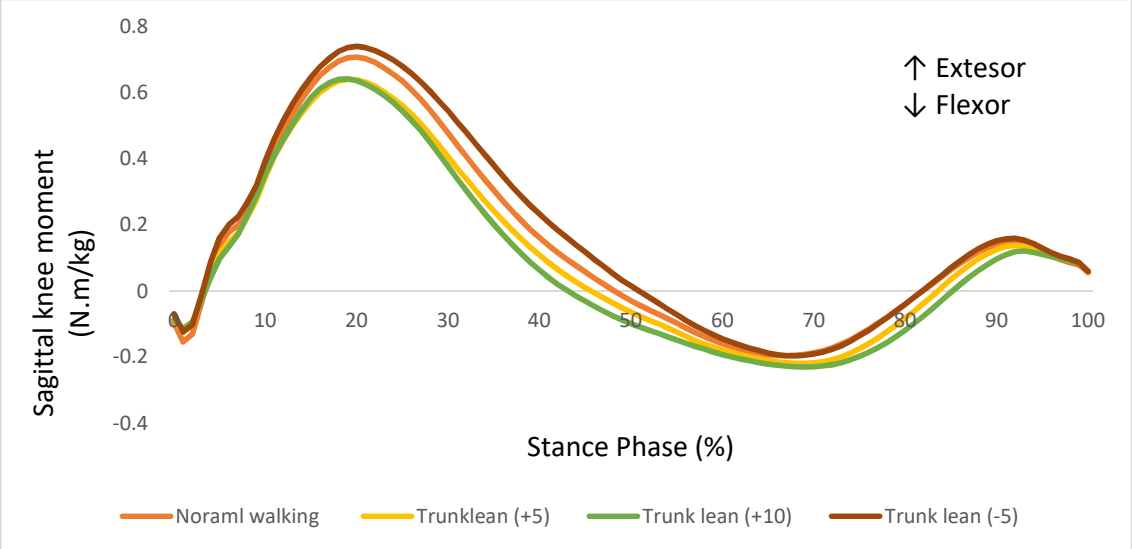


Figure 5-4 The ensemble average, across all 20 subjects, of the sagittal knee moment for normal walking and the three different trunk lean conditions.

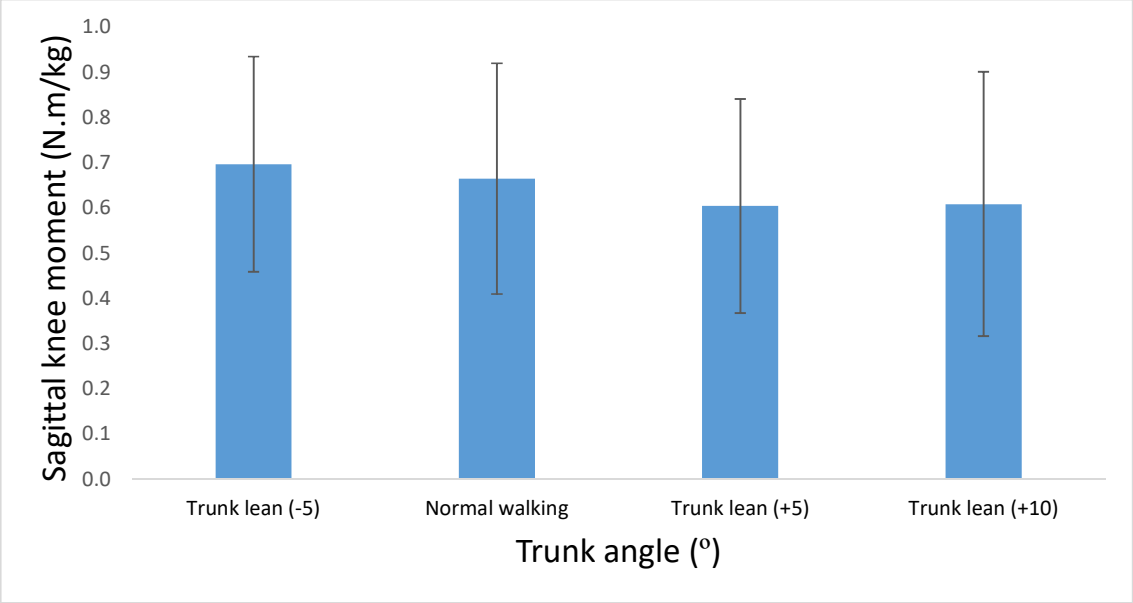


Figure 5-5 The sagittal of knee moment during walking and with different trunk lean, averaged across the period of 15-25% of stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation.

Table 5-3 The mean and standard deviation of the knee moment (Nm/ kg) during normal walking and with different trunk lean in the period of 15-25% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	0.70	0.67	0.60	0.61	.13
SD	0.24	0.26	0.24	0.29	

Ankle moment

The data showed that as the trunk angle was increased, there was a clear increase in the magnitude of the ankle plantar flexor moment in the early stance phase and a corresponding decrease in the ankle plantar flexor moment as trunk lean was decreased (Figure 5-6). Figure 5-7 and Table 5-4 show data on the plantar flexor moment, averaged between 15-25% of the stance phase, for each of the trunk lean conditions. The ANOVA test showed that there was a significant change in the plantar flexor moment as trunk lean was changed (P = .004). Pairwise analysis (shown in Figure 5-7) demonstrated that the plantar flexor moment increased significantly by 80% when trunk lean was increased by 5° and decreased significantly when trunk lean was decreased by 5°. A further statistical by LMM showed that increasing trunk lean had significant effect on ankle moment $p < 0.005$, specifically, when

trunk lean was increased by one degree, the ankle plantar flexor moment increased by .01 Nm/kg, with a standard error = 0.01 Nm/kg.

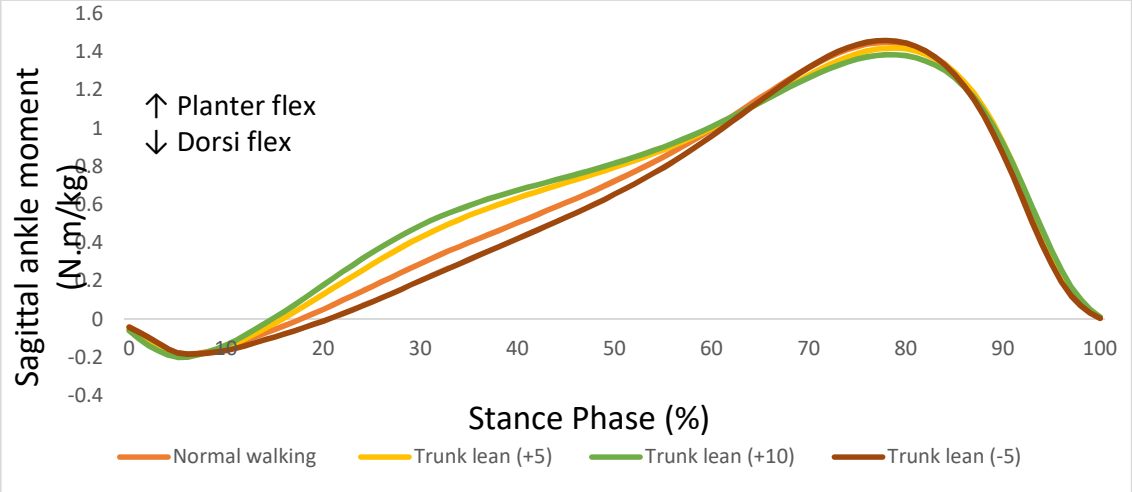


Figure 5-6 The ensemble average, across all 20 subjects, of the sagittal ankle moment for normal walking and the three different trunk lean conditions

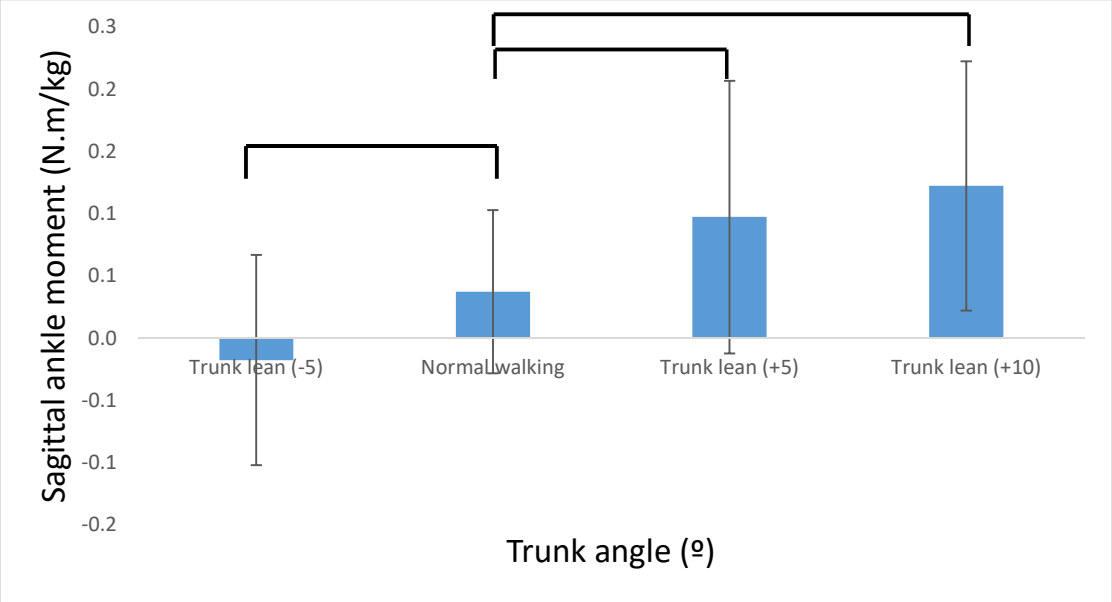


Figure 5-7 The sagittal of ankle moment during walking and with different trunk lean, averaged across the period of 15-25% of the stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation. Horizontal lines indicate significant ($p<0.05$) pairwise differences. Note for clarity only the pairwise comparisons to normal walking have been shown.

Table 5-4 The mean and standard deviation of the ankle moment (Nm/ kg) during normal walking and with different trunk lean in the period of 15-25% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	-0.02	0.04	0.10	0.12	.004
SD	0.08	0.07	0.11	0.10	

5.4.3 Muscle activation

Medial gastrocnemius (MG)

Figure 5-8 shows that as the trunk angle was increased, there was a slight increase in MG muscle activity and a slight decrease in the MG muscle activity as trunk lean was decreased in the early stance phase. Figure 5-9 and Table 5-5 show data on the MG muscle, averaged between 10-20% of the stance phase, for each trunk lean condition. The ANOVA test showed that there was a significant change in the MG muscle activity as trunk lean was changed ($P > .005$). Pairwise analysis (shown in Figure 5-9) demonstrated that the MG muscle increased significantly by 60% when trunk lean was increased by 5° and decreased significantly by 20% when trunk lean decreased by 5°. Furthermore, the LMM result shows increasing trunk lean had significant effect on MG muscle $p < 0.005$, specifically, when trunk lean was increased by one degree, the MG muscle activity increased by .01, with a standard error = 0.001.

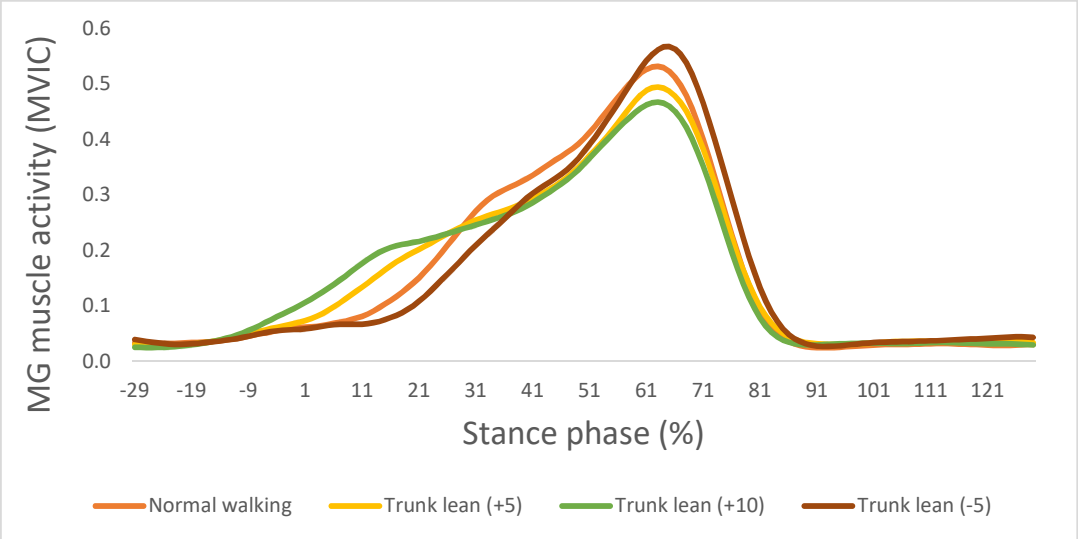


Figure 5-8 The ensemble average, across all 20 subjects, of the MG muscle activity for normal walking and the three different trunk lean conditions.

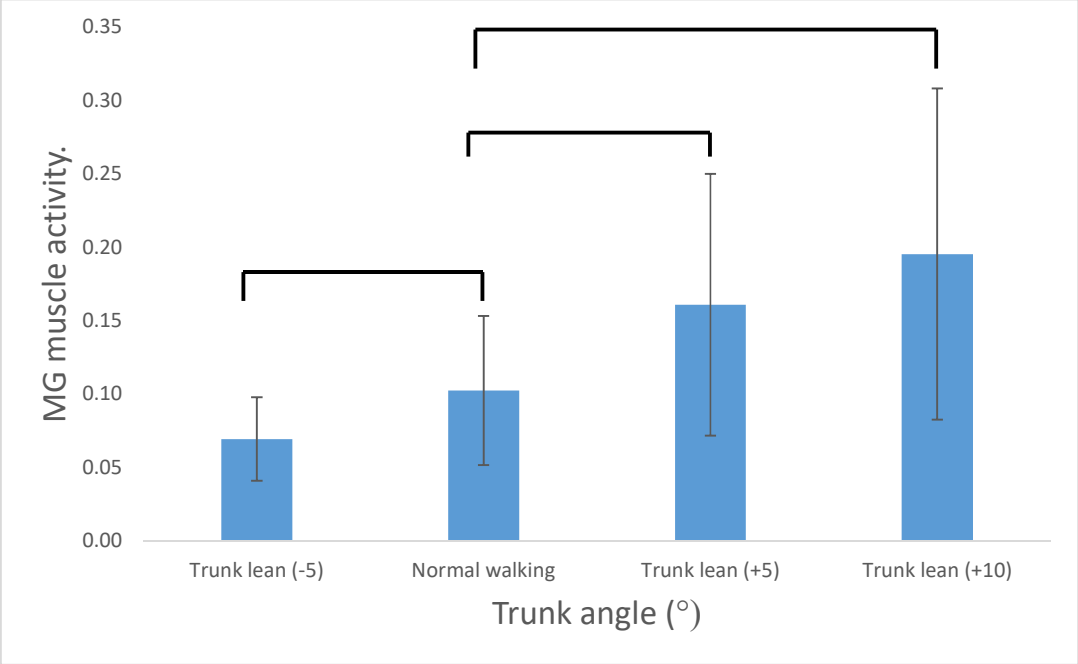


Figure 5-9 The MG muscle activity during walking and with different trunk lean, averaged across the period of 10-20% of the stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation. Horizontal lines indicate significant (p<0.05) pairwise differences. Note for clarity only the pairwise comparisons to normal walking have been shown.

Table 5-5 The mean and standard deviation of the MG muscle activity (MVIC: proportion of the MVIC) during normal walking and with different trunk lean in the period of 10-20% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	0.07	0.10	0.16	0.20	.00
SD	0.03	0.05	0.09	0.11	

Lateral gastrocnemius (LG)

Figure 5-10 shows that as the trunk was increased, there was a slight increase in the LG muscle activity and only minimal change was observed in the LG muscle activity in the early stance phase. Figure 5-11 and Table 5-6 provide data on the LG muscle, averaged between 10-20% of the stance phase, for each trunk lean condition. The ANOVA test showed that there was a statistically significant change in the LG muscle activity during walking with different trunk lean conditions (P < .005). Post hoc test (shown in Figure 5-11) revealed that only there was a statistically significant increased between the normal walking to the two forward leans p<

0.005. The LMM showed that increasing trunk lean had significant effect on LG muscle p< 0.005, specifically, when trunk lean was increased by one degree, the LG muscle activity increased by .003, with a standard error = 0.001.

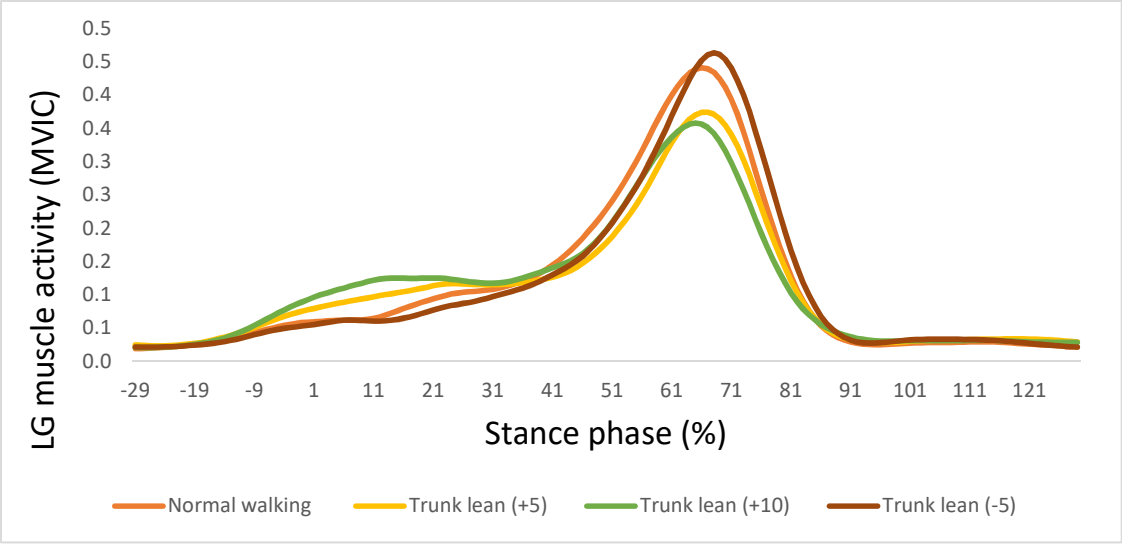


Figure 5-10 The ensemble average, across all 20 subjects, of the LG muscle activity for normal walking and the three different trunk lean conditions.

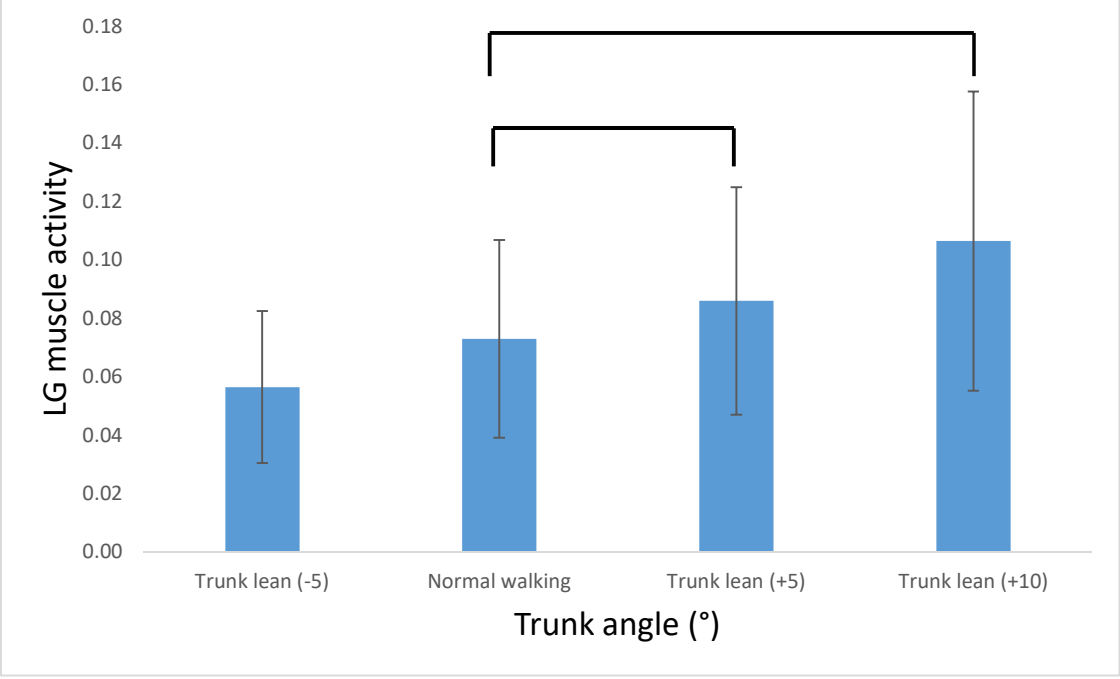


Figure 5-11 The LG muscle activity during walking and with different trunk lean, averaged across the period of 10-20% of the stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation. Horizontal lines indicate significant (p<0.05) pairwise differences. Note for clarity only the pairwise comparisons to normal walking have been shown.

Table 5-6 The mean and standard deviation of the LG muscle activity (MVIC: Proportion of the MVIC) during normal walking and with different trunk lean in the period of 10-20% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	0.06	0.07	0.09	0.11	.00
SD	0.03	0.03	0.04	0.05	

Vastus medialis oblique (VMO)

It is apparent from Figure 5-12 that there were no clear differences in the magnitude of the VMO muscle activation as trunk angle was increased or decreased. Figure 5-13 and Table 5-7 show data on the VMO muscle activation averaged between 10-20% of the stance phase for each of the trunk lean conditions. The ANOVA test showed that there were no statistical differences in the VMO muscle activation during walking with different trunk lean conditions (p = .37). As the ANOVA test did not show a difference, the LMM was omitted.

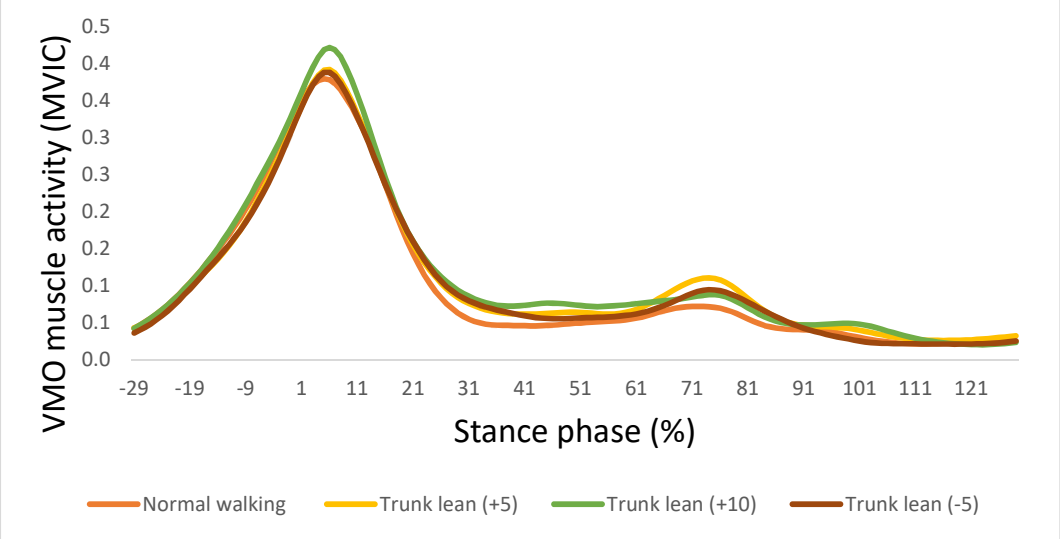


Figure 5-12 The ensemble average, across all 20 subjects, of the VMO muscle activity for normal walking and the three different trunk lean conditions.

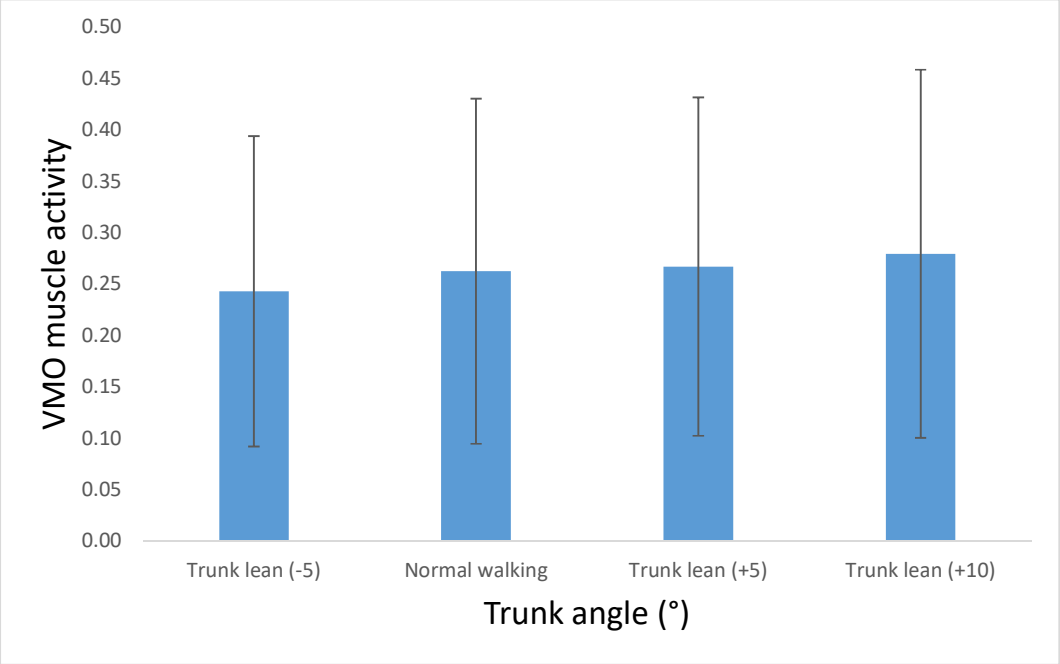


Figure 5-13 The VMO muscle activity during walking and with different trunk lean, averaged across the period of 10-20% of stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation.

Table 5-7 The mean and standard deviation of the VMO muscle activity (MVIC: proportion of the MVIC) during normal walking and with different trunk lean in the period of 10-20% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	0.24	0.26	0.27	0.28	.37
SD	0.15	0.17	0.16	0.18	

Vastus lateralis oblique (VLO)

Figure 5-14 also showed that there were no clear differences in the magnitude of the VLO muscle activation as the trunk angle was increased and decreased. Figure 5-15 and Table 5-8 show data on the VLO muscle activation, averaged between 10-20% of the stance phase for each of the trunk lean conditions. The ANOVA test showed that there were no statistically significant differences in the VLO muscle activation during walking with different trunk lean ($p = .46$). As the ANOVA test did not show a difference, the LMM was omitted.

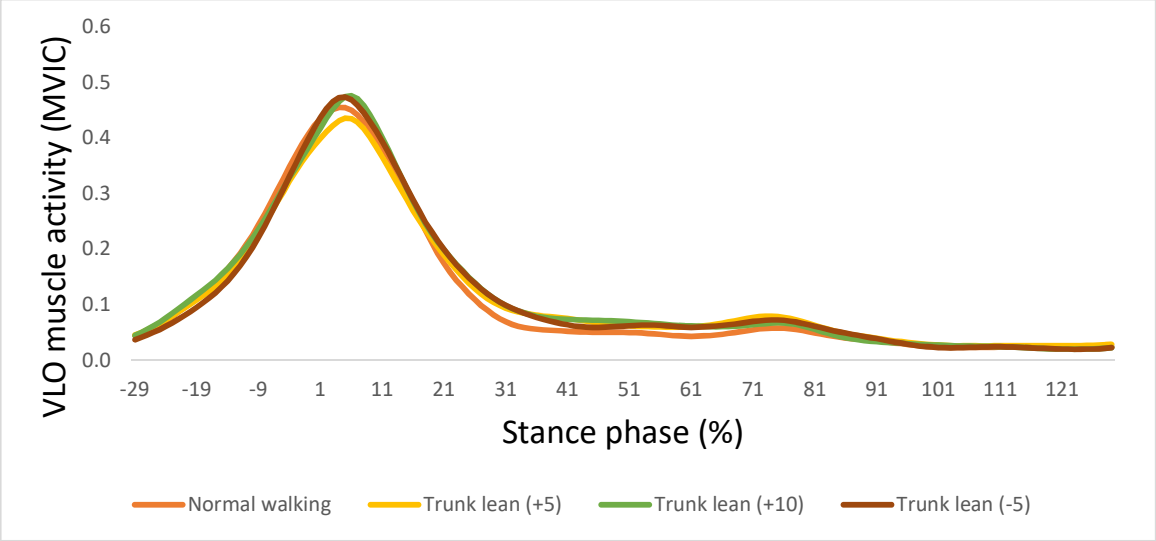


Figure 5-14 The ensemble average, across all 20 subjects, of the VLO muscle activity for normal walking and the three different trunk lean conditions.

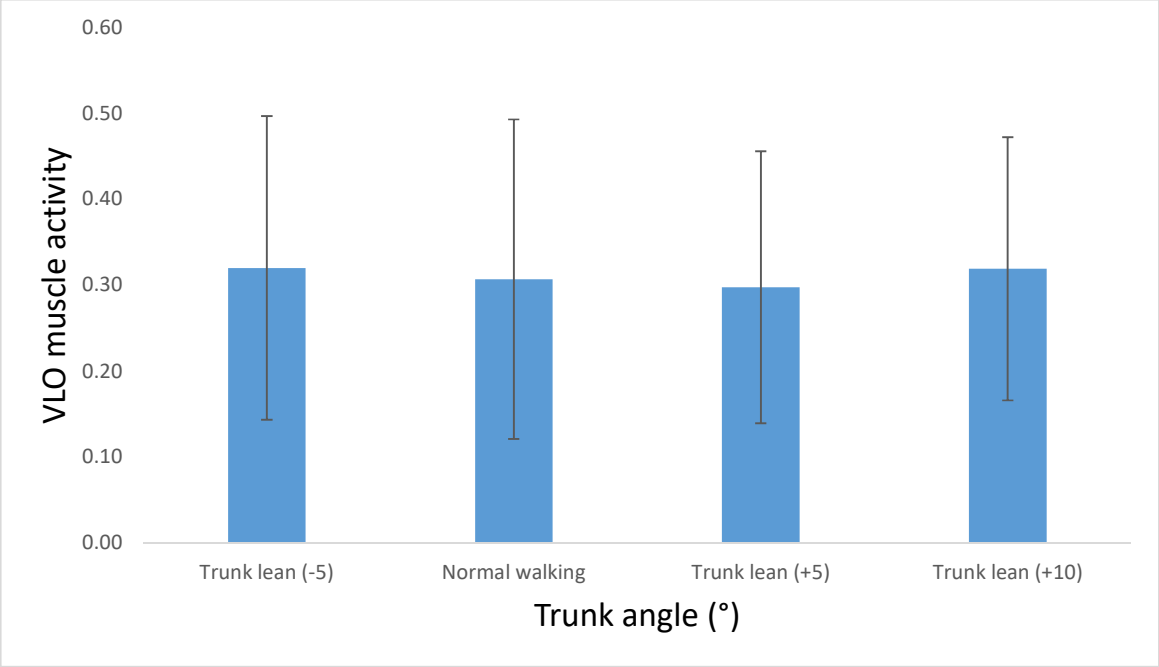


Figure 5-15 The VLO muscle activity during walking and with different trunk lean, averaged across the period of 10-20% of the stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation.

Table 5-8 The mean and standard deviation of the VLO muscle activity (MVIC: proportion of the MVIC) during normal walking and with different trunk lean in the period of 10-20% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	0.32	0.31	0.30	0.32	.46
SD	0.18	0.19	0.16	0.15	

Semitendinosus (ST)

In general, the data showed that as the trunk angle was increased, there was a clear increase in the magnitude of the ST muscle activation and a slight decrease as trunk lean was decreased (Figure 5-16). Figure 5-17 and Table 5-9 show data on the ST muscle activation, averaged between 10-20% of the stance phase, for each of the trunk lean conditions. The ANOVA test showed that there was a significant change in the ST muscle activity as trunk lean was changed (P< .005). Pairwise analysis (shown in Figure 5-17) indicated that the ST muscle activation increased and decreased significantly when trunk lean was increased and decreased, respectively. The LMM showed that increasing trunk lean had significant effect on ST muscle activity (p< 0.005). Data from the LMM showed that when trunk lean was increased by one degree, the ST muscle activation increased by .0013, with a standard error = .001.

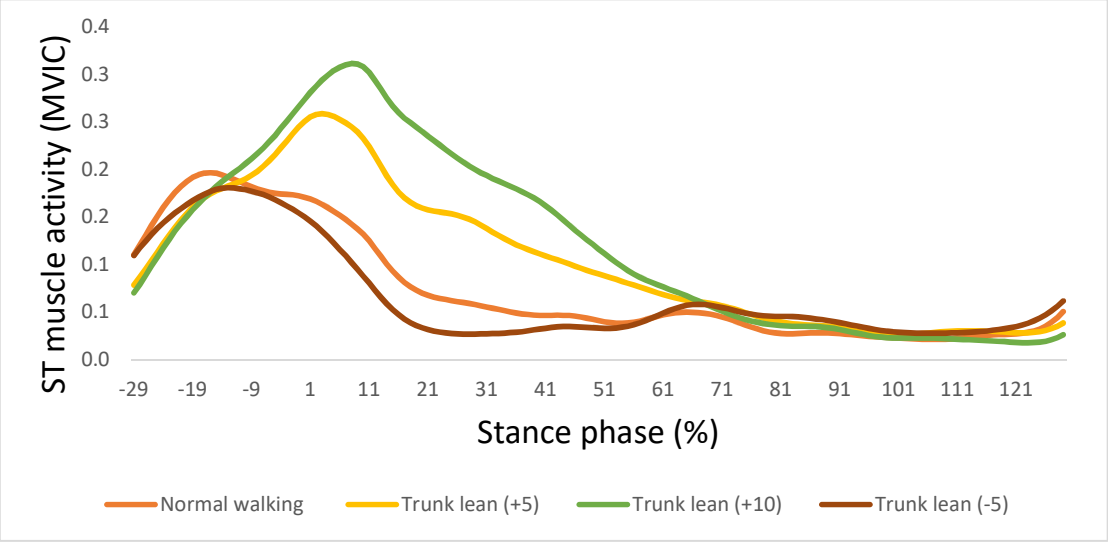


Figure 5-16 The ensemble average, across all 20 subjects, of the ST muscle activity for normal walking and the three different trunk lean conditions.

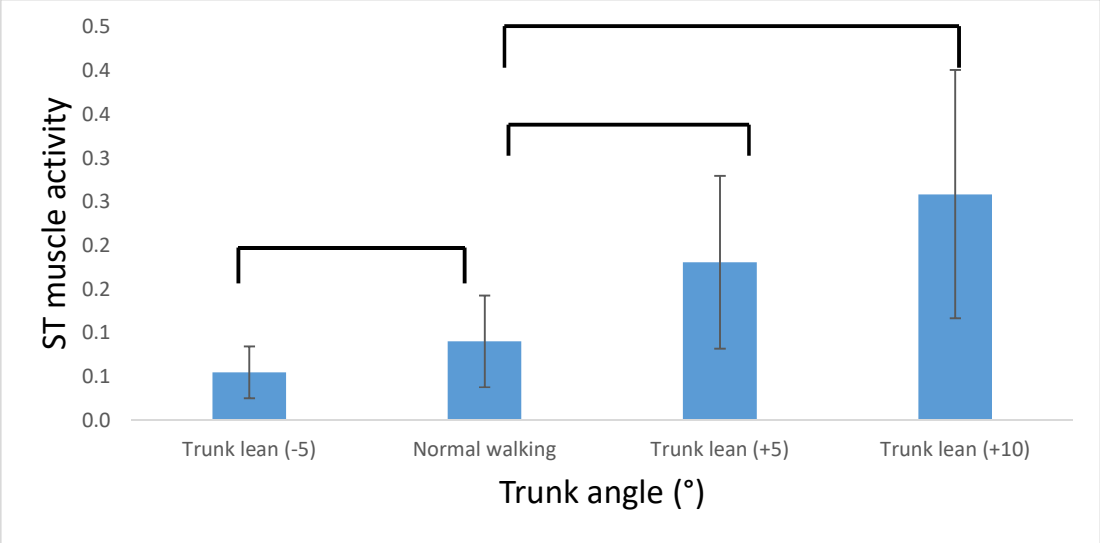


Figure 5-17 The ST muscle activity during walking and with different trunk lean, averaged across the period of 10-20% of the stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation. Horizontal lines indicate significant (p<0.05) pairwise differences. Note for clarity only the pairwise comparisons to normal walking have been shown.

Table 5-9 The mean and standard deviation of the ST muscle activity (MVIC: proportion of the MVIC) during normal walking and with different trunk lean in the period of 10-20% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	0.06	0.09	0.18	0.26	.00
SD	0.03	0.05	0.10	0.14	

Biceps femoris (BF)

In general, the data showed that as the trunk angle was increased, there was a clear increase in the magnitude of the BF muscle activity and a slight decrease as trunk lean was decreased (Figure 5-18). Figure 5-19 and Table 5-10 show data on the BF muscle activity, averaged between 10-20% of the stance phase, for each of the trunk lean conditions. The ANOVA test showed that there was a significant change in the BF muscle activity as trunk lean was changed ($P < .005$). The post hoc test (shown in Figure 5-19) demonstrated that the BF muscle activity increased significantly by 80% and 150% when trunk lean was increased by 5°and 10° respectively, with a corresponding decrease of 17% when trunk lean was decreased by 5°. The

LMM, also showed increasing trunk lean had significant effect on BF ($p < 0.005$) over interested period, specifically, when trunk lean was increased by one degree, the BF muscle activation increased by .007, with a standard error = .001.

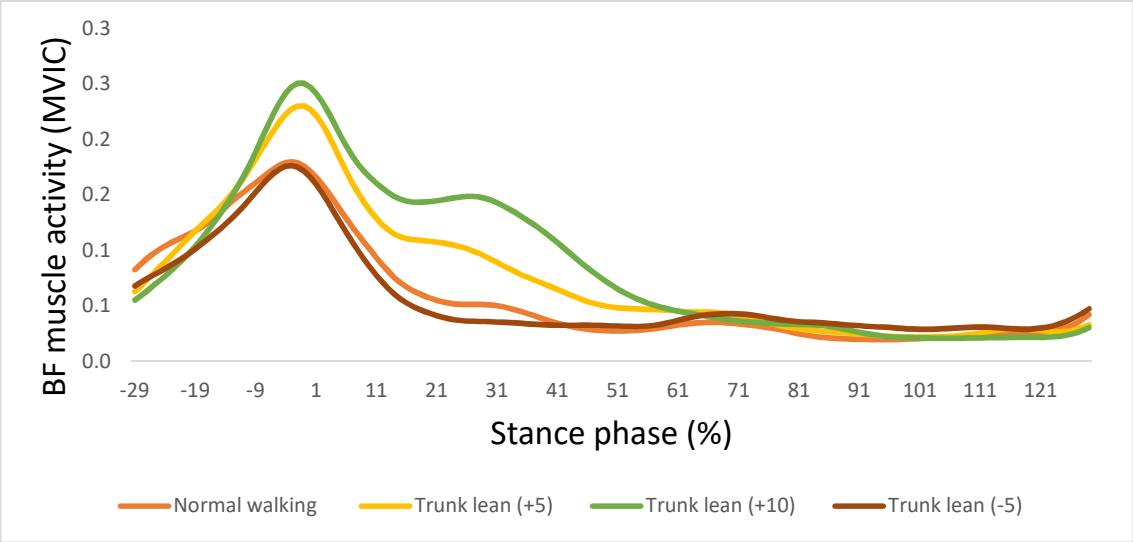


Figure 5-18 The ensemble average, across all 20 subjects, of the BF muscle activity for normal walking and the three different trunk lean conditions.

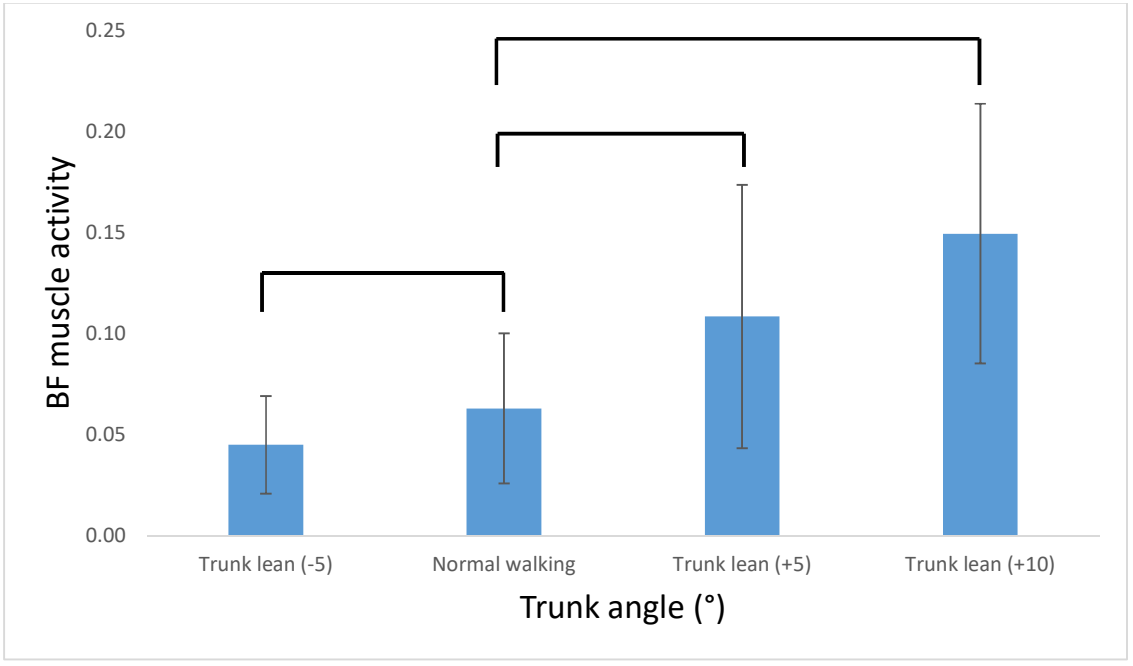


Figure 5-19 The BF muscle activity during walking and with different trunk lean, averaged across the period of 10-20% of the stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation. Horizontal lines indicate significant ($p<0.05$) pairwise differences. Note for clarity only the pairwise comparisons to normal walking have been shown.

Table 5-10 The mean and standard deviation of the BF muscle activity (MVIC: proportion of the MVIC) during normal walking and with different trunk lean in the period of 10-20% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	0.04	0.06	0.11	0.15	.00
SD	0.02	0.04	0.07	0.06	

5.4.4 Co-contraction

Medial gastrocnemius and medial quadriceps (MGVMO) co-contraction

It can be seen from Figure 5-20 that as the trunk angle was increased, there were slight increases in muscle co-contraction between the MGVMO. Figure 5-20 and Table 5-14 show data on the co-contraction between the MGVMO, averaged between 10-20% of the stance phase, for each of the trunk lean conditions. ANOVA test showed that there was a statistically significant change in the muscle co-contraction between the MGVMO during walking with different trunk lean ($p < .005$). The post hoc test (shown in Figure 5-20) indicated that the only statistically difference was between normal walking and the +10° condition ($p < 0.005$).

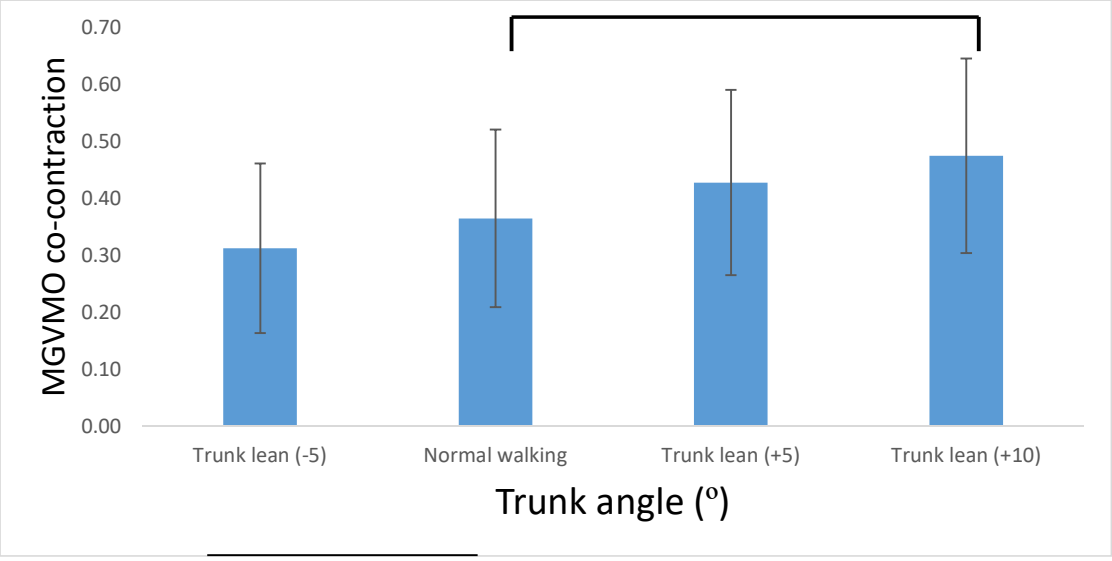


Figure 5-20 The muscle co-contraction between MGVMO during walking and with different trunk lean, averaged across the period of 10-20% of stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation. Horizontal lines indicate significant ($p<0.05$) pairwise differences. Note for clarity only the pairwise comparisons to normal walking have been shown.

Table 5-11 The mean and standard deviation of the co-contraction between the MGVMO activity (MVIC: proportion of the MVIC) during the normal walking and with different trunk lean in the period of 10-20% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	0.31	0.37	0.43	0.48	.00
SD	0.15	0.16	0.16	0.17	

Lateral gastrocnemius and lateral quadriceps (LGVLO) co-contraction

The data showed that as the trunk angle was increased or decreased, there was no change in the magnitude of the co-contraction between the LGVLO activations (Figure 5-21). Figure 5-21 and Table 5-12 show data on the co-contraction between the LGVLO, averaged between 10-20% of the stance phase, for each of the trunk lean conditions. The ANOVA test showed that there were no statistically significant changes in the co-contraction between the LGVLO during walking with different trunk lean (p = .53).

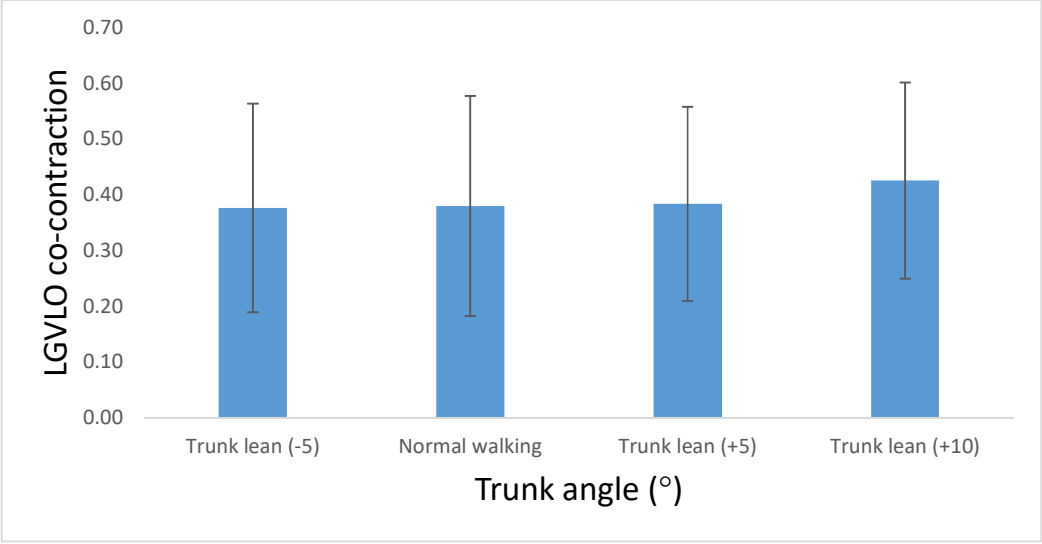


Figure 5-21 The muscle co-contraction between LGVLO muscles during walking and with different trunk lean, averaged across the period of 10-20% of stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation.

Table 5-12 The mean and standard deviation of the co-contraction between the LGVLO activity (MVIC: proportion of the MVIC) during normal walking and with different trunk lean in the period of 10-20% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	0.38	0.38	0.38	0.43	.53
SD	0.19	0.20	0.17	0.18	

Biceps femoris and lateral quadriceps (BFVLO) co-contraction

Figure 5-22 shows that as the trunk angle was increased, there was a slightly increase in the magnitude of the co-contraction between BF and VLO. Figure 5-22 and Table 5-13 show data on the co-contraction between the BFVLO, averaged between 10-20% of the stance phase, for each of the trunk lean conditions. The ANOVA test showed that there were statistically significant changes in the co-contraction between the BFVLO during walking with different trunk lean conditions (P < 005). However, pairwise analysis (shown in Figure 5-22) demonstrated that the only difference was between normal walking and the +10° condition (p< 0.005).

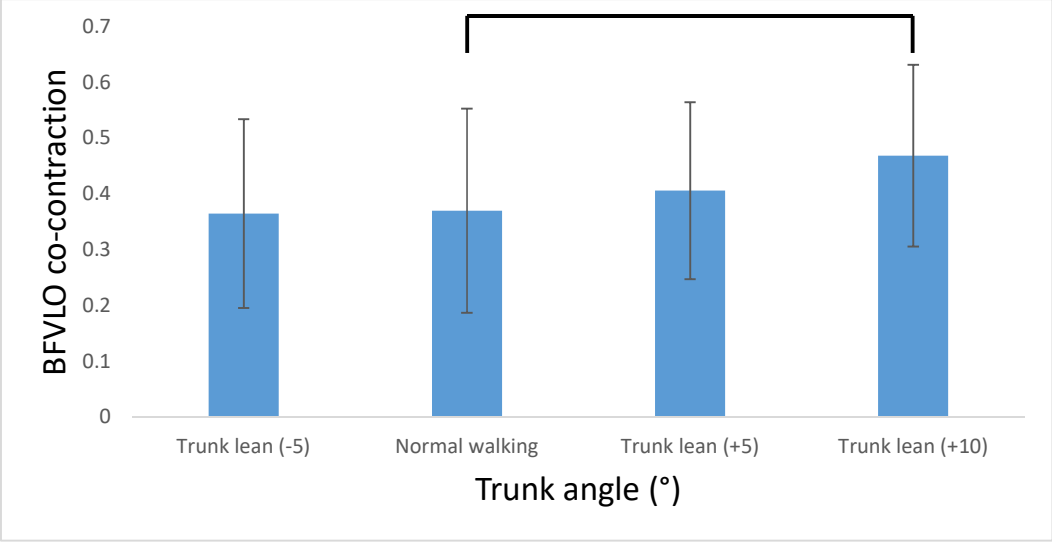


Figure 5-22 The muscle co-contraction between BFVLO muscles during walking and with different trunk lean, averaged across the period of 10-20% of stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation. Horizontal lines indicate significant (p<0.05) pairwise differences. Note for clarity only the pairwise comparisons to normal walking have been shown.

Table 5-13 The mean and standard deviation of the co-contraction between the BFVLO activity (MVIC: proportion of the MVIC) during normal walking and with different trunk lean in the period of 10-20% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	0.37	0.37	0.41	0.47	.001
SD	0.17	0.18	0.16	0.16	

Semitendinosus and medial quadriceps (STVMO) co-contraction

The data showed that as the trunk angle was increased, there was a clear increase in the magnitude of co-contraction between the ST and VMO and a corresponding decrease in the co-contraction between the STVMO as trunk lean was decreased (Figure 5-23).Figure 5-23 and Table 5-14 show data on the co-contraction between ST and VMO, averaged between 10-20% of the stance phase, for each of the trunk lean conditions. The ANOVA test showed that there was a statistically significant change during walking with different trunk lean conditions ($p < 0.005$) and pairwise analysis (shown in Figure 5-23) demonstrated that the differences were between normal walking and the two forward lean conditions ($p < 0.005$).

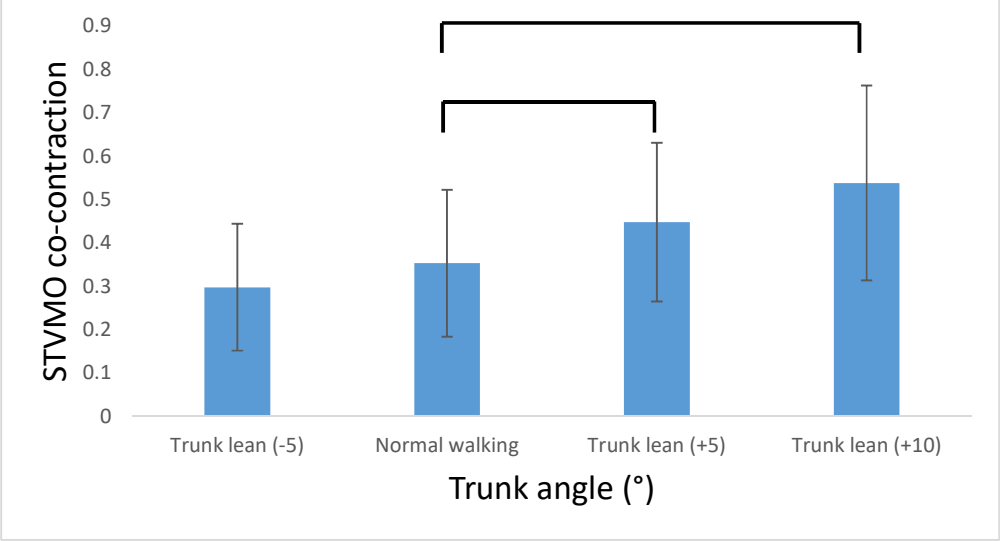


Figure 5-23 The muscle co-contraction between STVMO muscles during walking and with different trunk lean, averaged across the period of 10-20% of stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation. Horizontal lines indicate significant ($p<0.05$) pairwise differences. Note for clarity only the pairwise comparisons to normal walking have been shown.

Table 5-14 The mean and standard deviation of the co-contraction between the STVMO activity (MVIC: proportion of the MVIC) during normal walking and with different trunk lean in the period of 10-20% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	0.30	0.35	0.45	0.54	.00
SD	0.15	0.17	0.18	0.22	

5.4.5 Sagittal angles

Hip angle

The data showed that as trunk angle was increased, there was an increase in the overall degree of hip flexion angle across the gait cycle (Figure 5-24). Figure 5-25 and Table 5-15 show data on the hip flexion angle, averaged between 15-25% of the stance phase, for each of the trunk lean conditions. The ANOVA result showed that there was a statistically significant difference in the hip angle during walking with different trunk lean ($P < .005$). In addition, the post hoc test (shown in Figure 5-25) revealed that there was a significant increase between normal walking and the two forward lean conditions $p< 0.005$.

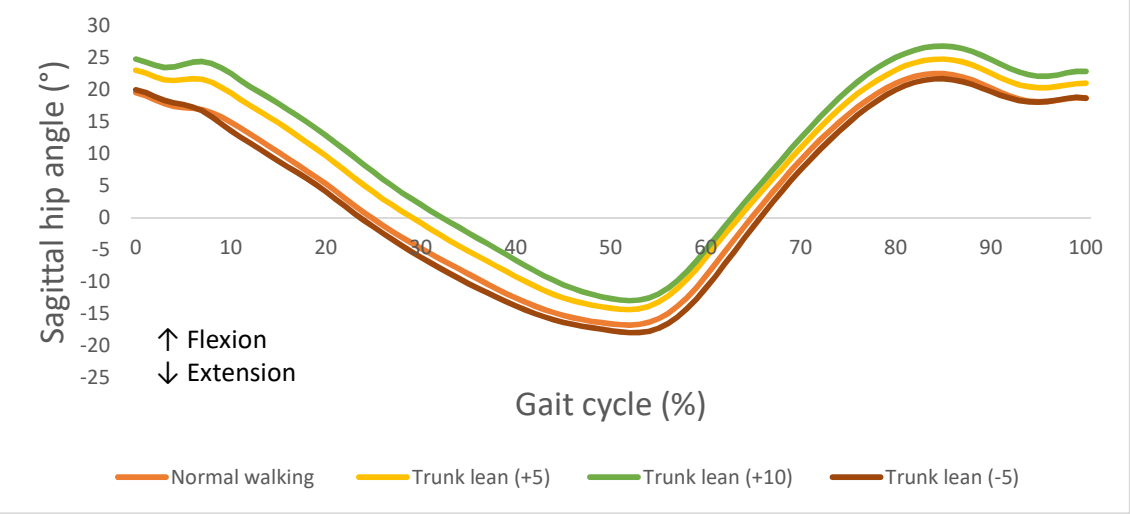


Figure 5-24 The ensemble average, across all 20 subjects, of sagittal hip angle for normal walking and the three different trunk lean conditions.

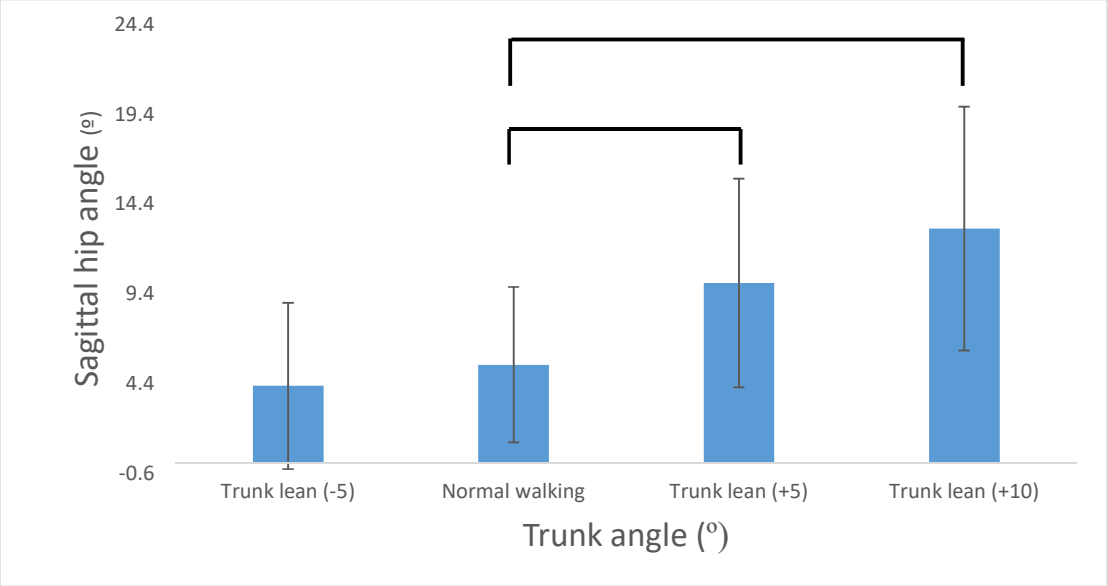


Figure 5-25 The sagittal hip angle during walking and with different trunk lean, averaged across the period of 15-25% of stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation. Horizontal lines indicate significant (p<0.05) pairwise differences. Note for clarity only the pairwise comparisons to normal walking have been shown.

Table 5-15 The mean and standard deviation of the sagittal hip angle (°) during normal walking and with different trunk lean in the period of 15-25% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	4.27	5.46	10.01	13.04	.00
SD	4.63	4.33	5.81	6.80	

Knee angle

It can be seen from Figure 5-26 that as the trunk lean was increased there was a slight increase in the knee flexion angle in the early stance phase. However, no change was observed in the knee angle when the trunk was decreased (Figure 5-26). Figure 5-27 and Table 5-16 show data on the knee angle averaged between 15-25% of the stance phase for each of the trunk lean conditions. The ANOVA test showed that there were significant changes in the knee angle during walking with different trunk lean conditions ($p < .005$). Pairwise analysis (shown in Figure 5-27) demonstrated that that the differences were between normal walking and the two forward lean conditions ($p< 0.005$).

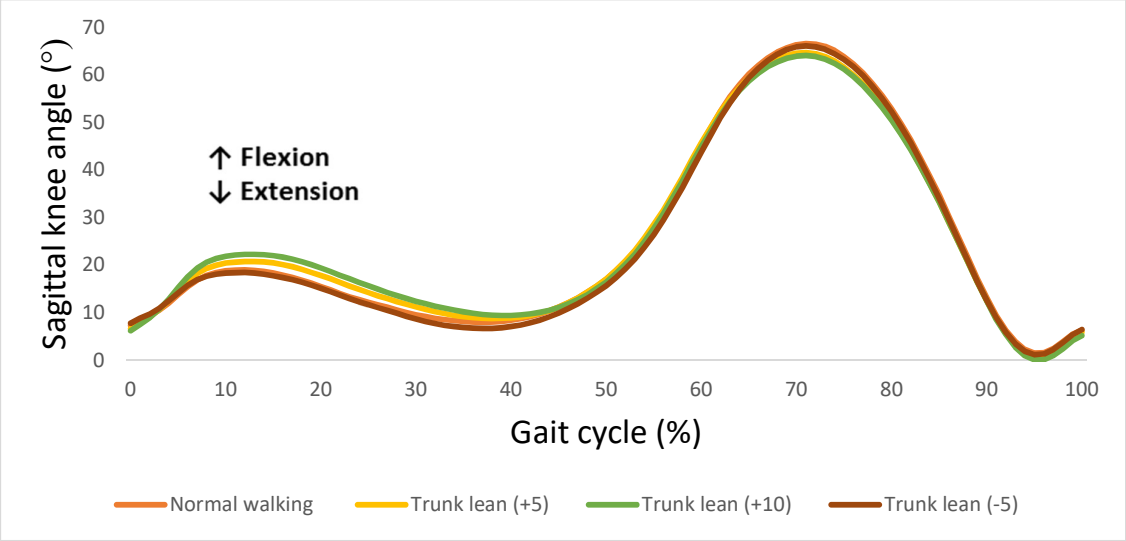


Figure 5-26 The ensemble average, across all 20 subjects, of sagittal knee angle for normal walking and the three different trunk lean conditions.

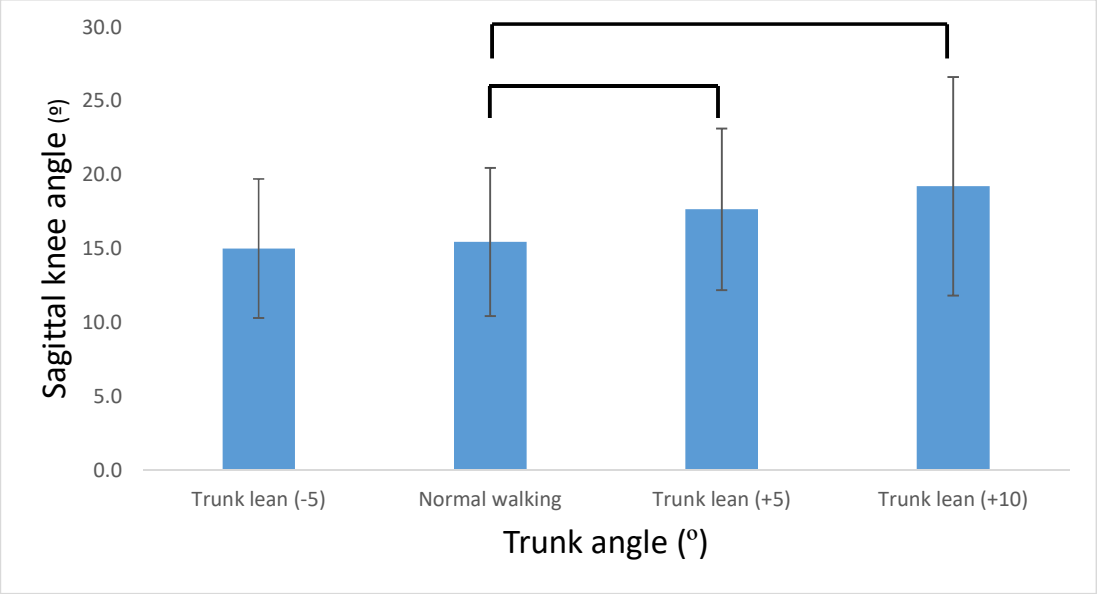


Figure 5-27 The sagittal knee angle during walking and with different trunk lean, averaged across the period of 15-25% of stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation. Horizontal lines indicate significant ($p<0.05$) pairwise differences. Note for clarity only the pairwise comparisons to normal walking have been shown.

Table 5-16 The mean and standard deviation of the sagittal knee angle (°) during normal walking and with different trunk lean in the period of 15-25% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	15.02	15.45	17.67	19.22	.00
SD	4.71	5.02	5.47	7.41	

Ankle angle

The data showed that, as the trunk angle was increased, there was a slight difference in the magnitude of the ankle angle in the early stance phase (Figure 5-28). Figure 5-29 and Table 5-17 show data on the ankle angle averaged between 15-25% of the stance phase for each of the trunk lean conditions. The ANOVA test showed that there were no statistically significant changes in the ankle angle during walking with different trunk lean p> 0.05.

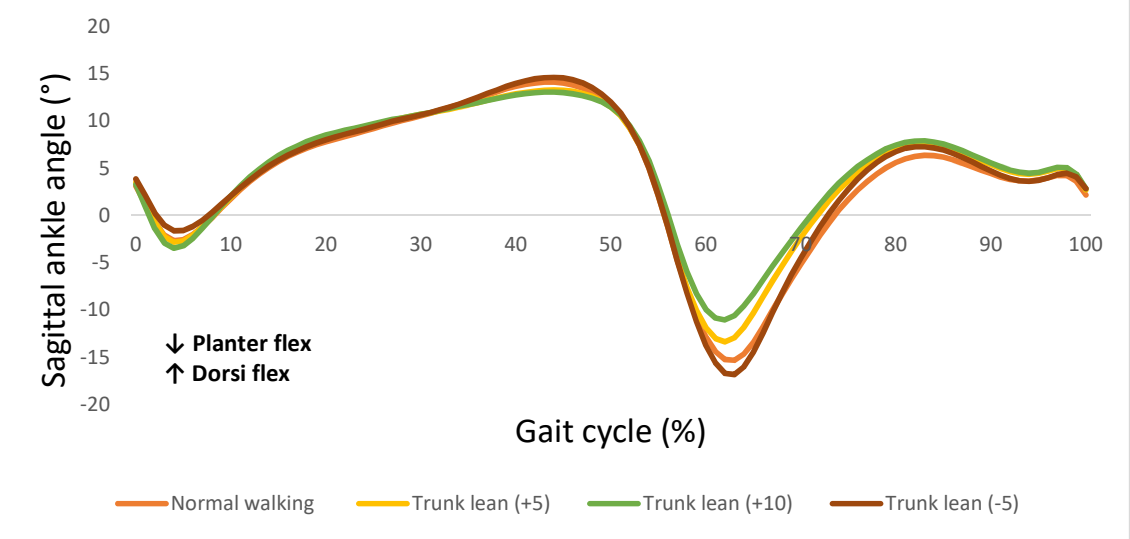


Figure 5-28 The ensemble average, across all 20 subjects, of sagittal ankle angle for normal walking and the three different trunk lean conditions.

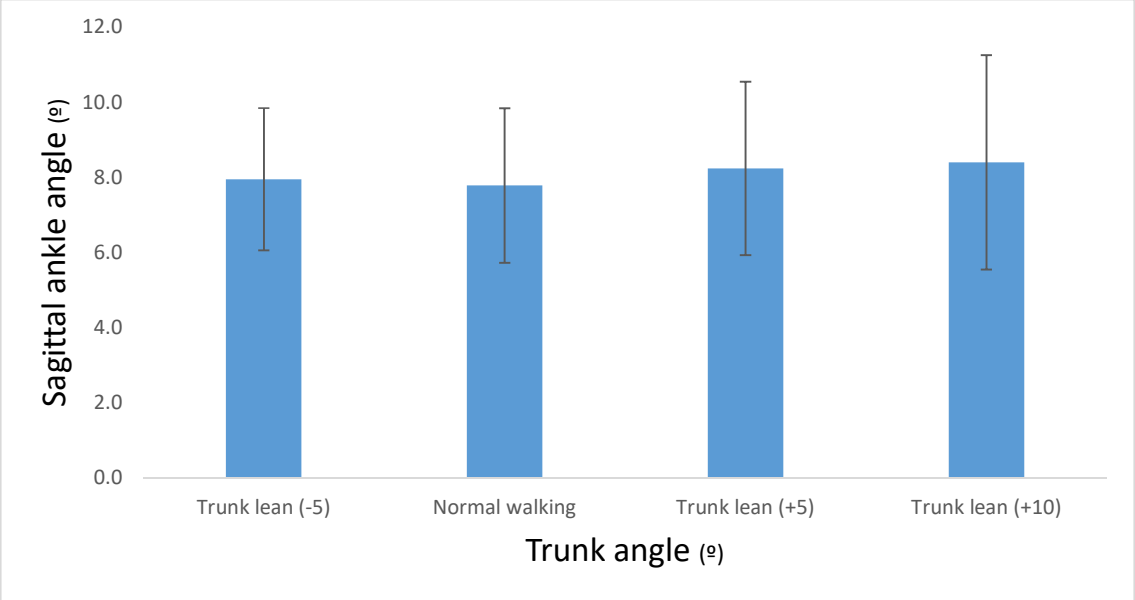


Figure 5-29 The sagittal ankle angle during walking and with different trunk lean, averaged across the period of 15-25% of stance phase. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation.

Table 5-17 The mean and standard deviation of the sagittal knee angle (°) during normal walking and with different trunk lean in the period of 15-25% of the stance phase.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	ANOVA P value
Mean	7.95	7.79	8.24	8.41	.22
SD	1.89	2.06	2.31	2.85	

5.4.6 Spatiotemporal

Speed

Figure 5-30 and Table 5-18 show data on the speed for each of the trunk lean conditions. It can be seen from Figure 5-30 that there were no differences in speed between the different trunk leans. The Freidman test showed that there were no statistically significant changes in the speed during normal walking and with different trunk leans $p > 0.05$.

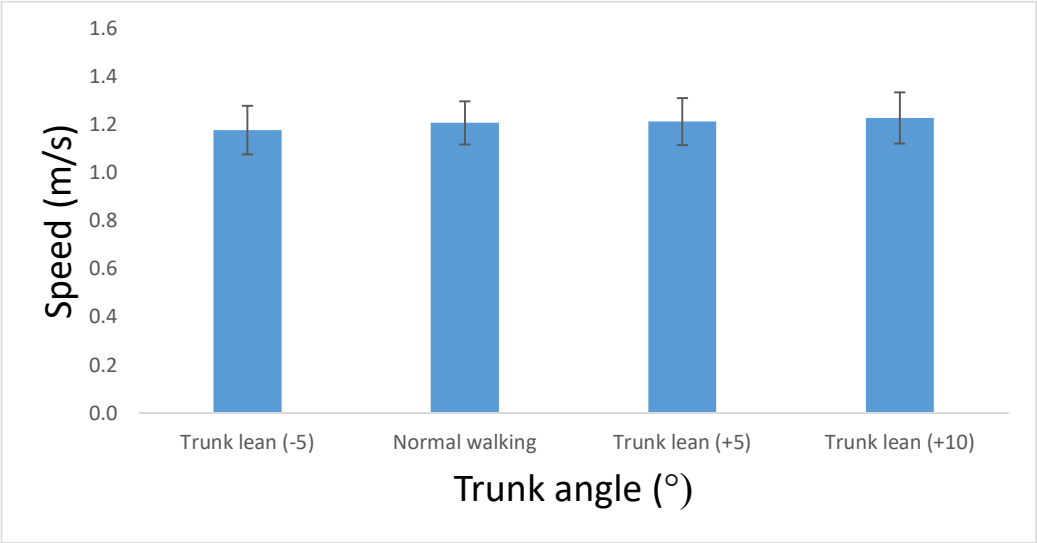


Figure 5-30 The speed during walking and with different trunk lean. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation.

Table 5-18 The median and standard deviation of the speed (m/s) during normal walking and with different trunk lean.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	Freidman P value
Median	1.18	1.21	1.21	1.23	.67
SD	0.10	0.09	0.09	0.10	

Step length

Figure 5-31 and Table 5-19 show data on the step length for each of the trunk lean conditions. It can be seen from Figure 5-31 that there were no changes in step length between the different trunk lean conditions. The Freidman test showed that there were no statistically significant changes in the step length during walking with different trunk lean conditions $p > 0.05$.

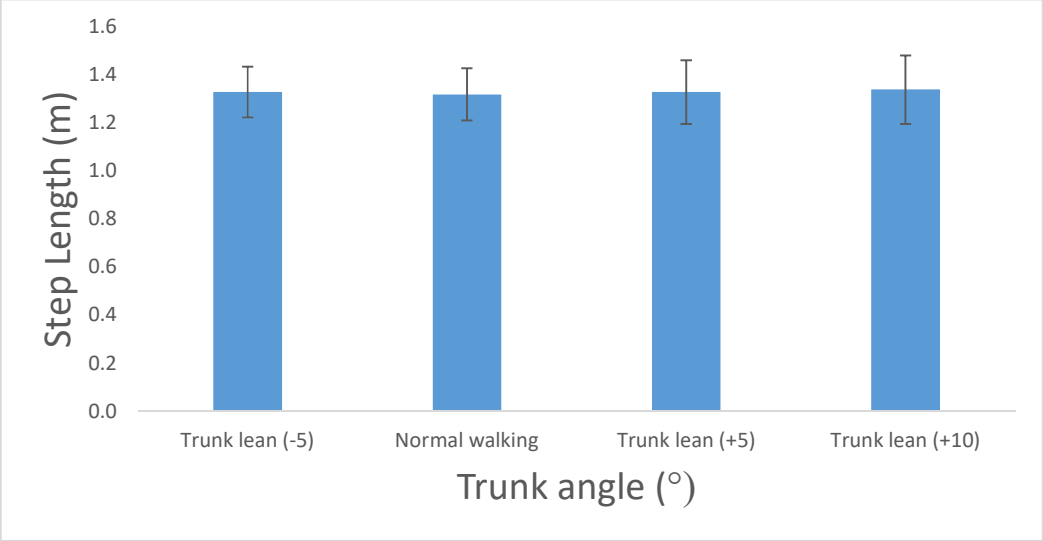


Figure 5-31 The step length during walking and with different trunk lean. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation.

Table 5-19 The median and standard deviation of the step length (m) during normal walking and with different trunk lean.

	Trunk lean (-5)	Normal walking	Trunk lean (+5)	Trunk lean (+10)	Freidman P value
Median	1.33	1.32	1.33	1.34	.65
SD	0.10	0.10	0.13	0.13	

5.5 Discussion

The present study was designed to determine the effect of increasing/decreasing trunk lean (+5°, +10°, and -5°) on the biomechanics of the hip, knee and ankle in young healthy subjects. Specifically, the study aimed to understand potential changes in kinetics and kinematics along with changes in the activation of the quadriceps, hamstrings and gastrocnemius muscles activation and the corresponding muscle co-contraction. The most pronounced effects were an increase in the hip extensor moment, an increase in activity in both hamstrings and gastrocnemius muscles and an increase in STVMO co-contraction. Interestingly, the data showed that, although there was a slight decrease in knee extensor moment, this difference was not significant between the normal walking condition and the +5 condition. Importantly only minimal changes were observed in spatiotemporal parameters. This demonstrates that

the observed changes were the result of changes in trunk inclination rather than changes in step length or walking speed.

The range of trunk angles selected in our study was small (+5°, +10° and -5°). This allows us to fully understand the precise link between the trunk inclination and lower limb joint moments and muscle activity for a range typical of normal variation. In contrast, the instructed trunk angles used in previous studies have been very large; for example, in the study by Kluger et al. (2014) and Saha et al. (2008) the trunk angle was increased by 25° ± 7° and 50° ± 7° and in the study by Grasso et al. (2000) it was increased by approximately 50 degrees. Therefore, this is the first study to fully quantify the effect of small, imposed, increases in trunk forward lean on lower limb moments and muscle activation patterns.

5.5.1 Kinematics and spatiotemporal parameters

Our result demonstrated that there were no changes in step length for each of the trunk lean conditions. In contrast to our findings, the result from Saha et al. (2008) showed that step length decreased when people were instructed to walk with an increased forward lean. They hypothesized these changes in the step length were the result of the posterior shift of the hip joint centre in the increased trunk lean condition compared to the position of the hip joint in normal walking. This difference between their findings and this study may be due to the difference in the trunk lean conditions (+5°, +10°, and -5° in our study) compared to (25° ± 7° and 50° ± 7° in their study). Importantly, our results are consistent with those of other studies showing speed did not change during walking with different trunk lean (Kluger et al., 2014; Lewis & Sahrmann, 2015; Saha et al., 2008). This demonstrates that the biomechanical changes observed in this study were not a result of changes in walking speed.

The changes in hip and knee kinematics observed in this study match those observed in earlier studies (Lewis & Sahrmann, 2015; Saha et al., 2008). Specifically, the hip flexion angle and the knee flexion angle increased during walking with a forward lean. Subjects in this current study were instructed to “move the hip backwards.” This should, in theory, lead to a change in the hip flexion angle and this was observed (Figure 5-25). However, our kinematic data showed that there was no significant change in hip angle when participants were instructed to walk with a backward lean. It would therefore appear that the backward lean condition required

more movement in the spinal column rather than at the hip. This may be due to muscular restriction at the hip, which may limit the degree to which the pelvis can posteriorly rotate during normal walking. The changes in the plantar flexion angle observed in the current study do not match with previous research. Saha et al. (2008) and Lewis and Sahrmann (2015) observed the peak of dorsiflexion angle was increased during walking over stance phase when leaning forward. However, our results showed that no differences in ankle angle during walking when leaning forward over interested period. This inconsistency may be due to the amount of trunk flexion during walking.

5.5.2 Kinetics

The current study found that there were changes in the hip, knee and ankle moments during walking with different trunk lean in the early stance phase. As mentioned in the literature review, if a forward lean is adopted, this will lead to a change in position and direction of the GRF vector. This change in the GRF vector will lead to alteration of joint moment and loading at the hip, knee, and ankle joints. For example, with forward lean, the GRF vector moves anterior relative to the hip joint (increasing the lever arm) and this will lead to an increase the hip extensor moment.

The present findings seem to be consistent with other research, which found that the hip extensor moment and the ankle plantarflexor moment increase in early stance phase as trunk angle increases (Kluger et al., 2014; Lewis & Sahrmann, 2015). In addition, our results agree with previous studies showing a small decreased in the knee extensor moment as forward lean increases (Kluger et al., 2014; Lewis & Sahrmann, 2015). However, results from (Lewis & Sahrmann, 2015) showed that the plantarflexor moment decreased as forward lean increased and this might have been caused by the subjects being positioned in slight knee flexion (Lewis & Sahrmann, 2015) in the forward lean condition which may have produced a posterior shirt of the centre of mass relative to the ankle joint centre. This effects was not observed in the current study as knee flexion remained relatively consistent across the different conditions.

What is interesting is that the data on increased forward lean are similar in many ways to the observed differences in this thesis between healthy people and people with knee OA (Chapter 4) and those observed in other research. Firstly, the current study showed that walking in 5°

of forward lean, increases the hip extensor moment by approximately 70%. Although only minimal evidence of a difference hip extensor moment was observed in chapter 4, other studies have shown that hip moment can be increased during walking in people with knee OA compared to healthy subjects (Huang et al., 2008; Liu et al., 2014). As demonstrated in this chapter, this difference could result from an altered forward lean which may be more pronounced in the more severely affected patients studied by Huang et al. (2008). This study observed that increasing forward lean led to a small, non-significant reduction in knee flexor moment during early stance. This finding is to some degree consistent with other research showing that knee extensor moment is decreased in knee people with knee OA, compared to healthy subjects (Astephen, Deluzio, Caldwell, & Dunbar, 2008; Kaufman et al., 2001; Liu et al., 2014; Manal et al., 2015). However, other research has shown no difference in knee moment (Selistre et al., 2017) between healthy people and those with knee OA. This might be because upper body position only has a small effect on knee moments.

5.5.3 Muscle activation

The results of the current study showed that there was an increase in the activity of the hamstring and gastrocnemius muscles during walking as forward trunk lean increased. These observed changes in muscle activity are consistent with the observations of an increased hip and ankle moment. Furthermore, there was minimal change in the activity of the medial and lateral quadriceps muscles when the trunk angle was increased and, again this is consistent with the observation of only a small change in knee moment. However, interestingly, there is some evidence of a small increase in medial quadricep activity with increased forward lean which seems counterintuitive given the slight decrease in knee moment. Nevertheless, effects on knee muscles and knee moments were not significant and therefore it is not clear whether they relate to any strong biomechanical effects. To date, there has been only one study which has investigated the effect of increasing forward trunk on muscle (hamstring) activity (Grasso et al., 2000). This study also observed higher hamstring muscle activity when subjects waked with a forward lean. However, participants leant forward by approximately 50° and therefore comparison of the magnitude of muscular changes in not appropriate.

It is interesting that data in this chapter are similar in many ways to the differences between healthy people and people with knee OA observed in this thesis (Chapter 4) and in previous

research. For example, a study by Astephen, Deluzio, Caldwell, Dunbar, et al. (2008) found that people with knee OA walk with 40% and 80% higher of ST and BF, respectively, compared to matched healthy subjects. In addition, data from Chapter 4 observed that people with knee OA walk approximately 50% higher hamstring activity compared to older healthy people. Interestingly, current study showed that walking with 5° more forward increased the ST and BF muscle activity by 90% and 80%, respectively. These data support the idea that increases in trunk flexion may be part of a mechanisms which underlies the alterations in hamstring activity observed in people with knee OA. However, the data presented here do not necessarily support the idea that the increase in quadriceps activity, associated with knee OA, could be related to altered trunk flexion.

5.5.4 Co-contraction

The magnitude of changes in hamstring-quadriceps co-contraction in this study are, to some degree, similar to the differences in co-contraction between individuals with knee OA and healthy subjects (Childs et al., 2004; Sritharan et al., 2016). For example, it was observed that hamstring-quadriceps co-contraction is increased by approximately 15% in people with knee OA compared to matched controls (Zeni et al., 2010b). This is actually smaller that the data presented in chapter 4 which demonstrates that patients with knee OA also walk with approximately 35% higher co-contraction. Interestingly, data in this chapter showed that walking with 5° more forward lean significant increases medial hamstring-quadriceps co-contraction by approximately 25%. Given that this figure is similar to the 35% differences observed in chapter 4, it would appear that increased forward lean may be part of the mechanism underlying increased co-contraction in people with knee OA. However, this current study did not observe major changes in quadriceps activity with increased forward lean. Therefore, other mechanisms are also likely to contribute to the observed differences in co-contraction between people with knee OA and healthy controls.

Data from recent study (Preece et al., 2018) and presented in chapter 4 show that people with knee OA tend to walk with approximately 3° more forward lean than the healthy groups. The data presented here shows a clear link between increased co-contraction and increased

forward lean. This increases in muscle co-contraction in OA population will increase joint loading and accelerate the progression of knee OA (Hodges et al., 2016a; Hubley-Kozey, Hatfield, et al., 2013). This suggests that altered upper body position may play a role in increasing joint loading and may actually negatively impact on OA. Thus, future clinical management of people with knee OA may need to focus on improving upper body position during normal walking. This idea is discussed in more detail in subsequent chapters.

5.5.5 Limitations

A number of important limitations need to be considered. First, it was difficult to instruct participants to walk with a precise trunk inclination angle. Therefore, there could have been some inter-subject differences in the trunk angle in each of the different conditions. However, we quantified mean trunk angle and the data (section 5.4.1) provided confidence in the biofeedback approach used. Furthermore, the ANOVA test for many of the variables studies showed strong effects with our within-subject design. In addition, we used a LMM which is able to take into account between-subject differences in the independent variable (trunk inclination). The results of this analysis agreed with our ANOVA testing, thus providing evidence that small differences in trunk angle between different people would be unlikely to affect the conclusions of this study.

Another limitation is that it is difficult to measure the trunk inclination as the spine is a multi-articulate structure. With the 3D modelling approach used in this thesis, it was necessary to approximate the thorax as single rigid segment. Clearly, this is not anatomically accurate, however, this was necessary as dividing the thorax into multiple segments would have introduced a level of complexity beyond the scope of the thesis. To ensure a valid and reliable measure approach, we used a technique recommended in previous research (Armand et al., 2014), which used markers placed over the IJ, T2 and T8. Compared with other possibilities, this marker configuration was found to be the optimal markers to track the movement of the thorax and to have good between-day reliability. This high repeatability was also observed in our reliability testing (Section 3.6).

5.5.6 Conclusion

This study set out to determine the effect of trunk flexion on lower limb moments, muscle activation patterns and co-contraction in healthy young people. The results of this study showed that increased trunk inclination is accompanied by a corresponding increase in the hip extensor moment and ankle plantarflexor moment, an increase in hamstring and gastrocnemius activity and an increase in hamstring-quadricep co-contraction. These data suggest the idea that alterations in the lower limb moment and muscle activation observed in people with knee OA, may be the result of alterations in trunk inclination during walking. In the subsequent chapter, we explore whether these same effects are observed in older people and those who suffer with knee OA.

Chapter Six

(Study three)

What is the biomechanical effect of instructing older healthy people and individuals with knee OA to walk with increased/decreased trunk inclination?

6.1 Introduction

Data presented in the previous chapter showed that when young healthy are instructed to walk with 5º more trunk lean, there is a clear increase in the magnitude of hip extensor moment and ankle plantar flexor moment. There is also an increase in hamstring and gastrocnemius activity and an increase in hamstring-quadricep co-contraction. This anterior trunk bend (or forward lean) will change the position and direction of the GRF and this explains the observed changes in lower limb moments and muscle activation patterns. These findings are consistent with other previous work which has also demonstrated the biomechanical effects of changing trunk inclination in healthy people (Grasso et al., 2000; Kluger et al., 2014; Leteneur et al., 2009; Lewis & Sahrmann, 2015; Saha et al., 2008). However, to date there is minimal knowledge about how alterations in forward lean could affect moments and muscle activation patterns in people with knee OA.

Previous studies have demonstrated that patients with OA exhibit a range of deviations in their gait biomechanics compared with matched healthy subjects. Data from chapter 4 and previous studies showed that the gait of people with knee OA is characterised by increased hamstring muscle activity and muscular co-contraction during the early-stance phase (Childs et al., 2004; Rudolph et al., 2007; Zeni et al., 2010a). It was suggested these differences could be a strategy to increase the body's stability during walking (Childs et al., 2004; Lewek et al., 2005), or could be a strategy to attempt to decrease knee joint loading (Andriacchi, 1994; Hubley-Kozey et al., 2006). In addition, Preece et al. (2018) and data from chapter 4 showed that people with knee OA walk with approximately 3° more trunk lean when compared to matched asymptomatic people. Importantly, the difference in trunk inclination of 5º between different conditions in the previous chapter is similar to the difference of 3º observed

between healthy people and those with knee OA. It is therefore possible that the alterations in lower limb kinetics and muscle coordination, associated with knee OA, could be the result of increased trunk flexion.

In the previous chapter (Chapter 5), the aim was to understand how young adults respond to changes in trunk inclination during walking. The result demonstrated that relatively small increases in forward trunk lean were associated with corresponding changes in lower limb kinematics, kinetics, muscle activation and muscle co-contraction. However, it is not clear that if instructing people with knee OA and the older healthy people to walk with an increase in forward lean will have the same response as young healthy people. Therefore, the aim of the current chapter was to understand the influence of increased/ decreased trunk lean on hip, knee and ankle kinetics and kinematics, along with muscle activation patterns and muscle co-contraction during walking in people with knee OA and matched healthy controls.

6.2 Research questions

The main objective of this study was to understand the biomechanical effect of instructing people with knee OA and older healthy people to walk with increased/decreased trunk forward lean. Five research questions were addressed in this chapter. This objective was achieved through five separate research questions:

Q 1 What is the effect of increasing/decreasing trunk inclination on sagittal hip, knee and ankle moments in people with knee OA and older healthy people during walking?

Q 2 What is the effect of increasing/decreasing trunk inclination on gastrocnemius, quadriceps and hamstring muscle activities in people with knee OA and older healthy people during walking?

Q 3 What is the effect of increasing/decreasing trunk inclination on gastrocnemius, quadriceps and hamstring muscle co-contraction in people with knee OA and older healthy people during walking?

Q 4 What is the effect of increasing/decreasing trunk inclination on sagittal hip, knee and ankle angles in people with knee OA and older healthy people during walking?

Q 5 What is the effect of increasing/decreasing trunk inclination on spatiotemporal parameters (speed and step length) in people with knee OA and older healthy people during walking?

6.3 Methods

As with the previous study, participants were instructed to walk at their self-selected speed normally and then with two different trunk lean conditions (normal +5° and normal and -5°). For this study, only two addition conditions were tested as pilot testing showed that older people and those with knee OA found it very challenging to walk with 10° more trunk inclination that their normal pattern. Biofeedback on trunk inclination was provided to guide the participants to walk at the target trunk angle. A full description of the methods used for this study were provided in the method chapter.

6.3.1 Sample and population

All testing was carried out in the Podiatry lab in the University of Salford. The criteria for inclusion and exclusion have been given before (Section 3.2). Forty subjects were recruited to participate in the study: 20 people with knee OA and 20 matched healthy control participants (Table 6-1). Independent t-tests were used to compare the demographic characteristics between the two groups and showed that there was no significant difference ($p > .05$) in any of the demographic factors.

Table 6-1 Participants' characteristics for both groups: people with knee OA and healthy people. Values are the mean ± Standard Deviation (SD).

	Knee OA people	**Older healthy people**
No. of subjects	20	20
Age (Years)	56.0 (8.7)	57.2 (8.7)
Height (M)	1.7 (.07)	1.7 (.06)
Mass (kg)	83.1 (14.4)	80 (11.3)
BMI (kg/m²)	28.7 (4.9)	27.4 (3.9)

6.3.2 Derivation of outcome measures

Outcome measures for each of the research question were similar to the derivation of outcome measures applied in Chapter 4. For each question, the 15–25% period of the stance phase was chosen for the kinematic and kinetics data, and the 10–20% period used for muscle activation and co-contraction (this difference in the time window of interest takes account of the electromechanical delay (EMD); see Section 3.7.1). This window was chosen based on modelling studies showing the early-stance period as the period of peak load on the knee joint during walking (Brandon et al., 2014; Sritharan et al., 2016). For more detail see Section 3.7.

6.3.3 Statistical analysis

In order to answer the above questions, a two-way mixed-design analysis of variance (ANOVA) and linear mixed model (LMM) analyses were performed. Effect sizes were determined using the partial eta squared, with .01, .06 and .14 defined as small, medium and large, respectively (Cohen, 1992; Richardson, 2011).

Two-way mixed ANOVA

Statistical analysis of results was carried out using the statistical package for social sciences (SPSS) version 24 for Windows. The two-way mixed design ANOVA was used to examine the effect of trunk inclination (+5°, NW and -5°) and subject (knee OA and older healthy) for each dependent variable, averaged across 15–25% of the stance phase in kinematics/kinetics data and 10–20% of the stance phase in EMG data. A two-way mixed effects ANOVA test can be used to understand if there are main effects of each of the two independent variables and also if there is an interaction between the two independent variables (group and trunk inclination). As analysis comparing group (healthy vs OA) was presented in chapter 4, this was omitted from this chapter. However, the results of the ANOVA testing for the main effect of trunk inclination and the interaction (group x trunk inclination) are presented for all dependent variables.

Five assumptions should be met in order to use the two-way mixed ANOVA. Firstly, the data should be normally distributed, as assessed by Shapiro-Wilk's test of normality ($P > .05$). Secondly, there should be no significant outliers in any tested variables, as checked by inspection of a box and whiskers boxplot. Following the parametric analysis, for between-subject analysis the homogeneity of variances and covariance were assessed by Levene's test and by Box's M test at ($P > .05$), respectively. In addition, for within subjects, Mauchly's test of sphericity was performed ($P > .05$). If the ANOVA showed a significant result, the Bonferroni adjustment was used to investigate possible pairwise differences. If the data showed that there was non-normally distributed due to a moderately positively skewed data to normality, a square root transformation was applied. The test was performed on the examined sample with the alpha level .05. However, if there was a violation of any above-mentioned assumption, a non-parametric test was performed (Friedman test).

Linear mixed model (LMM)

Linear mixed effects analysis was completed using the lme4 package (Bates, Maechler, Bolker & Walker, 2015) in R (R Core Team, 2017) to determine the effect of trunk angle and subject group on each outcome variable. Both trunk angle and subject group were defined as fixed effects. The model also included "Subject" as a random effect. Random intercepts were assumed for each subject, thus resolving the non-independence problem of multiple measures from the same subject, and accounting for baseline differences in trunk angle. In addition, the model assumes by-subject random slopes for the effect of the outcome variable, meaning the model can expect the effect of the outcome variable to be different for different subjects. The LMM is thus defined as follows:

$$\text{outcome} \sim \text{Trunk Angle} + \text{Subject Group} + (1 + \text{Trunk Angle} \mid \text{Subject})$$

where "Trunk Angle" is the fixed effect, "| Subject" implies random intercepts, and "1 + Trunk Angle" dictates the by-subject random slopes.

Prior to analysis, the significance of an interaction between the two fixed effects (trunk angle and subject group) was tested using a likelihood ratio test; where two models (one with and one without the interaction) were compared using ANOVA (model without intercept, model with intercept). A significant result would imply that there was inter-dependence between

trunk angle and subject group where the effect of trunk angle on the outcome variable was somehow modulated by the subject group (healthy older versus knee OA). The results showed that the interaction was not significant for all of the outcome variables. Therefore, it was not included in the model.

No obvious deviations from homoscedasticity or normality were identified by visual inspection of the residual plots. In addition, to test the significance of each of the fixed effects, a likelihood ratio test was used to compare two models, one with the fixed effect of interest (full model) and one without the fixed effect of interest (reduced model). This was done using ANOVA (reduced model, full model), where a significant result implies that the missing fixed effect (Trunk Angle or Subject Group) has a significant effect on the outcome variable.

Full model:

$$\text{outcome} \sim \text{Trunk Angle} + \text{Subject Group} + (1 + \text{Trunk Angle} \mid \text{Subject})$$

Reduced models:

$$\text{outcome} \sim \text{Trunk Angle} + (1 + \text{Trunk Angle} \mid \text{Subject})$$

$$\text{outcome} \sim \text{Subject Group} + (1 + \text{Trunk Angle} \mid \text{Subject})$$

Here, “Trunk Angle” and “Subject Group” are the fixed effects, “| Subject” implies random intercepts, and “1 + Trunk Angle” dictates the by-subject random slopes.

6.4 Results

6.4.1 Trunk angle

Figure 6-1 and Figure 6-2 illustrate the differences in trunk angle during the three different trunk inclinations: -5°, normal and +5° for people with knee OA (Figure 6-1) and healthy older subjects (Figure 6-2). Table 6-2 provides the trunk angle data or the people with knee OA and older healthy subjects.

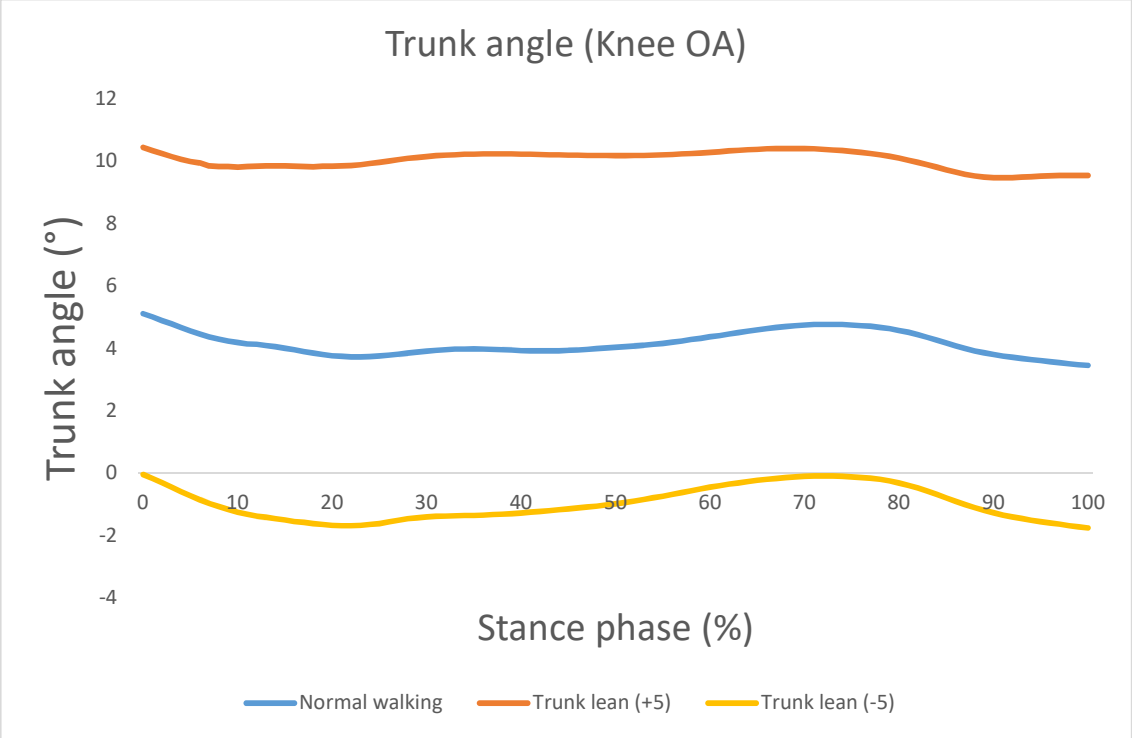


Figure 6-1 The ensemble average, across all 20 people with knee OA, of the mean of the sagittal trunk angle for normal walking and the two different trunk lean conditions.

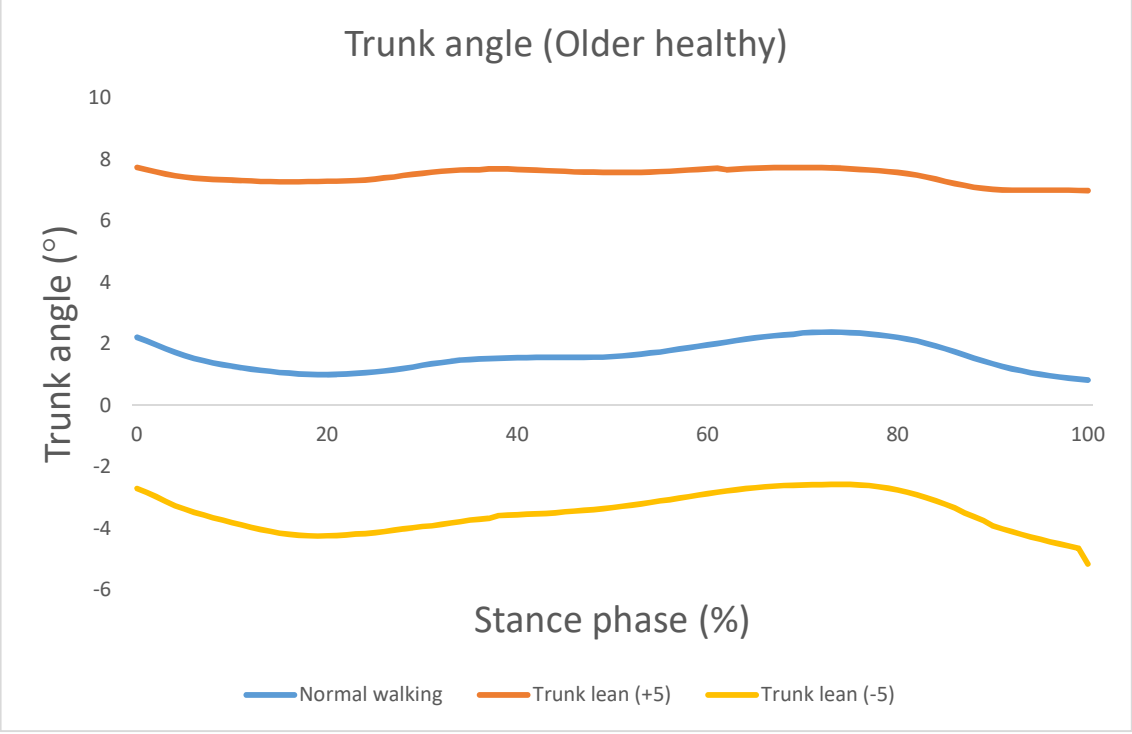


Figure 6-2 The ensemble average, across all 20 older healthy people, of the mean sagittal trunk angle for normal walking and the two different trunk lean conditions.

Table 6-2 The mean and standard deviation (SD) of the trunk angle (°) during normal walking and with different trunk lean over the full gait cycle for both groups.

	Normal walking	Trunk lean (+5°)	Trunk lean (-5°)
OA group	4.2° (1.9°)	10.3° (1.9°)	-1.09° (2.2°)
Older group	2.4° (2.8°)	7.1° (2.0°)	-3.3° (2.8°)

6.4.2 Sagittal moment

Hip moment

The ensemble curves for sagittal hip moment are shown in Figure 6-3 (knee OA) and Figure 6-4 (older healthy). There was a clear rise in the magnitude of hip extensor moment across the stance phase during walking with +5° trunk lean, and a corresponding decrease in hip moment when walking with -5° trunk lean. The two-way mixed ANOVA analysis across the 15–25% period of the stance phase showed a significant effect for trunk lean ($P < .05$) but no interaction was observed ($P = .679$) (Table 6-3). Post hoc results showed that there were significant differences between NW and both trunk inclination angles (Table 6-4). Similar to the ANOVA test, the results from the LMM showed that trunk angle significantly affected hip moment ($\chi^2(1) = 80.35$, $p < .001$). Specifically, increasing trunk angle by 1 degree increased the hip moment by .020 (.027) Nm/kg in the older healthy group, and a further .001 (.055) Nm/kg in the knee OA group.

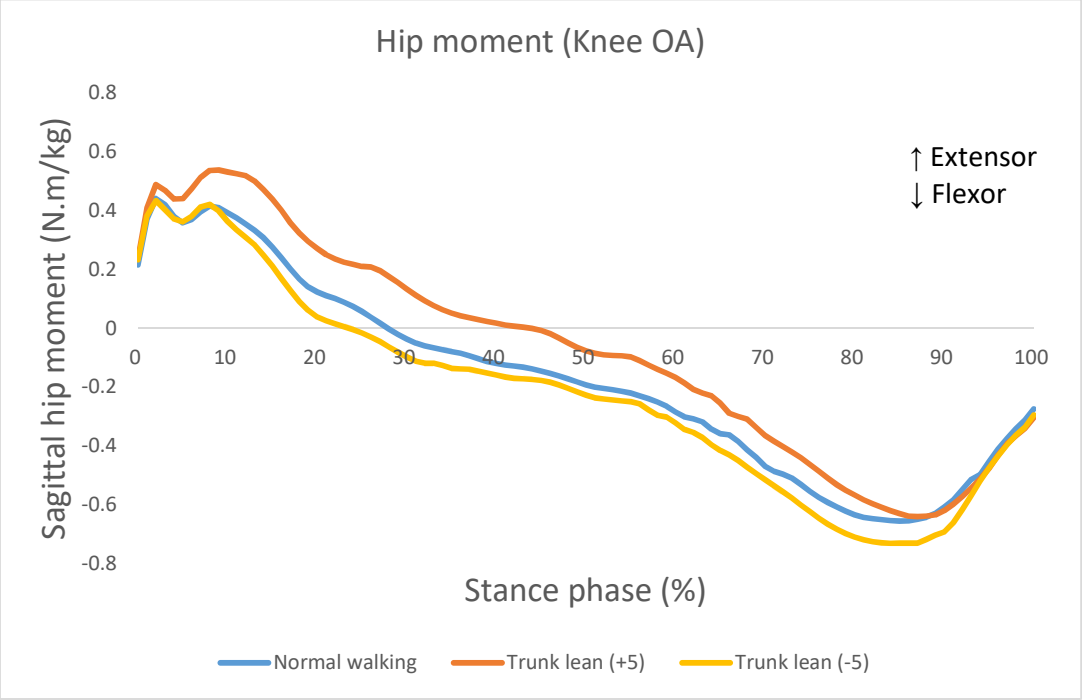


Figure 6-3 The ensemble average, across all 20 people with knee OA, of the mean sagittal hip moment for normal walking and the two different trunk lean conditions.

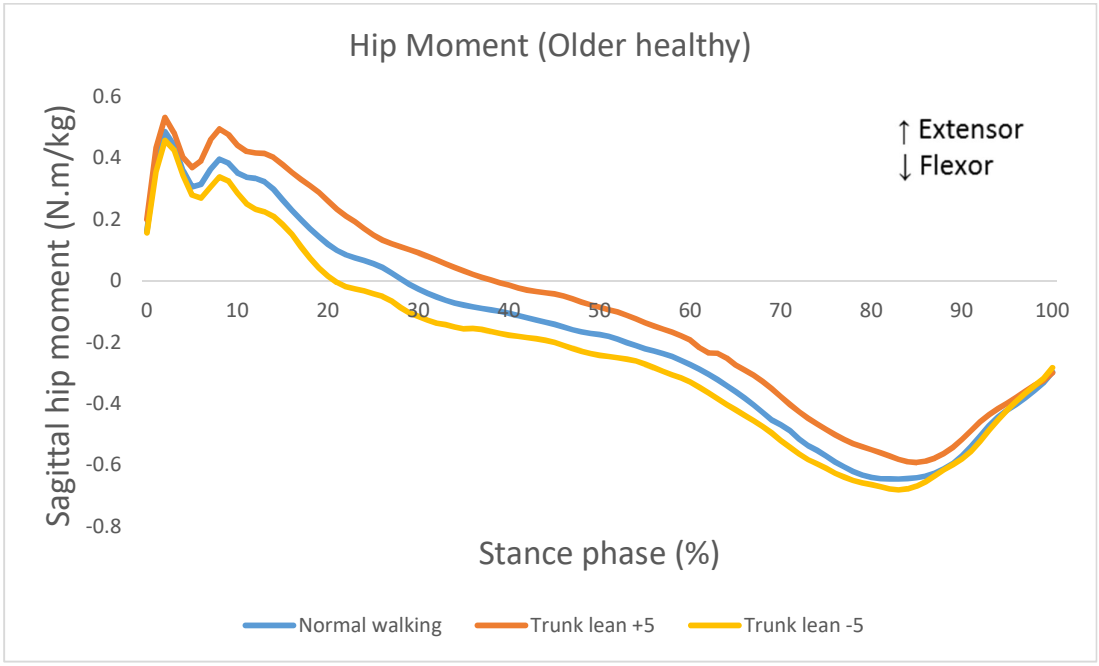


Figure 6-4 The ensemble average, across all 20 older healthy subjects, of the mean sagittal hip moment for normal walking and the two different trunk lean conditions.

*Table 6-3 Summary data for sagittal hip moment (Nm/kg) (average across 15–25% of stance phase) for the different trunk conditions . * indicated a significant difference between this condition and normal walking as identified by the post hoc test (p<0.05).*

	Normal walking	Trunk lean (+5°)	Trunk lean (-5°)	Significant test	Effect size
OA group	.15 (.19)	.31 (.18)*	.08 (.19)*	Effect of trunk inclination P = .00	.75
Older group	.14 (.17)	.27 (.19)*	.06 (.15)*	Interaction: P = .679	.008

Knee moment

Figure 6-5 and Figure 6-6 illustrate the knee moment data. There was a decrease in knee extensor moment during walking with +5° trunk lean compared to normal walking in both groups during the 10–60% period of the stance and a corresponding increase in knee extensor moment during walking with -5° trunk lean in both groups in the same period. Analysis from a two-way mixed ANOVA across the 15–25% period of the stance found that there was a significant effect of trunk lean (P = .00). However, post hoc tests found no significant changes in knee moment during walking with different trunk leans (P > .05). No interaction effect was identified (P = .191) (Table 6-4).

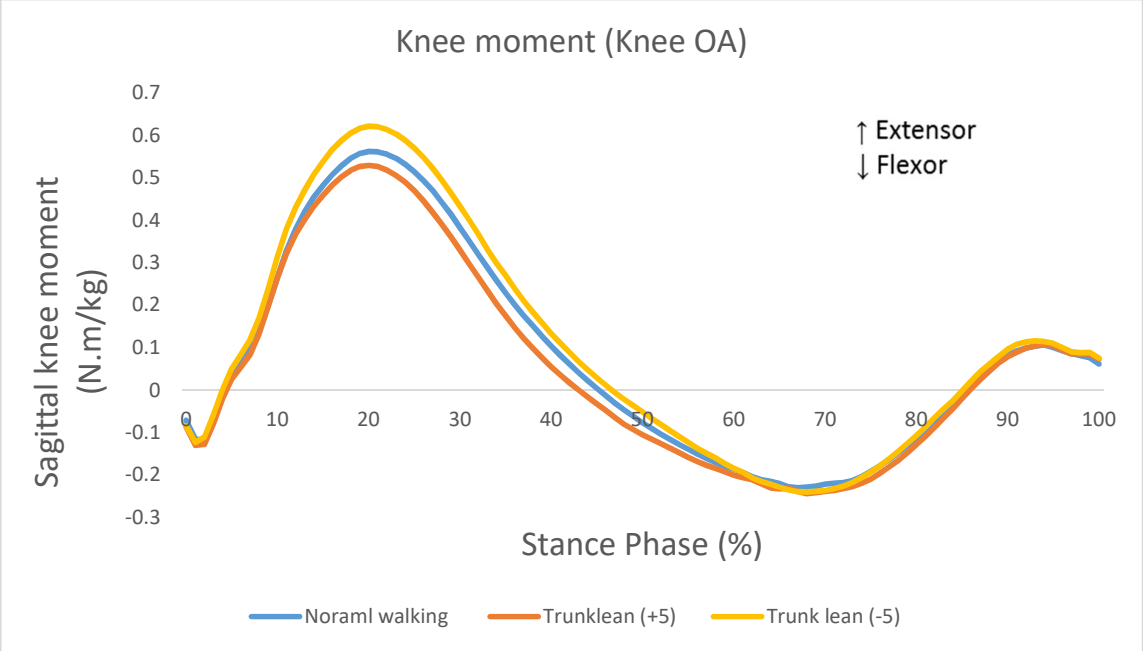


Figure 6-5 The ensemble average, across all 20 people with knee OA, of the mean sagittal knee moment for normal walking and the two different trunk lean conditions.

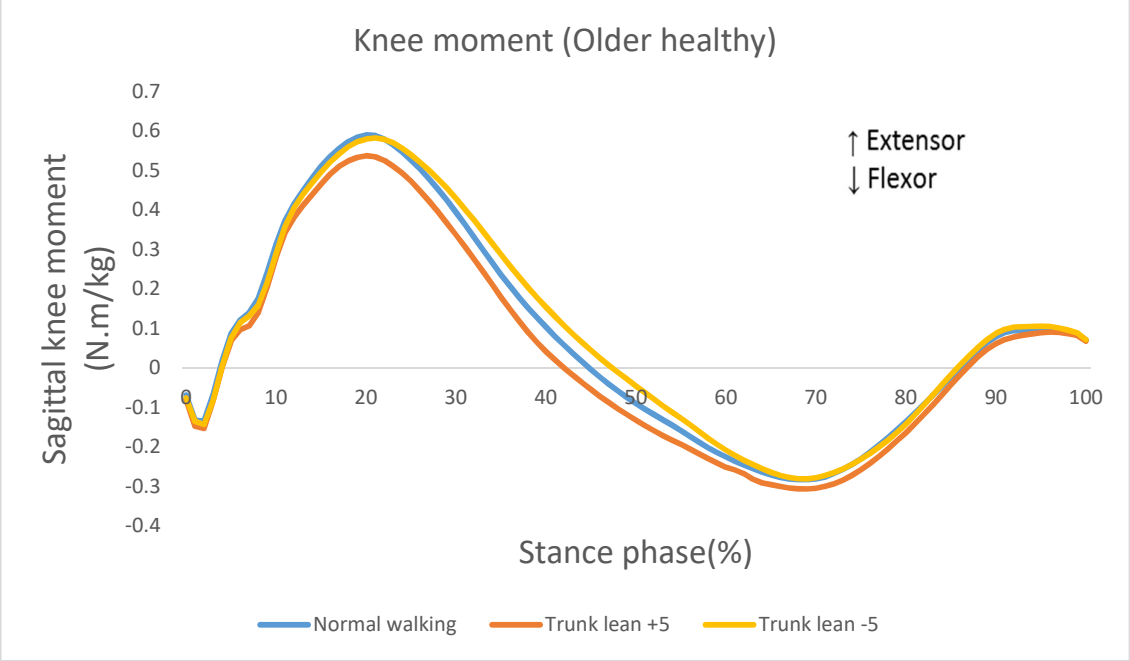


Figure 6-6 The ensemble average, across all 20 older healthy subjects, of the mean sagittal knee moment for normal walking and the two different trunk lean conditions.

Table 6-4 Summary data for sagittal knee moment (Nm/kg) (average across 15–25% of stance phase) for the different trunk conditions .

	Normal walking	Trunk lean (+5°)	Trunk lean (-5°)	Significant test	Effect size
OA group	.52 (.27)	.49 (.28)	.58 (.29)	Effect of trunk inclination P = .00	.14
Older group	.55 (.21)	.50 (.22)	.54 (.22)	Interaction: P = .191	.043

Ankle moment

In general, there was an increase in the magnitude of plantarflexor moment in the mid-stance phase as trunk lean was increased by 5° and a corresponding decrease when walking with -5° trunk lean in both groups (Figure 6-7 and Figure 6-8). Average values analysed by a two-way mixed ANOVA test (15–25% of stance) demonstrated that there was a significant effect for trunk lean (P = .00) but no interaction effect was observed (P = .959). Pairwise comparison showed significant differences between NW and -5° trunk lean in the knee OA group, and between NW and both trunk conditions in the older group. Further to the ANOVA results, the LMM for mean ankle moment demonstrated that trunk angle significantly affected ankle moment ($\chi^2(1) = 34.19$, $p < .001$). Specifically, increasing trunk angle by 1 degree increased the ankle moment by .007 (.046) Nm/kg in the healthy older group, and a further .001 (.030) Nm/kg in the knee OA group.

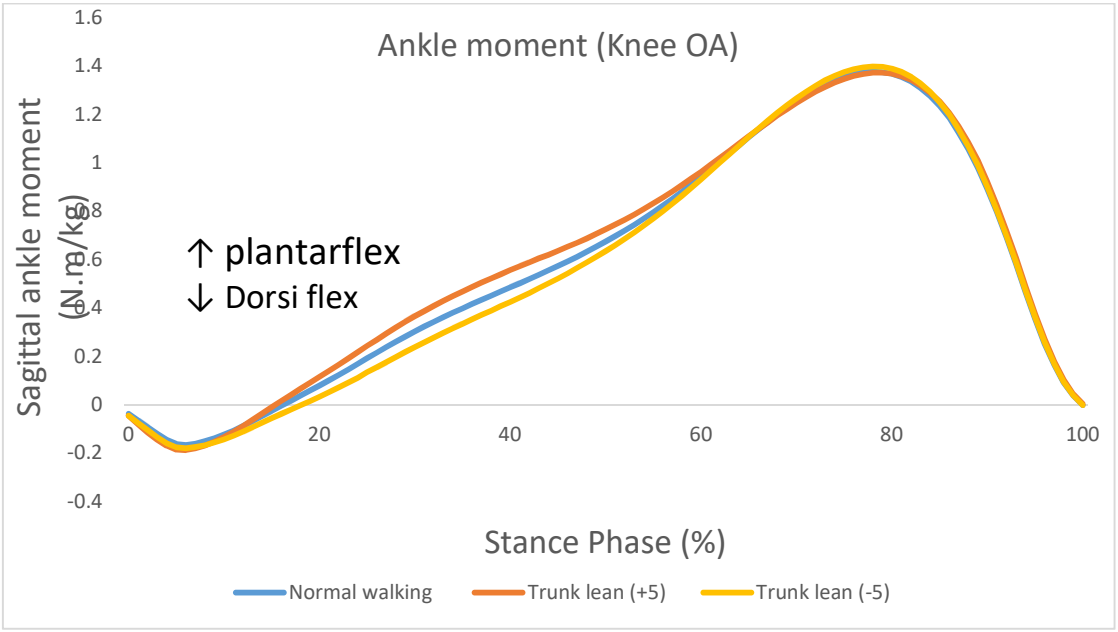


Figure 6-7 The ensemble average, across 20 people with knee OA, of the mean sagittal ankle moment for normal walking and the two different trunk lean conditions.

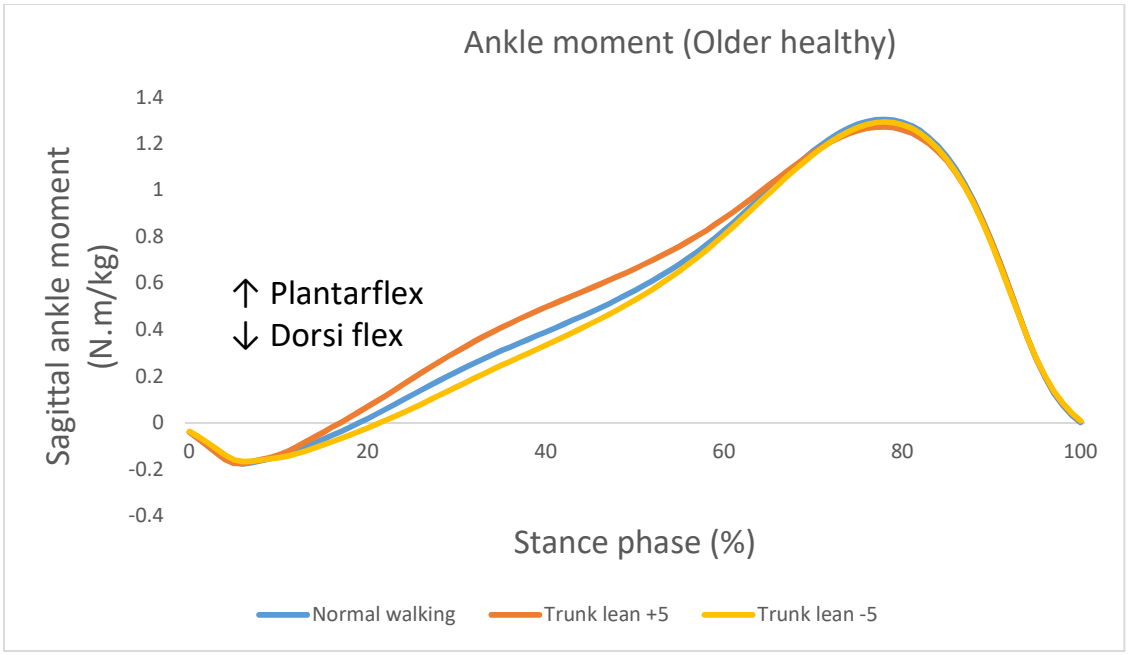


Figure 6-8 The ensemble average, across all 20 older healthy subjects, of the mean sagittal ankle moment for normal walking and the two different trunk lean conditions.

*Table 6-5 Summary data for sagittal ankle moment (Nm/kg) (average across 15–25% of stance phase) for the different trunk conditions . * indicated a significant difference between this condition and normal walking as identified by the post hoc test (p<0.05).*

	Normal walking	Trunk lean (+5°)	Trunk lean (-5°)	Significant test	Effect size
OA group	.06 (.11)	.10 (.15)	.02 (.10)*	Effect of trunk inclination P = .00	.45
Older group	.005 (.09)	.04 (.12)*	-.03 (.09)*	Interaction: P = .959	.001

6.4.3 Muscle activation

Medial gastrocnemius (MG)

Figure 6-9 and Figure 6-10 show that as trunk angle was increased, there was a slight increase in the MG muscle activity pattern in the early stance phase in both groups. However, as the trunk was decreased, only minimal change was observed in the MG muscle activity pattern in the early stance phase in the older group. From Table 6-6 , the results from mean MG muscle activity across 10–20% of the stance phase show that there was a significant effect for trunk lean (P <.005) and no interaction effect between groups was observed (P = .158). Post hoc tests showed that there were significant differences between the NW and +5° trunk lean in the knee OA group and between NW and both trunk inclinations in the older healthy group. The result from LMM demonstrated that trunk angle significantly affected MG muscle activity ($\chi^2(1) = 21.62$, $p < .001$). Specifically, increasing trunk angle by 1 degree increased MG muscle activity by .004 (.006) in the older healthy group, and a further .001 (.019) in the knee OA group.

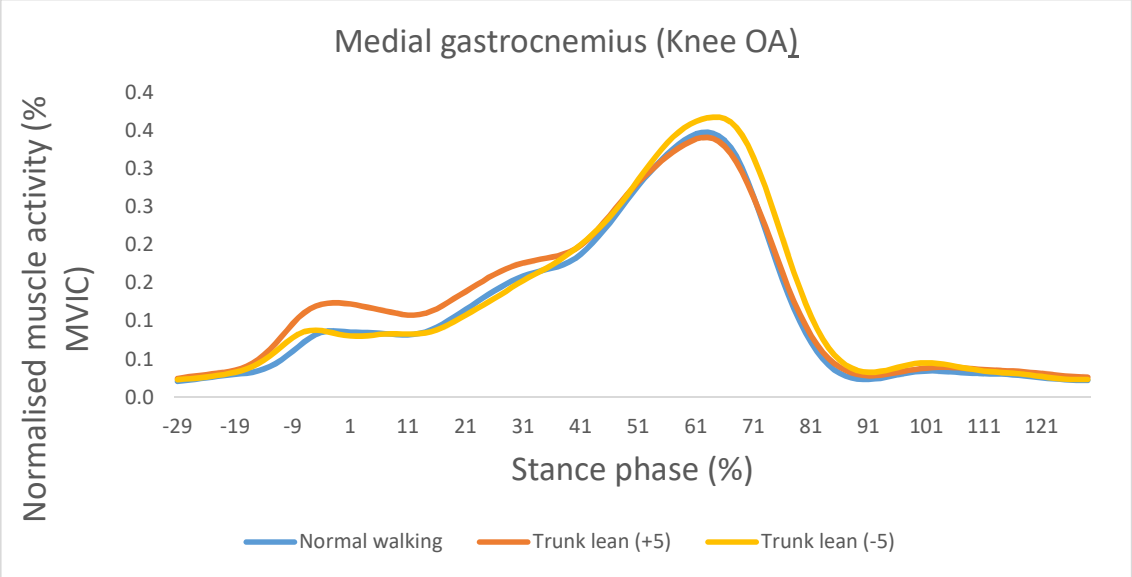


Figure 6-9 The ensemble average, across 20 people with knee OA, of the mean MG muscle activity normalised to %MVIC for normal walking and the two different trunk lean conditions.

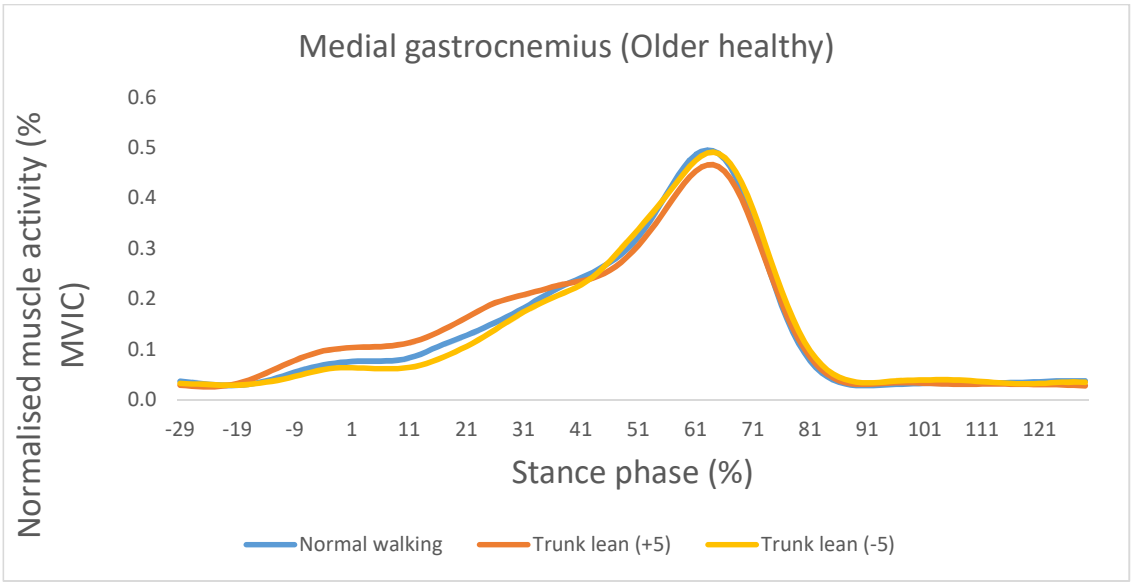


Figure 6-10 The ensemble average, across 20 older healthy people, of the mean MG muscle activity normalised to %MVIC for normal walking and the two different trunk lean conditions.

*Table 6-6 Summary data for MG muscle activation (MVIC: proportion of the MVIC) (average across 10–20% of stance phase) for the different trunk conditions . * indicated a significant difference between this condition and normal walking as identified by the post hoc test (p<0.05).*

	Normal walking	Trunk lean (+5°)	Trunk lean (-5°)	Significant test	Effect size
OA group	.08 (.05)	.10 (.07)*	.08 (.05)	Effect of trunk inclination P = .00	.35
Older group	.09 (.06)	.11 (.06)*	.06 (.03)*	Interaction: P = .158	.049

Lateral gastrocnemius (LG)

The ensemble curves in Figure 6-11 and Figure 6-12 show LG muscle activity. Similar to the MG, there was a significant effect of trunk lean (P < .005) and no interaction effects between groups (P = .413). Post hoc testing showed that the only significant difference was between NW and the +5° condition in the knee OA group (Table 6-7). Similar to the ANOVA result, LMM analysis showed that trunk angle significantly affected LG muscle activity ($\chi^2(1) = 19.07$, $p < .001$). Specifically, increasing trunk angle by 1 degree increased LG muscle activity by .004 (.007) in the older healthy group, and a further .001 (.018) in the people with knee OA.

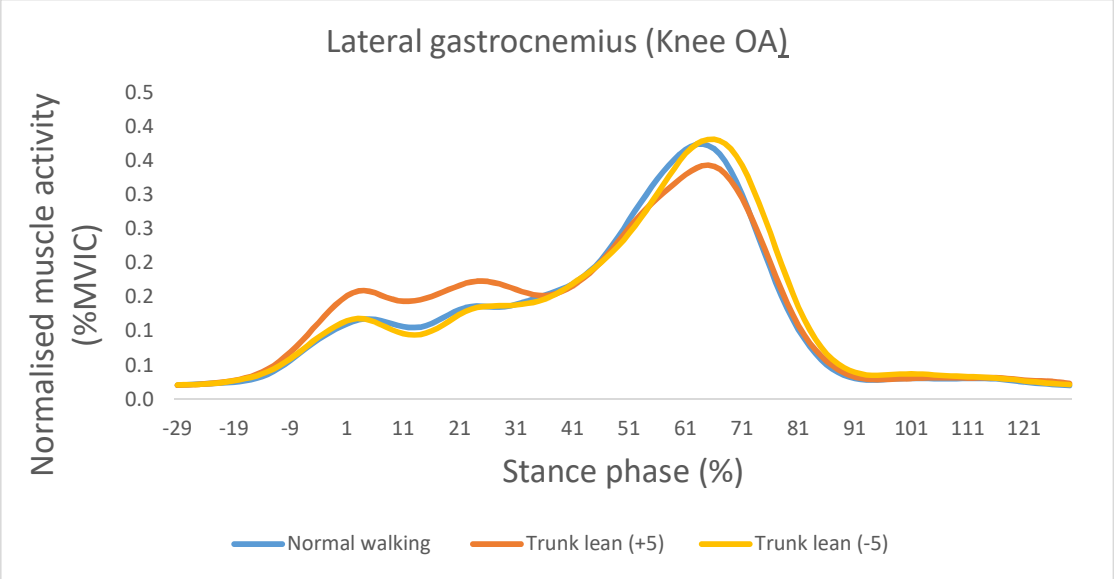


Figure 6-11 The ensemble average, across 20 people with knee OA, of the mean LG muscle activity normalised to %MVIC for normal walking and the two different trunk lean conditions.

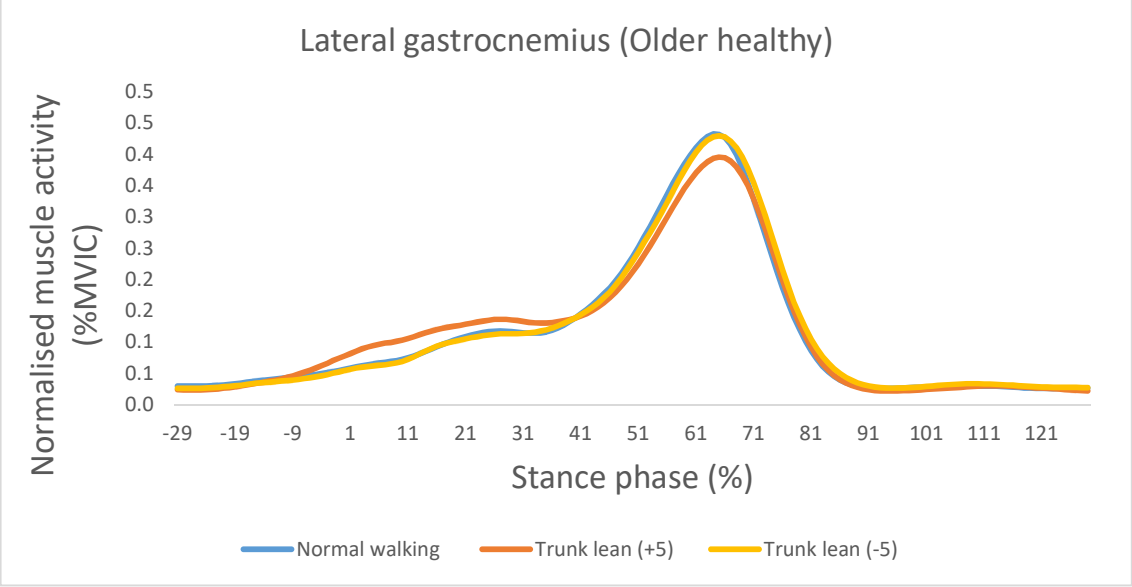


Figure 6-12 The ensemble average, across 20 older healthy people, of the mean LG muscle activity normalised to %MVIC for normal walking and the two different trunk lean conditions.

*Table 6-7 Summary data for LG muscle activation (MVIC: proportion of the MVIC) (average across 10–20% of stance phase) for the different trunk conditions . * indicated a significant difference between this condition and normal walking as identified by the post hoc test (p<0.05).*

	Normal walking	Trunk lean (+5°)	Trunk lean (-5°)	Significant test	Effect size
OA group	.10 (.06)	.14 (.07)*	.09 (.05)	Effect of trunk inclination P = .00	.32
Older group	.08 (.05)	.11 (.06)	.07 (.05)	Interaction: P = .413	.021

Vastus medialis oblique (VMO)

Figure 6-13 and Figure 6-14 show VMO muscle activity during walking and with +5° and -5° trunk lean in knee OA and healthy subjects, respectively. These plots illustrate clearly that there was very little change in VMO muscle activity between the different trunk lean conditions. For the effect of the VMO activity across the period of interest, 10–20% of the stance phase, there was no significant effect for trunk lean (P = .792) and no significant interaction was observed (P = .445) (Table 6-8).

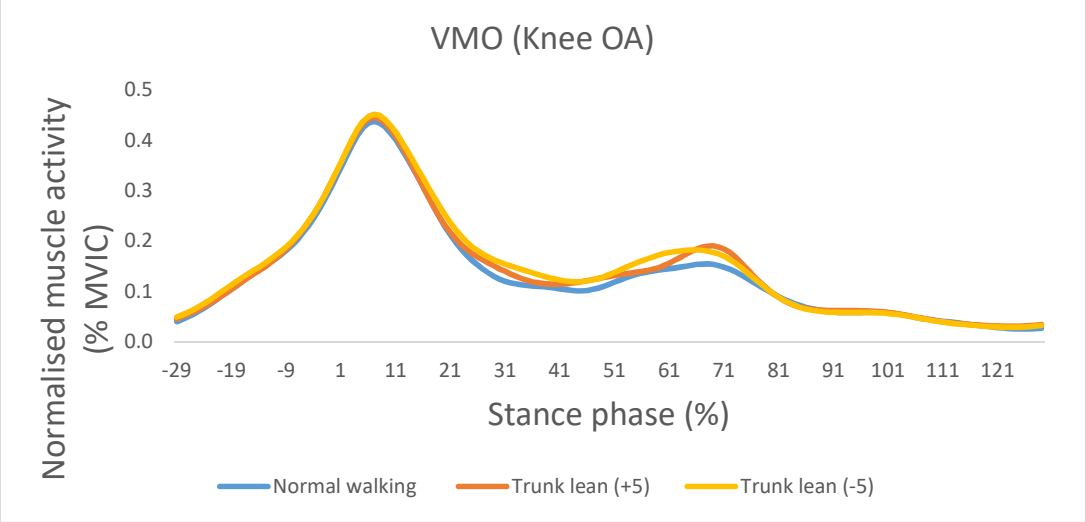


Figure 6-13 The ensemble average, across 20 people with knee OA, of the mean VMO muscle activity normalised to %MVIC for normal walking and the two different trunk lean conditions.

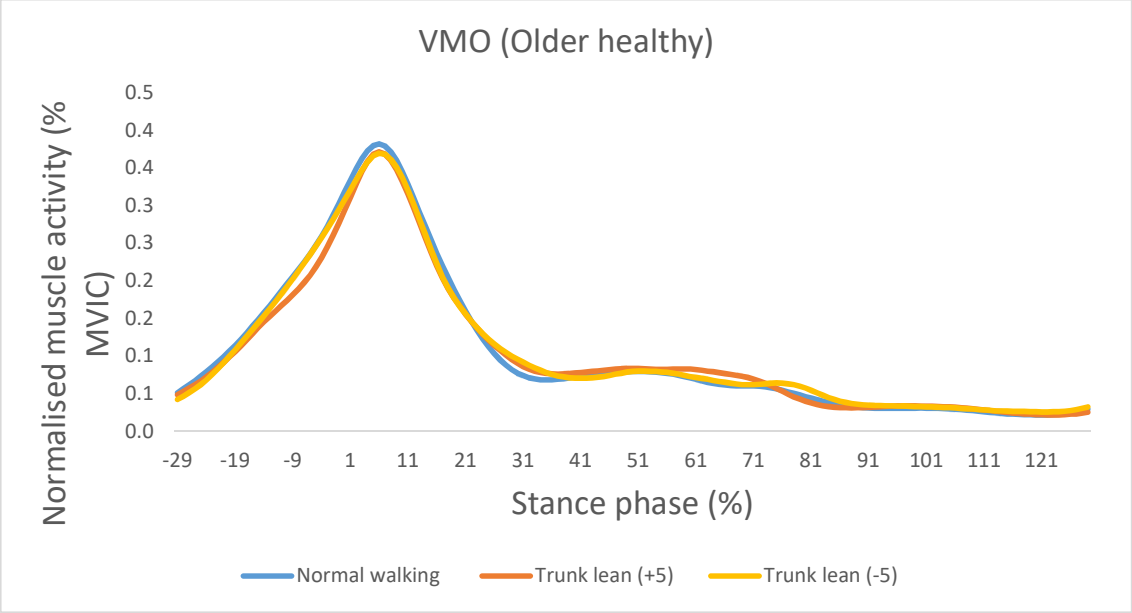


Figure 6-14 The ensemble average, across 20 older healthy people, of the mean VMO muscle activity normalised to %MVIC for normal walking and the two different trunk lean conditions.

Table 6-8 Summary data for VMO muscle activation (MVIC: proportion of the MVIC) (average across 10–20% of stance phase) for the different trunk conditions .

	Normal walking	**Trunk lean (+5°)**	**Trunk lean (-5°)**	**Significant test**	**Effect size**
OA group	.33 (.15)	.34 (.14)	.35 (.20)	Effect of trunk inclination P = .792	.004
Older group	.22 (.13)	.21 (.13)	.21 (.11)	Interaction: P = .445	.019

Vastus lateralis oblique (VLO)

The plots below show the ensemble average of normalised VLO muscle activity for both the knee OA and healthy groups (Figure 6-15, Figure 6-16). These profiles were similar to those of the VMO muscle activity, with minimal differences across the different trunk lean

conditions. The data in Table 6-9 indicates that there was no significant effect for trunk lean (P = .14) and no significant interaction between groups (P = .352).

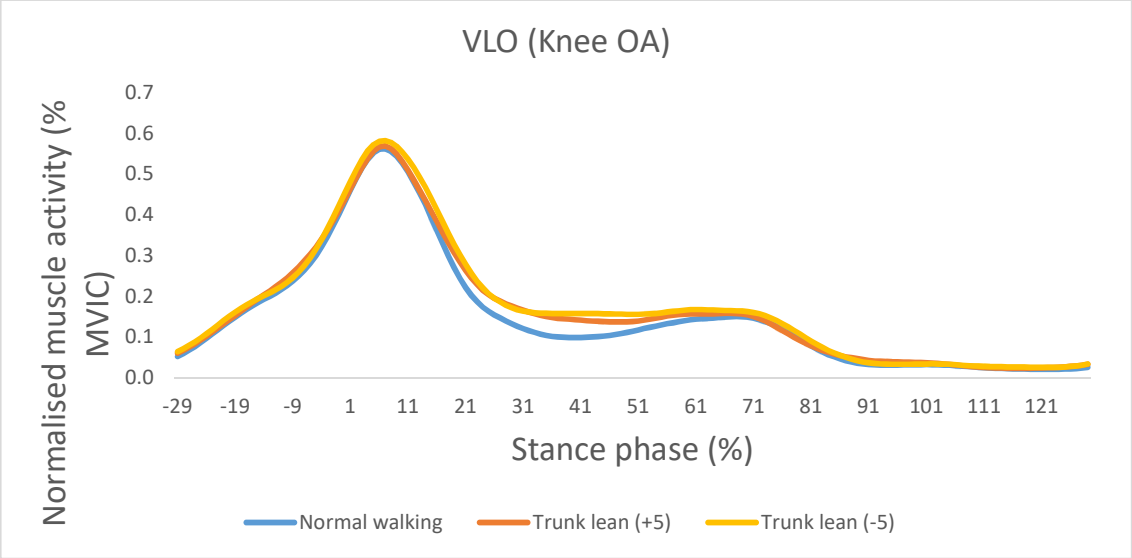


Figure 6-15 The ensemble average, across 20 people with knee OA, of the mean VLO muscle activity normalised to %MVIC for normal walking and the two different trunk lean conditions.

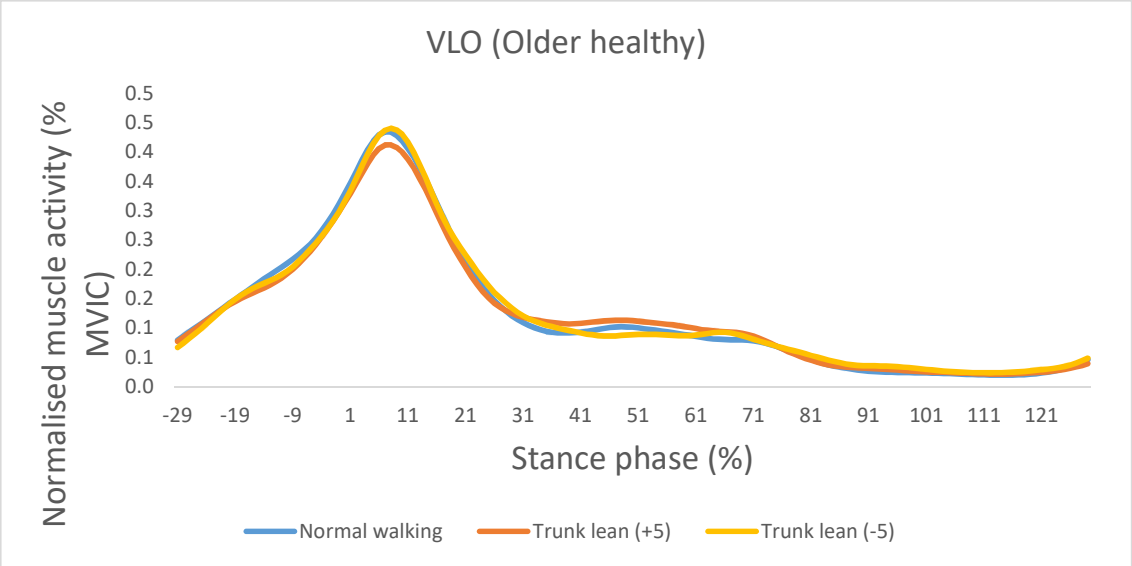


Figure 6-16 The ensemble average, across 20 older healthy people, of the mean VLO muscle activity normalised to %MVIC for normal walking and the two different trunk lean conditions.

Table 6-9 Summary data for VLO muscle activation (MVIC: proportion of the MVIC) (average across 10–20% of stance phase) for the different trunk conditions .

	Normal walking	Trunk lean (+5°)	Trunk lean (-5°)	Significant test	Effect size
OA group	.40 (.20)	.42 (.24)	.46 (.28)	Effect of trunk inclination P = .14	.050
Older group	.32 (.26)	.31 (.24)	.33 (.25)	Interaction: P = .352	.027

Semitendinosus (ST)

Ensemble average profiles for ST muscle are shown below. There was a clear rise in the magnitude of the ST muscle activity pattern across the whole stance phase when walking with a +5° trunk lean. The ANOVA test showed that there was significant effect for trunk lean (P < .005) but no interaction (P = .828) (Table 6-10). Pairwise comparison showed that there was a significant increase between the NW and +5° trunk lean in both groups. Data from LMM showed that trunk angle significantly affected ST muscle activity ($\chi^2(1) = 21.95$, $p < .001$). Specifically, when trunk angle increased by 1 degree there was an increase in ST muscle activity of .011 (.049) in the older healthy group, and a further .002 (.038) in the knee OA patients.

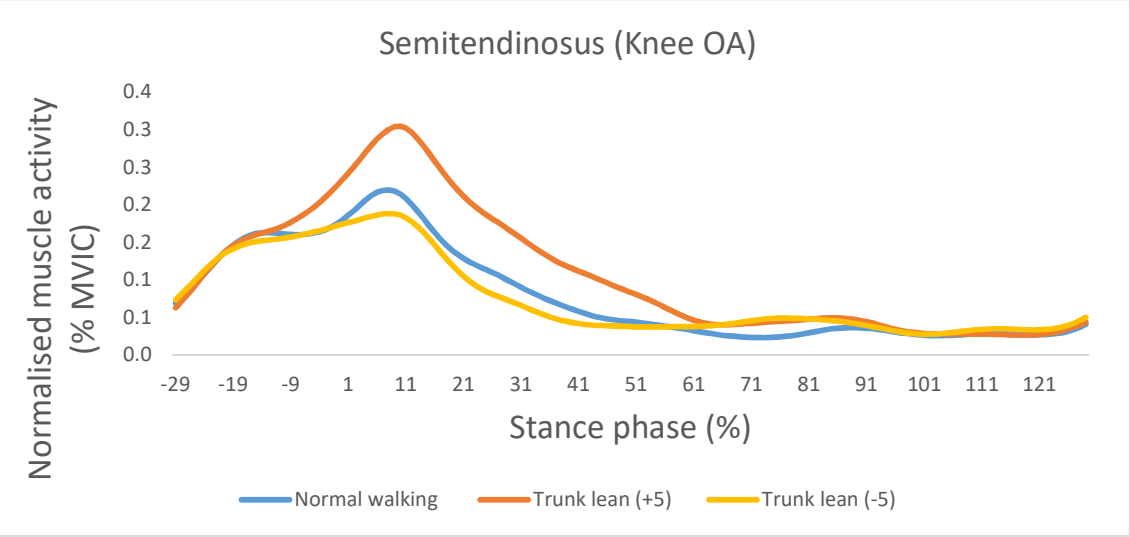


Figure 6-17 The ensemble average, across 20 people with knee OA, of the mean ST muscle activity normalised to %MVIC for normal walking and the two different trunk lean conditions.

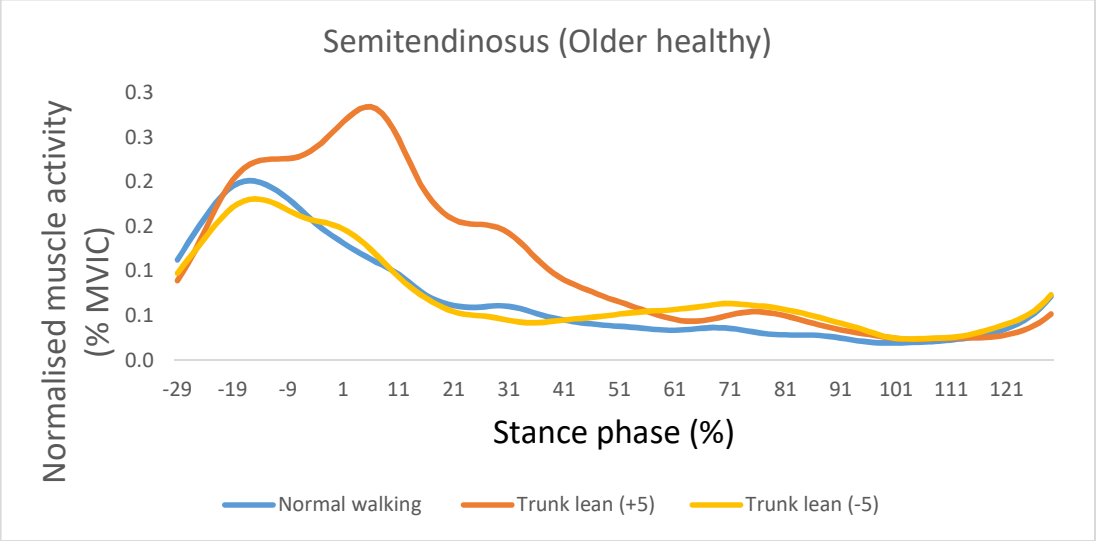


Figure 6-18 The ensemble average, across 20 older healthy subjects, of the mean ST muscle activity normalised to %MVIC for normal walking and the two different trunk lean conditions.

*Table 6-10 Summary data for ST muscle activation (MVIC: proportion of the MVIC) (average across 10–20% of stance phase) for the different trunk conditions . * indicated a significant difference between this condition and normal walking as identified by the post hoc test (p<0.05).*

	Normal walking	Trunk lean (+5°)	Trunk lean (-5°)	Significant test	Effect size
OA group	.16 (.13)	.27 (.25)*	.13 (.15)	Effect of trunk inclination P = .00	.38
Older group	.08 (.06)	.22 (.20)*	.07 (.08)	Interaction: P = .828	.002

Biceps femoris (BF)

BF muscle data is shown below and illustrates a clear trend of increasing magnitude in the BF muscle activity during walking with a +5° trunk lean in both groups. However, as the trunk lean decreased, only a minimal decrease was observed. The ANOVA result showed a significant effect of trunk lean (P <.005) and no interaction (P = .15) (Table 6-11). Post hoc test showed that there was a significant increase between the NW and +5° trunk lean in both groups. LMM also showed increasing trunk lean had a significant effect on BF muscle activity

($\chi^2(1) = 29.08$, $p < .001$). Specifically, when trunk lean was increased by 1 degree, BF muscle activity increased by .005 (.048) in the older healthy group, and a further .001 (.022) in the patient group.

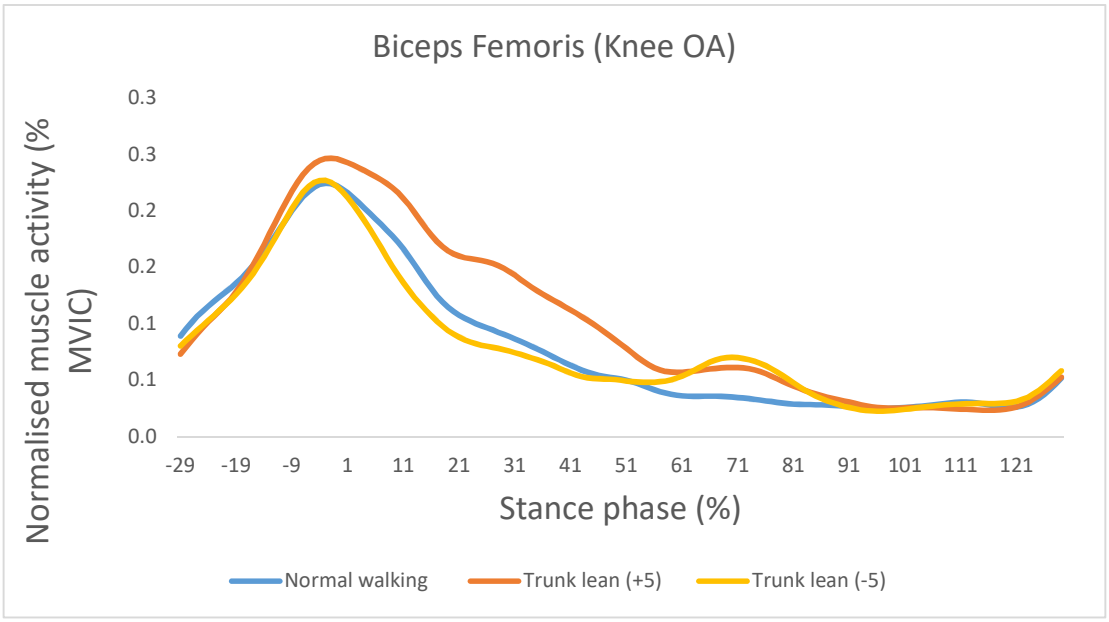


Figure 6-19 The ensemble average, across 20 people with knee OA, of the mean BF muscle activity normalised to %MVIC for normal walking and the two different trunk lean conditions.

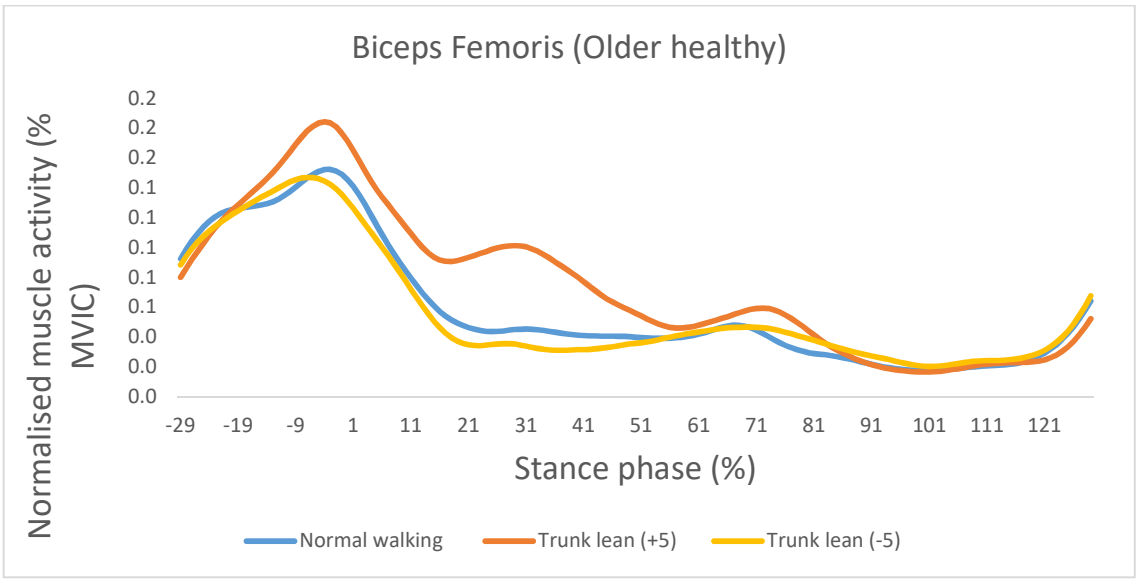


Figure 6-20 The ensemble average, across 20 older healthy people, of the mean BF muscle activity normalised to %MVIC for normal walking and the two different trunk lean conditions.

*Table 6-11 Summary data for BF muscle activation (MVIC: proportion of the MVIC) (average across 10–20% of stance phase) for the different trunk conditions . * indicated a significant difference between this condition and normal walking as identified by the post hoc test (p<0.05).*

	Normal walking	Trunk lean (+5°)	Trunk lean (-5°)	Significant test	Effect size
OA group	.14 (.10)	.18 (.13)*	.11 (.09)	Effect of trunk inclination P = .00	.44
Older group	.06 (.04)	.09 (.05)*	.05 (.03)	Interaction: P = .156	.048

6.4.4 Muscle co-contraction

Medial gastrocnemius and vastus medialis oblique (MGVMO) muscle co-contraction

Table 6-12 presents the muscle co-contraction between the MGVMO, averaged across 10–20% of the stance phase. Although the results from ANOVA showed that there was a significant effect of trunk lean on the MGVMO (P = .03), post hoc tests showed that there were no significant differences between the different trunk leans conditions in either groups. ANOVA showed that there was no interaction between the two groups (P = .282) (Table 6-12).

Table 6-12 Summary data for MGVMO co-contraction (MVIC: proportion of the MVIC) (average across 10–20% of stance phase) for the different trunk conditions .

	Normal walking	Trunk lean (+5°)	Trunk lean (-5°)	Significant test	Effect size
OA group	.63 (.02)	.66 (.03)	.64 (.03)	Effect of trunk inclination P = .03	.08
Older group	.54 (.02)	.56 (.02)	.52 (.02)	Interaction: P = .282	.03

Lateral gastrocnemius and vastus lateralis oblique (LGVLO) muscle co-contraction

The ANOVA analysis showed no significant effect of trunk lean on the co-contraction of LGVLO (P = .12) and no interaction observed between groups (P = .49) (Table 6-13).

Table 6-13 Summary data for LGVLO co-contraction (MVIC: proportion of the MVIC) (average across 10–20% of stance phase) for the different trunk conditions.

	Normal walking	**Trunk lean (+5°)**	**Trunk lean (-5°)**	**Significant test**	**Effect size**
OA group	.73 (.18)	.72 (.19)	.69 (.15)	Effect of trunk inclination P = .12	.05
Older group	.63 (.18)	.61 (.18)	.61 (.19)	Interaction: P = .49	.02

Semitendinosus and vastus medialis oblique (STVMO) muscle co-contraction

Table 6-14 shows STVMO co-contraction data. The ANOVA showed that there was a significant effect of trunk lean (P = .00) but no interaction effect between groups (P = .568) (Table 6-14). In addition, post hoc tests revealed that there was a significant increase between NW and +5° in both groups.

*Table 6-14 Summary data for STVMO co-contraction (MVIC: proportion of the MVIC) (average across 10–20% of stance phase) for the different trunk conditions . * indicated a significant difference between this condition and normal walking as identified by the post hoc test (p<0.05).*

	Normal walking	**Trunk lean (+5°)**	**Trunk lean (-5°)**	**Significant test**	**Effect size**
OA group	.68 (.16)	.76 (.20)*	.67 (.20)	Effect of trunk inclination P = .00	.41
Older group	.54 (.12)	.64 (.18)*	.52 (.12)	Interaction: P = .568	.02

Biceps femoris and vastus lateralis oblique (BFVLO) muscle co-contraction

BFVLO muscle co-contraction data are presented in Table 6-15. The ANOVA analysis showed a significant effect of trunk lean (P = .00) but no interaction effect was observed (P = .730) (Table 6-15). Bonferroni post hoc tests showed that there were no significant differences between the groups trunk lean conditions.

Table 6-15 Summary data for MG muscle activation (MVIC: proportion of the MVIC) (average across 10–20% of stance phase) for the different trunk conditions.

	Normal walking	**Trunk lean (+5°)**	**Trunk lean (-5°)**	**Significant test**	**Effect size**
OA group	.72 (.04)	.76 (.04)	.67 (.04)	Effect of trunk inclination P = .00	.16
Older group	.59 (.04)	.64 (.04)	.52 (.02)	Interaction: P = .730	.00

6.4.5 Sagittal angles and spatiotemporal parameters

ANOVA testing showed no significant differences in the knee and ankle angles (averaged between 15-25%) between the trunk lean conditions and no interactions (P > .05). However, a significant increase in the hip flexion angle between the normal walking and +5 trunk lean condition was observed in both groups. Spatiotemporal (speed and step length) parameters did not differ between the different trunk lean conditions and no interaction effects were observed.

6.5 Discussion

6.5.1 Overview

This study aimed to investigate the impact of increasing/decreasing trunk lean (+5° and -5°) on lower limb kinematics, kinetics, muscle activation patterns and muscle co-contraction in knee OA patients and older healthy people during walking. The most important findings were marked increases/decreases in the hip moment, increases in hamstring muscle activity and subtle change in co-contraction. Importantly, the data showed that for all parameters studied,

there was no interaction between the healthy people and those with knee OA, showing that changing trunk angle led to similar effects in both groups.

This study was designed to build on the findings of the previous chapter, which demonstrated that small changes in trunk posture lead to an alteration in lower limb biomechanics in young healthy people. However it was not clear from the data in the previous chapter whether these biomechanical changes would be observed in older people and people with knee OA. There has been no previous studies which have attempted to understand the effect of different trunk leans in knee OA and older healthy people during walking. Therefore, rather than compare with previous research, this discussion section is focused on contrasting the results of the previous chapter with those reported in the previous section. This data is presented in tabulated form and then discussed for most important biomechanical parameters.

6.5.2 Kinematics and spatiotemporal parameters

Table 6-16 presents spatiotemporal parameters for people knee OA, the older and young healthy subjects. The findings for the older healthy group and the group with knee OA patients appear to be consistent with those of the young group (Chapter 5), which also showed no difference in the spatiotemporal parameters as walk with different trunk lean (Table 6-16). Taken together, these data show that the instruction provided in this study did not lead to changes in speed or step length. This provides confidence that the observed changes in biomechanics were not the result of changes in spatiotemporal parameters.

Table 6-16 The differences in the spatiotemporal parameters with different trunk leans between knee OA, older healthy and young healthy groups. Speed: (ms) Step length: (m).

Variable	Group	NW	NW +5°	NW -5°
Speed	OA	1.18 (.11)	1.22 (.12)	1.21 (.12)
	Older	1.20 (.16)	1.21 (.13)	1.18 (.14)
	Young	1.21 (.09)	1.21 (.09)	1.18 (.10)
Step length	OA	1.28 (.08)	1.29 (.12)	1.30 (.09)
	Older	1.29 (.12)	1.29 (.09)	1.29 (.10)
	Young	1.32 (.10)	1.33 (.13)	1.33 (.10)

Table 6-17 present kinematic data for the three groups and shows the percentage increase in each parameter between NW and both the +5° and -5° conditions. In general, the results from people with knee OA and matched healthy participants seems to be consistent with the data from the young healthy group, with the primary change being an increase in hip flexion angle when trunk inclination was increased by 5°. Taken together, these data demonstrated that the influence on lower limb kinematics of changing the trunk lean was consistent between the young healthy group (Chapter 5) and older healthy and knee OA participants.

Table 6-17 The differences in kinematics results (°) in knee OA , older healthy and young healthy groups with different trunk lean over the average 15–25% of gait cycle. indicated a significant difference between this condition and normal walking.*

Variable	Group	NW +5°	NW -5°
Hip angle (°)	OA	↑ 50% *	↓ 22%
	Older	↑ 46% *	↓ 14%
	Young	↑ 80% *	↓ 22%
Knee angle (°)	OA	↑ 13%	↓ 2%
	Older	↑ 11%	↓ 5%
	Young	↑ 14%*	↓ 3%
Ankle Angle (°)	OA	↑ 4%	↑ 4%
	Older	↑ 5%	↓ 3%
	Young	↑ 2%	↑ 5%

6.5.3 Kinetics

Table 6-18 contrasts changes in lower limb moments when participants walked with the two different trunk angles. These results indicated the typical changes in lower limb joint moments, associated with changing trunk inclination, are consistent between the three groups. Specifically, there was an increase/decrease in hip moment when trunk inclination was increased/decreased and an increase/decrease in ankle moment when trunk inclination was increased/decreased. Although there was a non-significant effect of increasing trunk inclination on ankle moment, in people with OA, the magnitude of the effect was similar to the older adults. Therefore, in general, these results demonstrate that the influence of changing trunk lean on hip, knee and ankle moments is relatively consistent between the young healthy, older healthy and knee OA groups.

*Table 6-18 The different results of the hip, knee and ankle kinetics (Nm/kg) during walking with different trunk angles in people with knee OA, older healthy and young healthy groups over the 15–25% period of the stance phase. * indicated a significant difference between this condition and normal walking.*

Variable	Group	NW +5°	NW -5°
Hip moment (nm/kg)	OA	↑ 106% *	↓ 47% *
	Older	↑ 93% *	↓ 57% *
	Young	↑ 47% *	↓ 52% *
Knee moment (nm/kg)	OA	↓ 6%	↑ 11%
	Older	↓ 9%	↓ 2%
	Young	↓ 10%	↑ 5%
Ankle moment (nm/kg)	OA	↑ 67%	↓ 67% *
	Older	↑ 70% *	↓ 70%*
	Young	↑ 150% *	↓ 150% *

6.5.4 Muscle activation

The results showed no significant interactions (group by trunk inclination) in the quadriceps, hamstrings and gastrocnemius muscle activations between the older healthy participants and patients with knee OA. This suggests that muscle changes associated with changes in trunk flexion are not dependent of subject characteristics. Table 6-19 compare the magnitude of change in EMG with different trunk inclinations (+5°, -5°), across the three groups. The consistency of the increase in MG is similar across the three groups, however, LG muscle activity were not significant increased in the older healthy participants between the NW and the +5° trunk lean. Nevertheless, all three groups showed small (non-significant) changes in quadriceps activity.

The data from the older healthy people and patients with knee OA for the hamstring muscle activity appears to be consistent with that of the young group when trunk inclination was increased (Table 6-19). However, interestingly, when trunk inclination was decreased by 5° hamstring muscle activity decreased by approximately 30% in the young group but there were no significant changes in older or OA group. A possible reason for this could be the difference in kinematics of hip joint and which captures how different groups respond to the instruction to walk with a backward lean. Figure 6-21 shows a more distinct change in hip joint kinematics with increased/ decreased trunk lean in young people. However, this change was less pronounced in people with knee OA and the older healthy people. This suggests that these participants responded to the instruction to walk with a backward lean with more adaptation in the spinal rather than at the hip joint.

In summary, the data on muscle activation suggest that the influence of instructing participants to walk with increased forward lean is consistent (across the three groups) for the quadriceps, hamstring and gastrocnemius muscles. However, when instructed to walk with a backwards lean, although the younger group was able to decrease hamstring activity, the older and OA groups demonstrated only small non-significant reductions in hamstring activity. We suggest this could be related to subtle differences in the kinematic pattern.

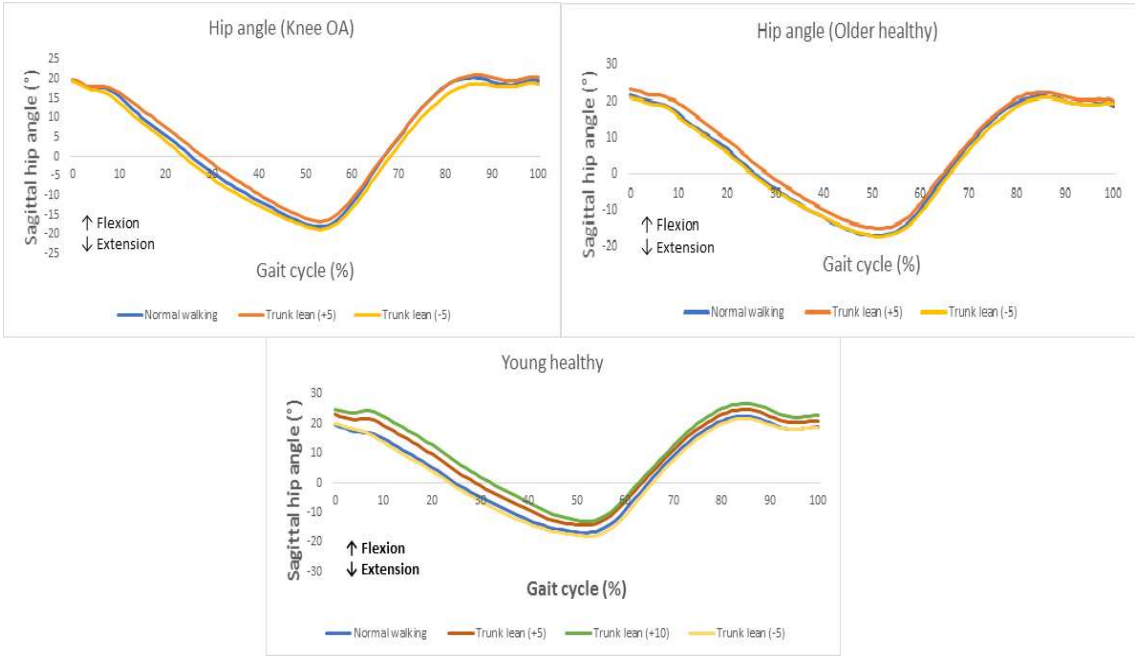


Figure 6-21 Hip angles during walking with different trunk lean in people with knee OA, the older healthy people and young healthy people.

*Table 6-19 The differences in EMG (MVIC: proportion of the MVIC) for the quadriceps, hamstrings and gastrocnemius during walking with different trunk leans in people with knee OA, older healthy and young healthy groups over the 10–20% period of the stance phase. * indicated a significant difference between this condition and normal walking.*

Variable	Group	NW +5°	NW -5°
MG	OA	↑ 25% *	0%
	Older	↑ 23% *	↓ 33% *
	Young	↑ 60% *	↓ 30% *
LG	OA	↑ 40% *	↓ 10%
	Older	↑ 38%	↓ 12%
	Young	↑ 28% *	↓ 14%
VMO	OA	↑ 3%	↑ 6%
	Older	↓ 4%	↓ 5%
	Young	↑ 3%	↓ 7%
VLO	OA	↑ 5%	↑ 15%
	Older	↓ 3%	↑ 3%
	Young	↓ 3%	↑ 3%
ST	OA	↑ 70% *	↓ 19%
	Older	↑ 150% *	↓ 13%
	Young	↑ 100 % *	↓ 33 % *
BF	OA	↑ 28% *	↓ 20%
	Older	↑ 50% *	↓ 17%
	Young	↑ 83% *	↓ 29% *

6.5.5 Muscle co-contraction

The data showed that no significant interactions (group x trunk inclination) in any of the muscle co-contraction measures, showing the effect of changing forward lean is consistent between healthy older and people with knee OA. Table 6-20 compares the change in the co-contraction measures (each corresponding to a different muscle pair) different trunk between normal walking and the two inclinations (+5°, -5°). The data demonstrates that only ST-VMO co-contraction increases with increased forward lean but that this effect is relatively consistent across the three groups. Given the minimal changes in VMO, reported above, this change in co-contraction is the result of increased medial hamstring activity.

*Table 6-20 The differences in muscles co-contraction (MVIC: proportion of the MVIC) during walking with different trunk leans in all groups over the 10–20% period of the stance phase. * indicated a significant difference between this condition and normal walking.*

Variable	Group	NW +5°	NW -5°
MGVMO	OA	↑ 5%	↑ 2%
	Older	↑ 4%	↓ 4%
	Young	↑ 16%	↓ 16%
LGVLO	OA	↓ 1%	↓ 5%
	Older	↓ 3%	↓ 3%
	Young	0%	0%
STVMO	OA	↑ 12% *	↓ 2%
	Older	↑ 18%*	↓ 4%
	Young	↑ 11%*	0%
BFVLO	OA	↑ 6%	↓ 7%
	Older	↑ 8%	↓ 11%
	Young	↑ 28%	↓14%

6.5.6 Implications for progression and clinical management of knee OA

The data presented in this chapter show that when people (with or without knee OA) are instructed to walk with increased the trunk lean, there is a distinct change in muscle activation patterns. Importantly, medial hamstring-quadriceps co-contraction was the parameter which appeared to be most strongly affected by increasing trunk inclination. Interestingly, data from longitudinal study showed that co-contraction between these two muscles was correlated positively with more rapid disease progression of medial knee OA (Hodges et al., 2016b). In addition, at 8 years follow up study by Hubley-Kozey, Hatfield, et al. (2013) showed that patients with knee OA had higher in quadriceps and hamstring co-contraction and this may result in choosing to go for a knee joint replacement earlier (Hubley-Kozey, Hatfield, et al., 2013). These two studies demonstrated that increased muscle co-contraction may accelerate disease progression. Given that medial hamstring-quadriceps co-contraction was the parameter which appeared to be most strongly affected by increasing trunk inclination in this study, it is possible that altered trunk inclination may play a role in the progression of knee OA.

Previous modelling studies show that altered muscle patterns may increase the load on the knee joint (Brandon et al., 2014; Sritharan et al., 2016). Sritharan et al. (2016) studied the influence of muscle activation on medial joint loading, looking at both individuals with knee OA and healthy controls. They observed that patients with knee OA demonstrate higher hamstring but lower quadriceps muscle activity. However, they showed higher hamstring-quadriceps co-contraction over early stance in the OA group which were associated with increased joint compressive forces. Give the similar findings, in this current study, of increased hamstring activity but minimal changes in quadriceps activity, it is possible that altered trunk inclination may play a role in increasing joint loading. As well as accelerating disease progression, this could exacerbate pain and impair function in people with knee OA.

Walking with increased trunk flexion would appear to be associated with increased muscular co-contraction which has been linked to increased joint loading (Brandon et al., 2014; Sritharan et al., 2016) and an increased rate of disease progression (Hodges et al., 2016a; Hubley-Kozey, Hatfield, et al., 2013). Therefore, future clinical strategies to decrease muscular co-contraction, targeted at improving upper body posture during walking may lead to clinical

benefits for those with knee OA. The current study showed that, when walking with 5° less trunk lean, there was a small decrease in knee muscle co-contraction, however, the result did not reach significance. Therefore, there is a need to develop effective techniques for decreasing trunk flexion in patients with knee OA. These therapeutic interventions could focus on stretching exercises, muscle strengthening exercises and postural training programs. Specifically, hip flexor muscle stretching may be an important clinical interventions and is discussed further in next chapter.

6.5.7 Limitations

In this investigation there are several limitations. Firstly, the baseline, habitual trunk inclination trunk angle during normal walking differed between the participants. Nevertheless, a biofeedback approach was used to ensure a precise and consistent angles in the two conditions (normal waking +5° and -5°). In addition a LMM was performed to understand the effect of increasing trunk angle in each of the groups OA and which can deal with between subject differences in the independent variable. This LMM analysis was consistent with the ANOVA analysis and therefore provide confidence that the findings would have been unaffected by between-subject differences in baseline trunk angle.

A further limitation of the study was that was not possible to collect data at the +10° condition as older subjects and people with knee OA found this too difficult. To deal with this difference, data was presented first for the younger people (previous chapter) and here for the other two subject groups. A comparison was also provided in the discussion section which highlighted the similarities and key difference between the three groups. However, it is important to note that people with OA are unlikely to walk with an increase of 10° in trunk inclination, especially given that they walk with 3° more forward lean than healthy people. Therefore, the data presented in this chapter provide a clear understanding of the effect of changing trunk lean over a physiologically realistic range.

Another final limitation is that it was not possible to instruct participants to increase trunk lean by changing their hip joint position rather than by altering vertebral alignment. As described in the method chapter, all subjects were instructed to “move the hip backwards. (for more details, see method chapter 3.5.11). Though pilot work, this instruction was found

be the most effective to produce a motion at the hip. However, when people with knee OA and older healthy subjects were instructed and to walk with lower trunk inclination, the change in the hip flexion/extension angles was less pronounced than that in young healthy people which suggests that these subjects may have adapted in the spinal rather than at the hip joint. As explained earlier, this could explain the differences in hamstring muscle activity response when people with knee OA were instructed to walk with 5° less trunk lean. Further work should therefore focus on developing more complex biofeedback systems which could be used to specifically target the hip.

6.5.8 Conclusion

This study was designed to determine the effect of increasing/decreasing trunk lean (+5° and -5°) on lower limb kinematics, kinetics, muscle activation, and muscle co-contraction in patients with knee OA and matched healthy subjects. The results of this study showed that increased trunk inclination is accompanied by a corresponding increase in the hip extensor moment and ankle plantar flexor moment, an increase in hamstring and medial gastrocnemius activity and an increase in medial hamstring-quadricep co-contraction in both groups. In general, these changes were consistent with those of the young group. However, whereas in young healthy people, there was a decrease in hamstring activity when trunk lean was decreased, this effect was less pronounced in people with knee OA. The data in this chapter demonstrates strong evidence that the alterations in the lower limb moment and muscle activation observed in people with knee OA, might be the result of increased trunk lean. However, to further validate this idea, it is necessary to investigate the potential differences between two groups of healthy people who habitually walk with different trunk inclination angles and who do not suffer with pain. This is explored in the following chapter.

Chapter Seven

Study four

How do interindividual variations in habitual trunk inclination during walking affect joint moments and muscle activation in healthy people?

7.1 Introduction

Data from the previous chapter demonstrated that when individuals with knee OA walk with increased/decreased trunk lean, there are changes in lower limb biomechanics. Specifically, the hip flexor moment increases and along with hamstring muscle activity and muscular co-contraction. Importantly, these changes are very similar to the differences observed between healthy people and those with knee OA. However, a within-subject design does not definitely address the question of whether two groups of individuals, with differences in habitual trunk flexion, would walk with corresponding differences in lower limb biomechanics in the absence of knee pain. To address this question, it is necessary to investigate the biomechanical differences which are associated with natural variations in trunk inclination in healthy participants.

There are two studies in the literature that have investigated the effect of natural trunk inclination on lower limb moments during walking (Leteneur et al., 2009; Sato & Maitland, 2008). These studies demonstrate that the natural trunk lean in healthy subjects could affect hip, knee and ankle moments during walking. For example, Leteneur et al. (2009) showed a clear difference in hip moments between forward leaners compared to backward leaners during walking. Although this study showed that in forward leaners there was a small decrease in knee moment and a small increase in the ankle plantar flexor moment, there were no statistical differences between groups. However, to date, there have been no studies which have investigated muscle activation. Therefore, the aim of this study was to quantify differences in muscle activation and biomechanics between two groups of young healthy subjects who habitually walk with different trunk flexion angles.

7.2 Research questions

The main objective of this study was to understand the biomechanical effect of natural variation of trunk inclination on lower limb biomechanics. This objective was achieved through five separate research questions:

Q 1 What is the effect of natural variation in trunk inclination on hip, knee and ankle moments in young healthy people during walking?

Q 2 What is the effect of natural variation in trunk inclination on gastrocnemius, quadriceps and hamstring muscle activities in young healthy people during walking?

Q 3 What is the effect of natural variation in trunk inclination on gastrocnemius, quadriceps and hamstring muscles co-contraction in young healthy people during walking?

Q 4 What is the effect of natural variation in trunk inclination on hip, knee and ankle angles in young healthy people during walking?

Q 5 What is the effect of natural variation in trunk inclination on spatiotemporal parameters of gait in young healthy people during walking?

7.3 Method

A full description of the method for this study was provided in the method chapter. In this study only, normal walking data were used from the healthy participants. In the following paragraphs, the sample characteristics and primary outcome measures used to analyse the data and the appropriate statistical tests for this study are presented.

7.3.1 Sample and population

A total of 34 adult healthy participants across both genders were recruited in this study (Table 7-1). A modified median split approach was used (described below) which involved using data from n=29 subjects (with 5 excluded). These 29 subjects were split into two groups based on the median of the mean trunk inclination angle over the gait cycle: a group of forward leaners (FW) and backward leaners (BW). The FW groups had a mean trunk lean above the median, whereas the BW group had a mean trunk lean below the median (Table 7-2). Five subjects

were excluded as they were within the standard error of measurement for the trunk angle, which was approximately one degree (Appendix IX).

Table 7-1 Participants' characteristics for the young healthy people. Values are the mean ± Standard Deviation (SD).

Variables	Young healthy people
No. of participants	34
Age (Years)	28.1 (7)
Height (M)	1.7 (.1)
Mass (Kg)	65.3 (8.08)
Body mass index (kg/m2)	22.5 (4.5)
Gender (M/F)	22/12

Table 7-2 Participants' characteristics for the forward and backward leaners groups. Values are the mean ± Standard Deviation (SD).

	Forward group	Backward group
Number of subjects	15	14
Age (Years)	26.9 (6)	28.77 (7)
Height (M)	1.7 (.08)	1.7 (.05)
Mass (Kg)	62.3 (8.5)	68.38 (7.5)
Body mass index (kg/m2)	21.5 (3.9)	22.70 (4.6)
Trunk lean during walking (º)	4.6 (1.3)	.48 (1.8)
Limb (R or L)	(9 R leg and 6 L leg)	(10 R leg and 4 L leg).
Gender	10 M and 5 F	9 M and 5 F

7.3.2 Derivation of outcome measures

The aim of this study was to investigate the biomechanical differences between the FW and BW groups during walking. Two different periods of gait cycle were chosen to analyse the gait data: a specific period of stance phase (15-25%, or 10-20% to account for electromechanical delay) and a specific period of gait cycle which was centred on peak muscle activity or peak moment/angle. In the following paragraphs, the primary outcomes measures for each research questions are explained in detail:

Trunk angle

The trunk angle was defined and tracked following the protocol explained in section 3.6.4 (Armand et al., 2014). The ensemble average of trunk angle over stance phase was calculated during walking. Then, the trunk lean was averaged over the stance phase for all trials and for each of the participants. The scatter diagram below shows how the subjects were divided into two groups (FW and BW) and five subjects excluded, based on the SEM for the trunk angle (Figure 7-1). A comparison of the demographic characteristics between the two groups is provide in Table 7-2.

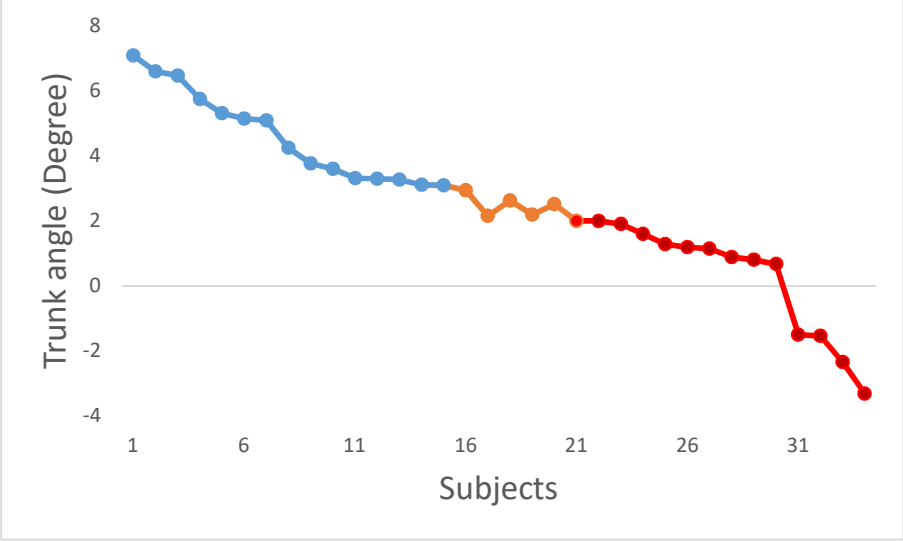


Figure 7-1 The scatter diagram shows the two groups and the excluded subjects. The red dotted line is the backward leaners, the blue dotted line is the forward leaners and the yellow colour is the middle values, which are excluded.

Sagittal lower limb moment

For each subject, the ensemble average curves for sagittal hip, knee and ankle moment were calculated during normal walking. Then, the mean over the specific window of the stance phase (15-25%) was calculated, which has been shown to correspond to the peak in the knee contact force (Brandon et al., 2014; Sritharan et al., 2016). In addition, the peak values over a specific window of stance phase were identified following the procedures in the study by Leteneur et al. (2009).

Muscle activity

As explained in the method section, a smoothed linear envelope signal, normalised by the MVIC signal, was created for each walking trial. For each subject, the ensemble average curves for two hamstrings, two quadriceps and two gastrocnemius muscles were calculated during normal walking. Following this, the means of these muscles' activity over the specific period of the stance phase (10-20%) were calculated. In addition, the mean across a muscle-specific the time window was calculated. These windows were chosen based on the peak activity for each muscle group.

Muscle co-contraction

In order to calculate knee muscle co-contraction during walking, the method that involves a separate summing of the medial and lateral muscles activation was used (for more details, see Section 2.5). Specifically, co-contraction was calculated as the sum of the quadriceps with the hamstring and the quadriceps with the gastrocnemius (medial and lateral) over the period of 10-20% of the stance phase.

Sagittal lower limb angles

For each subject, the ensemble average curves for sagittal hip, knee and ankle angles were calculated during normal walking. Again, two windows were studied. The first involved calculating the mean angle over 15-25% of stance. For the second window, the peak values over the whole of stance phase were chosen.

Spatiotemporal parameters

Speed and step length were calculated to ensure that there were no differences between the participants in both groups.

7.3.3 Statistical methods

To answer the above questions, the statistical package for social studies (SPSS) version 24 for Windows was used. In this study, an independent-sample t-test was performed to determine the differences between the two independent groups (FW and BW) for each of the dependent variables. In order to use the parametric test, the normally distribution, assumption of sphericity and data outlier were tested. Normal distribution was tested by using the Shapiro-Wilk's test (P>0.05) for each dependent variable. In addition, the homogeneity of variances, as assessed by Levene's test for equality of variances, should be not significant (P>0.05) for each dependent variable. Furthermore, for each tested variable, there should be no outliers in the data, as assessed by inspection of a whiskers boxplot. Therefore, if the results met the parametric assumption, then parametric data analysis was possible. However, if the result violated the parametric assumption, the non-parametric independent t-test (Mann-Whitney U) was performed. In addition, effect sizes value were calculated by using Cohen's d method, which defines 0.2, 0.5 and 0.8 as small, medium and large respectively (Cohen, 1992).

7.4 Results

7.4.1 Trunk angle

Figure 7-2 below illustrates the differences in trunk angle between the FW and BW leaners during normal walking over the stance phase. The solid line represents the mean across the 15 FW subjects and the dotted line represents the mean across the 14 BW subjects. It can be seen that the difference in trunk angle between the FW and BW groups is approximately 4^{o} across the stance phase (Figure 7-3). Specifically, the average natural trunk inclination during walking was 4.6^{o} (SD 1.3^{o}) for the FW group, while it was $.48^{o}$ (SD 1.8^{o}) for the BW group. This difference was statistically significant ($P < .005$).

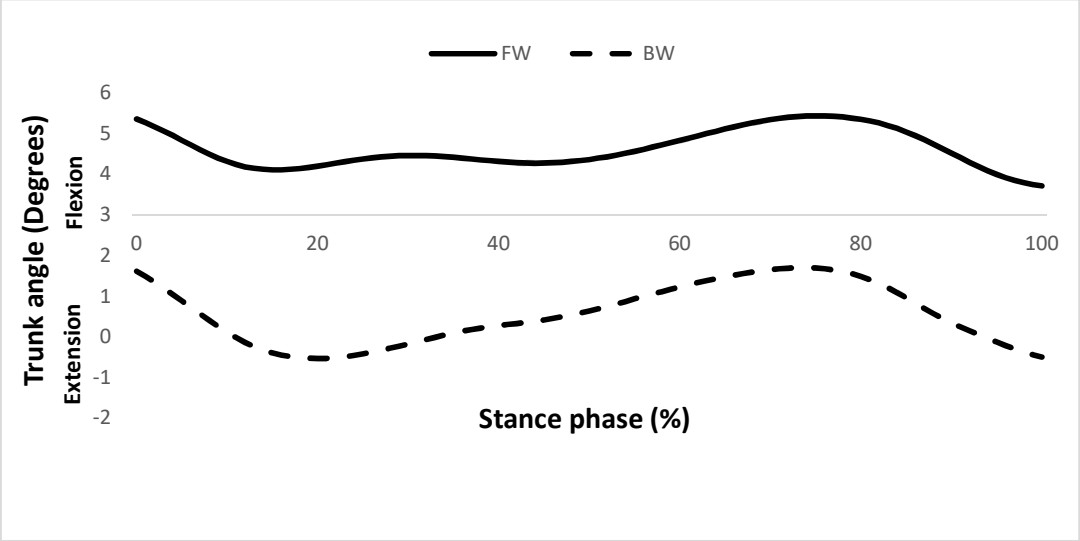


Figure 7-2 The ensemble average of the forward and backward leaners of the sagittal trunk angle while normally walking over the stance phase.

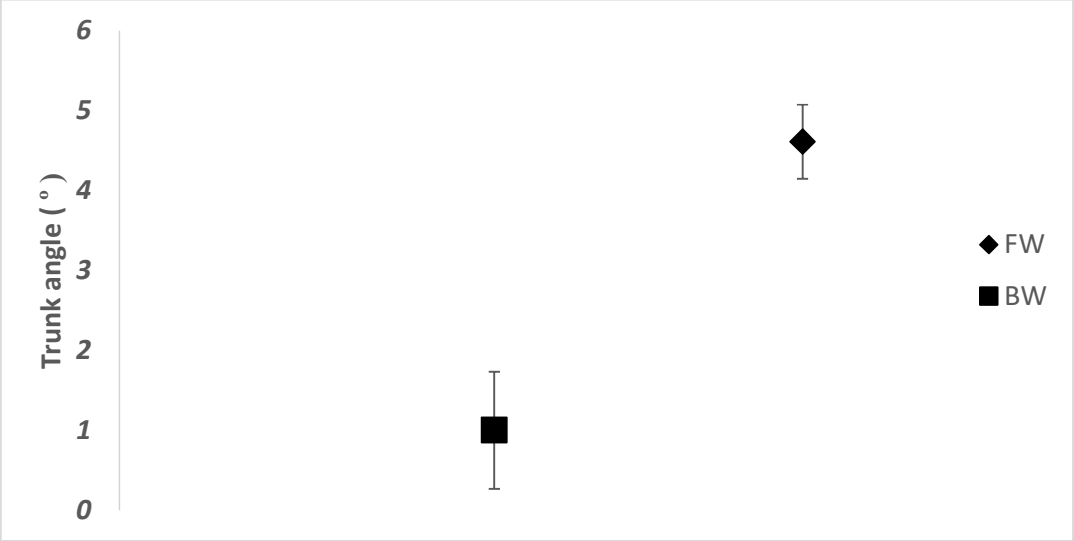


Figure 7-3 The mean and slandered deviation of the trunk angle among FW, with an average of 4.6° (SD 1.3°) and BW groups .48° (SD 1.8°).

7.4.2 Sagittal joint moments

Hip moment

Figure 7-4 shows the ensemble average (across the two separate groups, FW and BW) of the sagittal hip moment during walking over the stance phase. The data showed that, in the FW group, there was a clear rise in the magnitude of the hip extensor moment in the early stance

phase (Figure 7-4). The result showed that there were no statistically significant differences in the hip moment during walking across the focused window (15-25%). However, result for the peak hip moment (which occurred between 0-50% of stance phase), was significantly larger in the FW group compared to the BW group (Table 7-3).

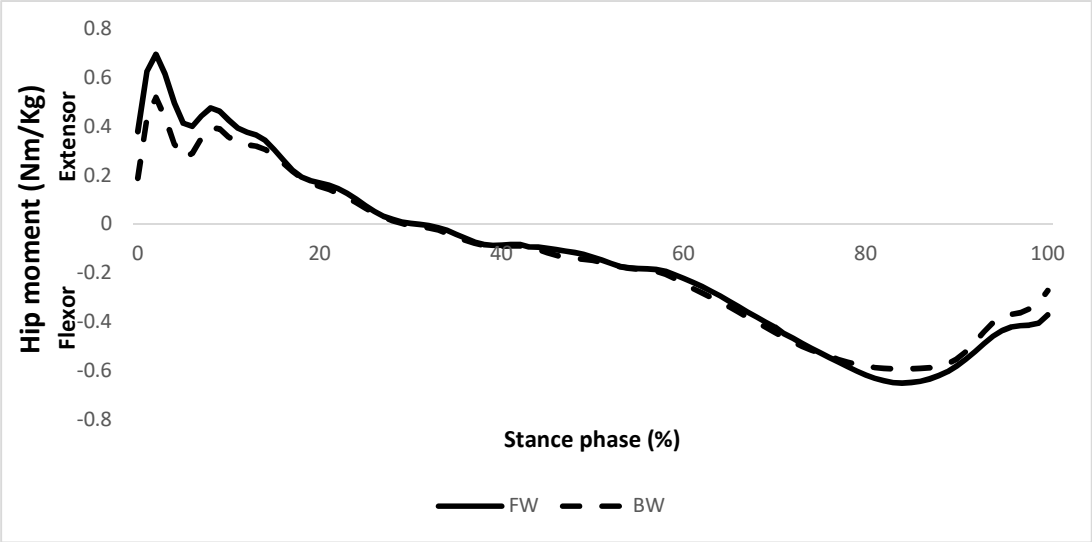


Figure 7-4 The ensemble average of the sagittal hip moment for normal walking in the FW and BW groups over the stance phase.

Table 7-3 The mean, standard deviation and P value of the hip moment (Nm/kg) in FW and BW subjects during normal walking in the average (15-25%) and peak periods (0-50%) of the stance phase.

Sagittal hip moment	FW group	BW group	P value	Effect size
Average (15-25%)	.17 (.24)	.16 (.16)	.87	.5
Peak (0-50%)	.71 (.21)	.56 (.10)	.02 *	.9

Knee moment

Figure 7-5 shows the ensemble average profile of the sagittal knee moment for the two groups of participants across the stance phase. The plot shows that the knee extensor moment was slightly decreased in the FW group compared to the BW group, however, there

were no significant differences in the average over (15-25%) or the peak knee moment between the two groups (Table 7-4).

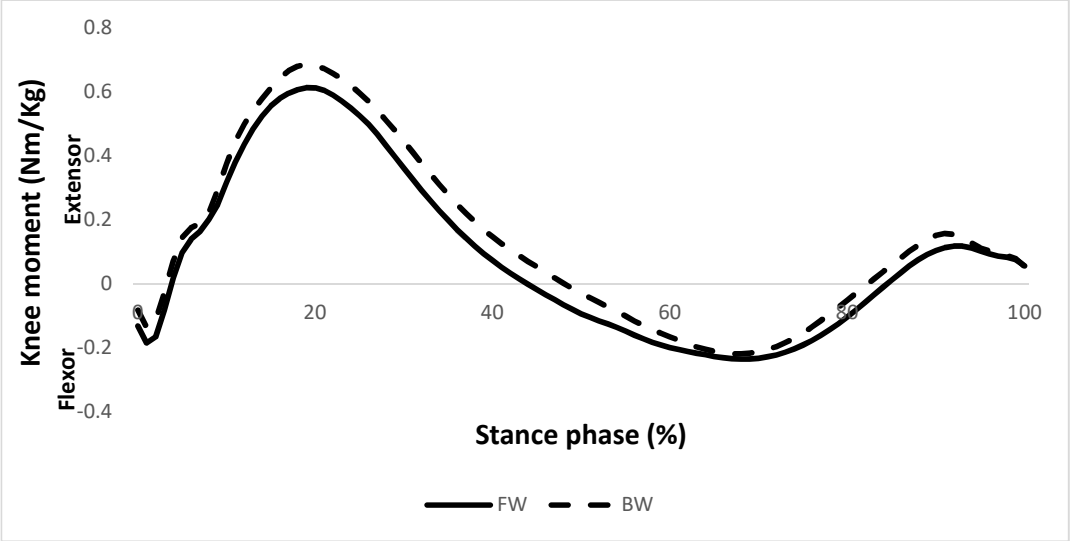


Figure 7-5 The ensemble average of the sagittal knee moment in the FW and BW group during normal walking over the stance phase.

Table 7-4 The mean, standard deviation and P value of the knee moment (Nm/kg) in FW and BW subjects during normal walking in the average period (15-25%) and peak period (0-50%) of the stance phase.

Sagittal knee moment	FW group	BW group	P value	Effect size
Average (15-25%)	.62 (.26)	64 (.21)	.77	.2
Peak (0-50%)	.67 (.25)	.68 (.23)	.91	.12

Ankle moment

The plot in Figure 7-6, below, illustrates the ensemble average of the sagittal ankle moment for the two groups during the stance phase. It can be seen from Figure 7-6 below that there was a slight increase in plantarflexor moment at the mid-stance phase in the FW leaners. However, there were no significant differences (Table 7-5) between both groups in either mean of peak moment.

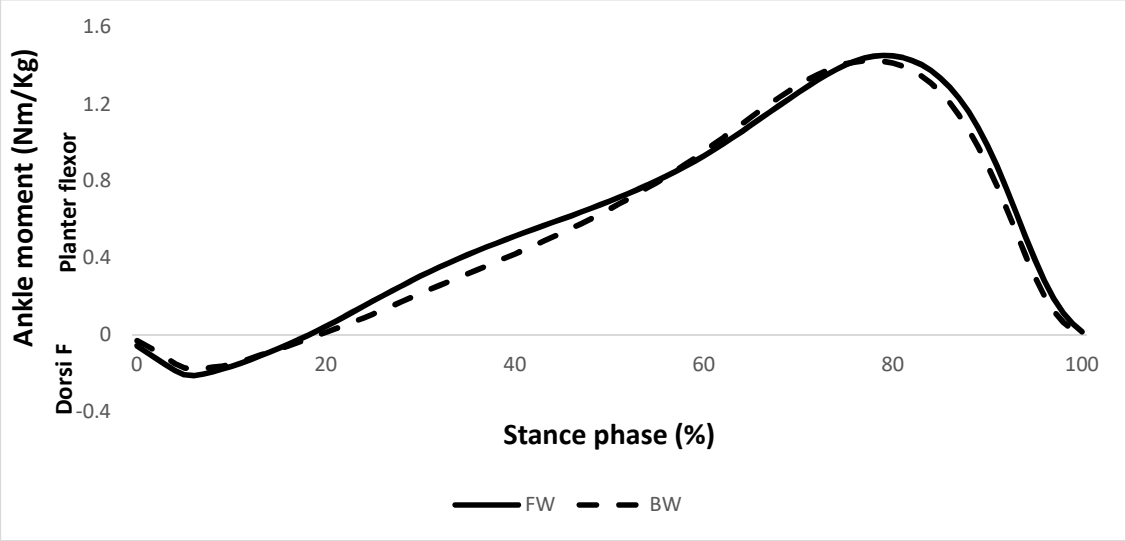


Figure 7-6 The ensemble average of the ankle moment for the FW and BW groups during walking.

Table 7-5 The mean, standard deviation and P value of the ankle moment (Nm /kg) in FW and BW subjects during normal walking in the period of 15-25% and peak period (40-100%) of the stance phase.

Sagittal ankle moment	FW group	BW group	P value	Effect size
Average (15-25%)	.04 (.06)	.01 (.08)	.25	.4
Peak (40-100%)	1.47 (.15)	1.43 (.14)	.52	.3

7.4.3 Muscle activation

Medial gastrocnemius (MG)

The plots below show the ensemble average of the normalised MG activity for both the FW and BW groups across the stance phase (Figure 7-7). This plot illustrates that MG muscle activity was higher in the FW group for almost all of the stance phase. Although there was no significant differences in the early stance period (10-20%), a non-parametric test showed that there was a significant difference in mean MG muscle activity over the window 55-85% of

stance phase (P = .00 ,Table 7-6), with the FW group having ~30% higher muscle activity than the BW group.

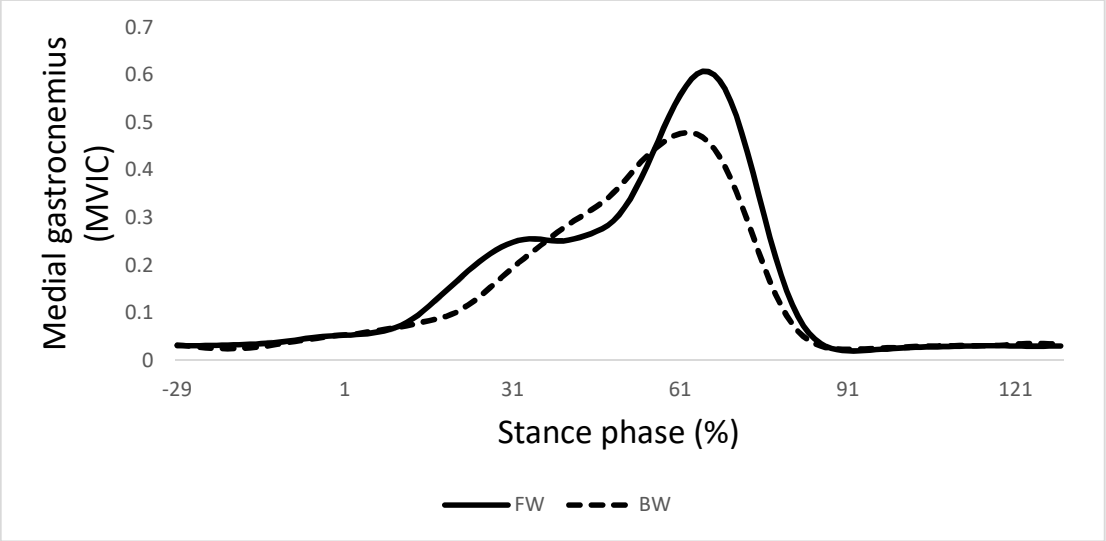


Figure 7-7 The ensemble average of the MG muscle activity in the FW and BW groups during walking.

Table 7-6 The mean, standard deviation and P value of the MG muscle activity (MVIC: proportion of the MVIC) in FW and BW subjects during normal walking in the periods of 10-20% and 55-85% of the stance phase.

MG	FW group	BW group	P value	Effect size
Average (10-20%)	.10 (.04)	.08 (.05)	.28	.4
Average (55-85%)	.39 (.13)	.31 (.09)	.012*	.7

Lateral gastrocnemius (LG)

Ensemble average profiles for the LG muscle activity for both groups are shown in Figure 7-8. These profiles were similar to those of the MG muscle activity (Figure 7-7). Again, there was increased activation in the FW group, but the difference was more pronounced during the period of 50-90% and there appeared to be minimal differences in the early stance. The analysis showed that there was no significant difference between the groups across the 10-20% stance. However, there was a significant difference across the period 55-85%, in which the FW group had muscle activation of ~50% more than the BW group (Table 7-7).

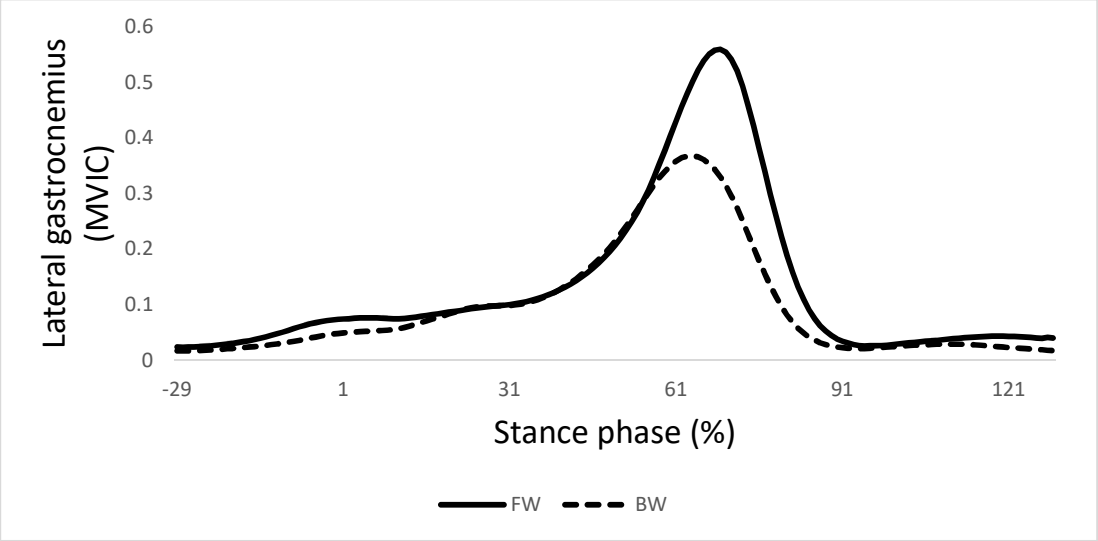


Figure 7-8 The ensemble average curves of LG activity during walking in the stance phase for the FW and BW groups.

Table 7-7 The mean, standard deviation and P value of the LG muscle activity (MVIC: proportion of the MVIC) in FW and BW subjects during normal walking in the period of 10-20% and 50-80% of the stance phase.

LG	FW group	BW group	P value	Effect size
Average (10-20%)	.07 (.04)	.06 (.03)	.46	.3
Average (55-85%)	.37 (.18)	.25 (.09)	.03*	.8

Vastus oblique medialis (VMO)

The ensemble average curves in Figure 7-9 illustrate the characteristic pattern of the VMO muscle activity in both groups, and show that the only difference was around the early stance phase and with minimum differences across the rest of the stance phase. There were no significant differences in the VMO muscle across the period 10-20% or the period -20-10% of the stance phase (Table 7-8).

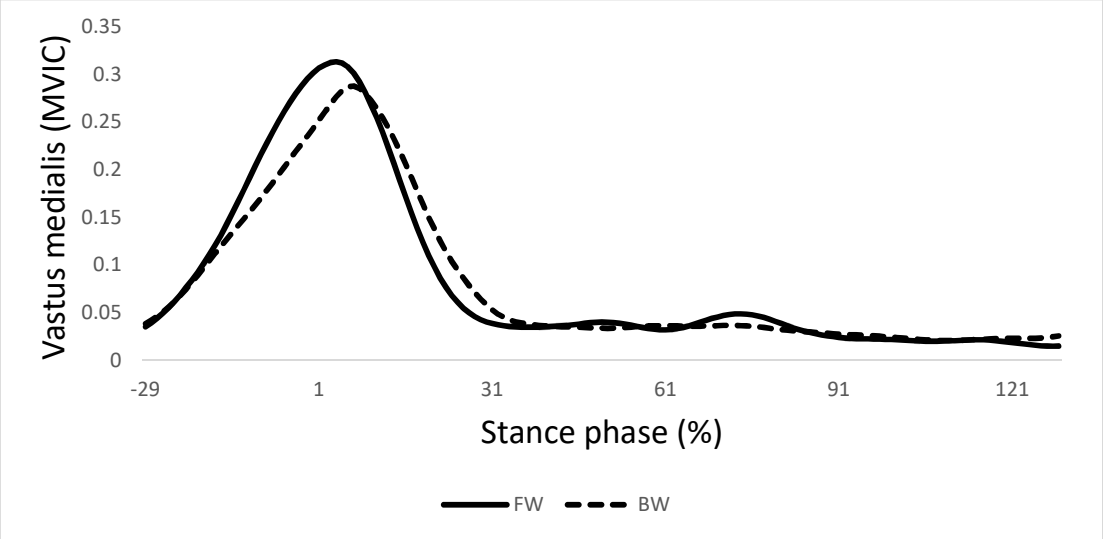


Figure 7-9 The ensemble mean average curves for the VMO activity during walking in the stance phase for the FW and BW groups.

Table 7-8 The mean, standard deviation and P value of the VMO muscle activity (MVIC: proportion of the MVIC) in FW and BW subjects during normal walking in the period of 10-20% and -20-10% of the stance phase.

VMO	FW group	BW group	P value	Effect size
Average (10-20%)	.19 (.14)	.21 (.12)	.73	.2
Average (-20-10%)	.24 (.15)	.20 (.12)	.44	.3

Vastus oblique lateralis (VLO)

The ensemble average for the VLO muscle activity for the two groups, FW and BW participants, is presented in Figure 7-10. These plots were similar to those of the VMO muscle activity, which showed that there was a tendency for more and longer activity in the FW group (Figure 7-9). However, there were no significant differences in the VLO muscle activity across either periods.

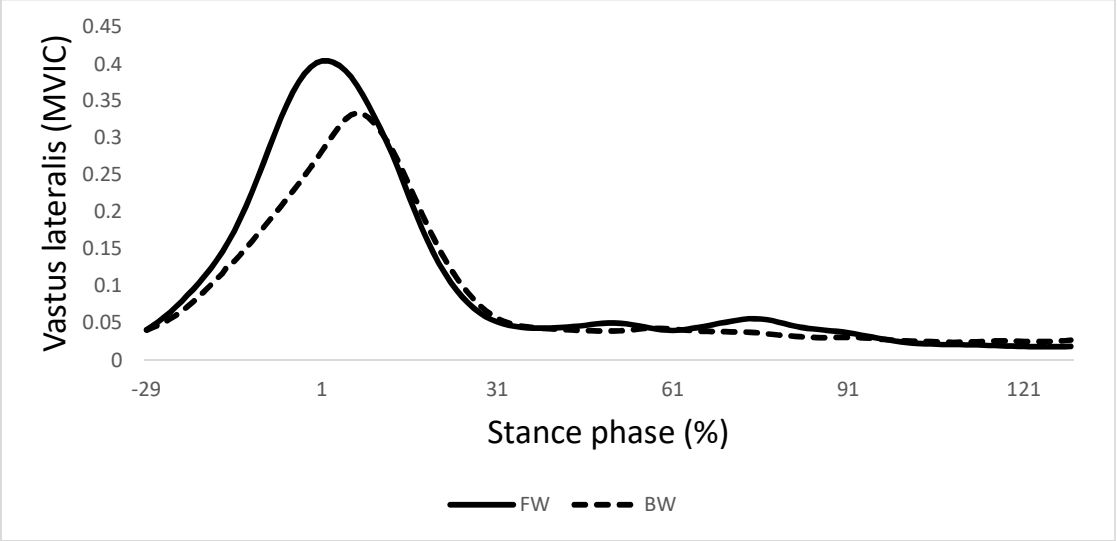


Figure 7-10 The ensemble mean average curves for the VLO activity during walking in the stance phase for FW and BW groups.

Table 7-9 The mean, standard deviation and P value of the VLO muscle activity (MVIC: proportion of the MVIC) in FW and BW subjects during normal walking in the period of 10-20% and -20-10% of the stance phase.

VLO	FW group	BW group	P value	Effect size
Average (10-20%)	.23 (.14)	.24 (.13)	.84	.07
Average (-20-10%)	.29 (.21)	.21 (.12)	.28	.6

Semitendinosus (ST)

Ensemble average profiles for the ST muscle activity for both groups are shown in Figure 7-11. These plots showed that there is a tendency for more and longer activity in the FW group in the early stance phase. However, the analysis showed that there was no significant difference between the groups in the ST muscle across the 10-20% or the -29-0% window (Table 7-10). However, for the window 10-20%, there was a large effect (0.8) which almost reached statistical significance (p=0.05).

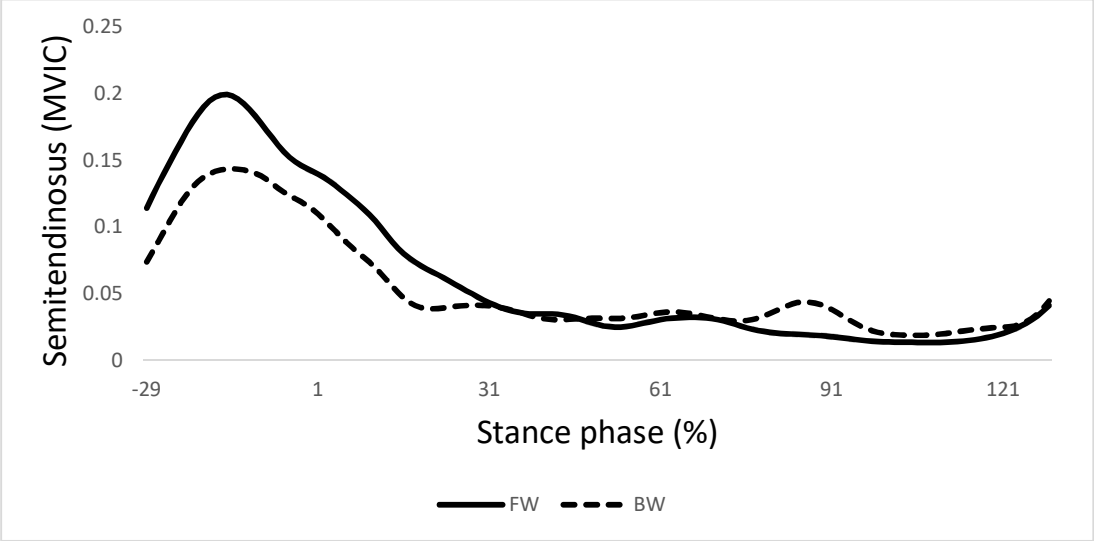


Figure 7-11 The ensemble mean average curves for the ST muscle activity during walking in the stance phase for FW and BW groups.

Table 7-10 The mean, standard deviation and P value of the ST muscle activity (MVIC: proportion of the MVIC) in FW and BW subjects during normal walking in the period of 10-20% and -29-0 of the stance phase.

ST	FW group	BW group	P value	Effect size
Average (10-20%)	.08 (.03)	.05 (.04)	.05	.8
Average (-29-0%)	.16 (.09)	.12 (.06)	.32	.5

Biceps femoris (BF)

The plots below show the ensemble average of the normalised BF muscle activity for both the FW and the BW group across the stance phase (Figure 7-12). These plots illustrate clearly that the BF muscle activity was higher in the FW group for almost all of the early and mid-stance phases. The results reveal that the BF muscle activity in the FW group was over twice as high as the BW group (P = 0.036) over the 10-20% stance. Furthermore, the analysis showed that there was a significant difference (P < .005) of approximately 70% in the BF muscle activation across the time window 29-0% in the FW group compared to the BW group (Table 7-11).

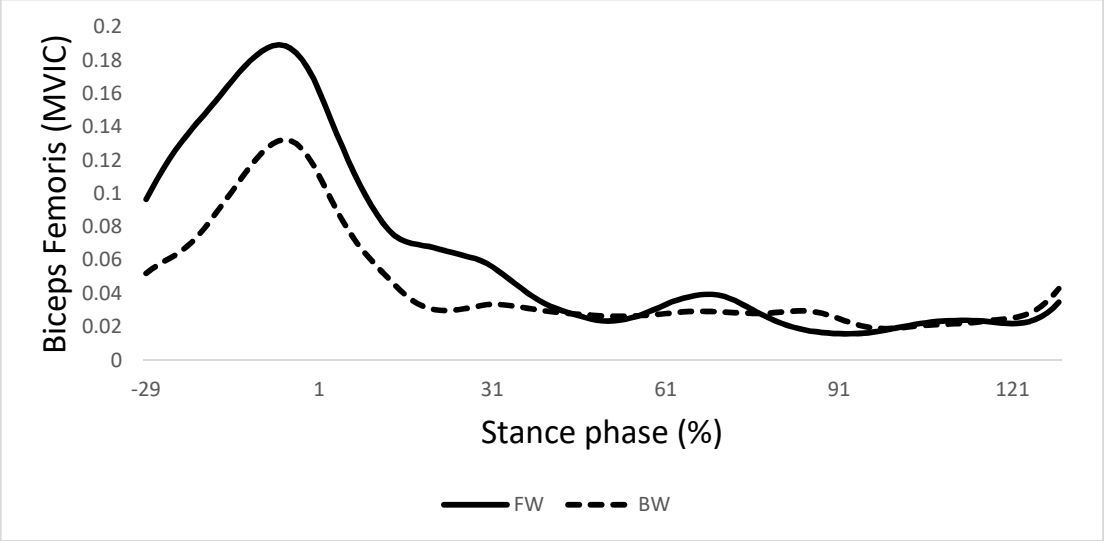


Figure 7-12 The ensemble mean average curves for the BF muscle activity during walking in the stance phase for FW and BW groups.

Table 7-11 The mean, standard deviation and P value of the BF muscle activity (MVIC: proportion of the MVIC) in FW and BW subjects during normal walking in the period of 10-20% and -29-0% of the stance phase.

BF	FW group	BW group	P value	Effect size
Average (10-20%)	.14 (.07)	.04 (.03)	.00 *	1.8
Average (-29-0%)	.16 (.05)	.09 (.01)	.00 *	1.9

7.4.4 Muscle co-contraction

The data comparing co-contraction for the four muscle pairs showed that no significant differences were observed between the two groups. Only minimum differences were noted between the groups; for example, the MGVMO co-contraction was 10% higher in the FW leaners than the BW leaners. In addition, the BFVLO co-contraction was 13% higher in the FW leaners than the BW leaners.

7.4.5 Sagittal angles

Hip angle

Figure 7-13 shows the ensemble average (across the two separate groups, FW and BW) of the sagittal hip angle during the full gait cycle. The data showed that there were more pronounced differences between the groups during the end of stance phase (40-60%). However, the result showed that there were no statistically significant differences in mean hip angle over the period 15-25% or the peak (40-60%) between the two groups (Table 7-12).

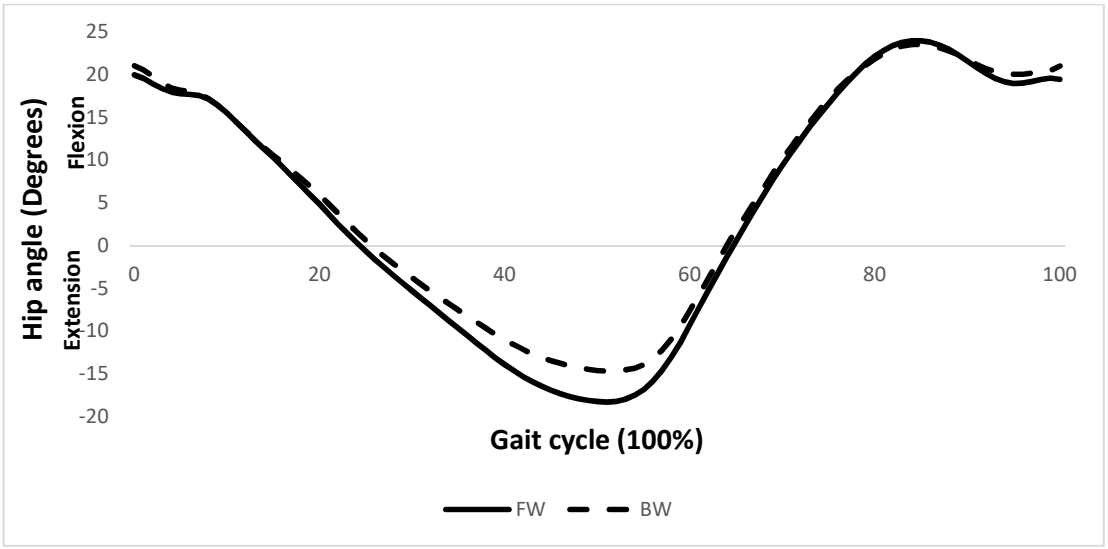


Figure 7-13 The ensemble average of the sagittal hip angle for normal walking in FW and BW groups over the gait cycle.

Table 7-12 The mean, standard deviation and P value of the hip angle (°) in FW and BW subjects during normal walking in the average (15-25%) and peak periods (40-60%) of the gait cycle.

Sagittal hip angle	FW group	BW group	P value	Effect size
Average (15-25%)	6.9 (4.2)	5.3 (3.3)	.26	.4
Peak (40-60%)	-9.3 (2.0)	-8.6 (4.7)	.60	.2

Knee angle

The ensemble averages of knee angle during walking are presented in Figure 7-14 and shows the differences across the full gait cycle. The overall knee angle remained relatively constant irrespective of trunk posture and there were only minimal differences between the two groups, with a reduced peak in the BW group at around 20% of the gait cycle. There was no significant difference in the average knee angle (15-25%) or the peak (0-30%) of the gait cycle (Table 7-13).

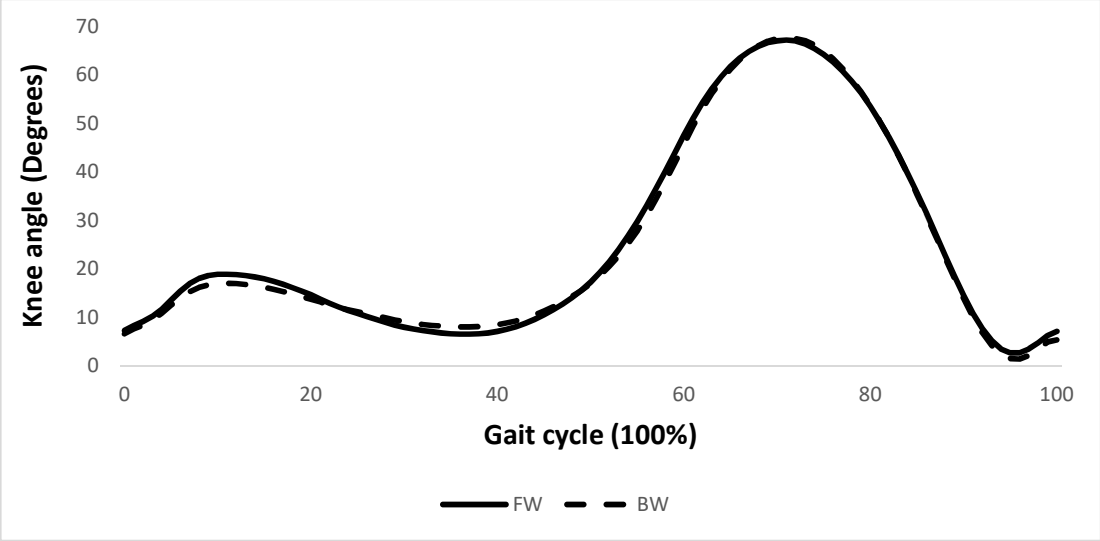


Figure 7-14 The ensemble average of the sagittal knee angle for normal walking in FW and BW groups over the gait cycle.

Table 7-13 The mean, standard deviation and P value of the knee angle (°) in FW and BW subjects during normal walking in the average (15-25%) and peak periods (0-30%) of the gait cycle.

Sagittal knee angle	FW group	BW group	P value	Effect size
Average (15-25%)	15.8 (5.2)	12.9 (4.2)	.11	.6
Peak (0-30%)	19.1 (5.2)	16.7 (4.2)	.20	.5

Ankle angle

The ensemble average ankle angle data are shown in Figure 7-15 and again show only minimal differences between the two groups. The analysis showed that there were no significant differences between the two groups in the mean across 15-25% or the peak over 50-80% of the gait cycle (P > 0.05, Table 7-14).

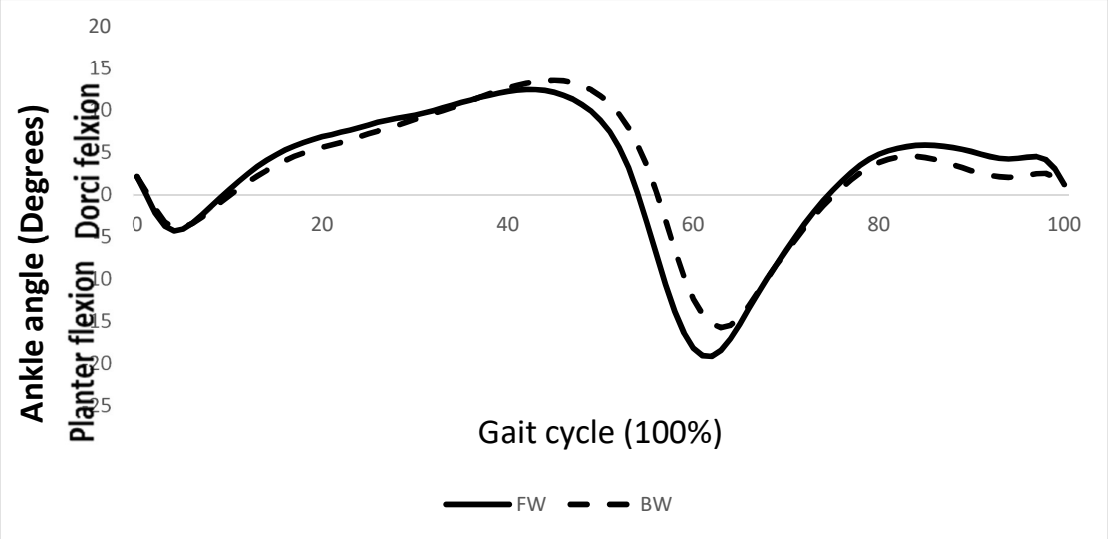


Figure 7-15 The ensemble average of the sagittal ankle angle for normal walking in FW and BW groups over the gait cycle.

Table 7-14 The mean, standard deviation and P value of the ankle angle (°) in FW and BW subjects during normal walking in the average (15-25%) and peak periods (50-80%) of the gait cycle.

Sagittal ankle angle	FW group	BW group	P value	Effect size
Average (15-25%)	5.9 (2.9)	5.5 (5.5)	.81	.1
Peak (50-80%)	-20.1 (10.5)	-16.7 (6.22)	.30	.4

7.4.6 Spatiotemporal (speed and step length)

There were no speed differences between the two groups (P= .39) (Table 7-15). In addition, the Mann Whitney test showed that there were no statistically significant changes in the step length (P = .12) (Table 7-15).

Table 7-15 The mean, standard deviation and P value of the spatiotemporal in FW and BW subjects during normal walking. Speed: (ms) step length: (m).

Spatiotemporal	FW group	BW group	P value	Effect size
Speed	1.32 (0.15)	1.27 (.14)	.39	.3
Step length	1.35 (.10)	1.29 (0.08)	.12	.7

7.5 Discussion

7.5.1 Overview of the results

The present study was designed to investigate the effect of inter-subject variability in natural trunk inclination (FW and BW) on lower limb kinematics, kinetics, muscle activation and muscle co-contraction during walking in healthy subjects. For all outcome variables, two distinct periods were analysed, a window of 15-25% stance phase (or 10-20% for muscle data) which corresponds to the first peak in compressive loading at the knee joint and a second window chosen to include peak moment/angle/muscle activity. The main finding was a significantly large peak of hip extensor moment in the FW group and an increased activity of the BF and ST muscles over the period 10-20%. Furthermore, both gastrocnemius muscle activities were higher in the FW group over a window in late stance phase (55-85%) by approximately 30% compared to the BW group. In the following section, the findings of this study are contrasted with previous researches and then the implications and limitation are presented.

7.5.2 Comparison with previous research

The findings of the current study are consistent with other studies, which found no differences in the spatiotemporal parameters between both the FW leaners and BW leaners during walking (Leteneur et al., 2009; Sato & Maitland, 2008). This lack of a difference suggests that the observed differences in the lower limb biomechanics are due to natural trunk inclination and are not the result of differences in spatiotemporal parameters. There has been no previous work that has studied the effect of natural trunk lean in lower limb kinematics. The present study found that there were only minimal non-significant differences in the hip, knee and ankle kinematics between the two groups. This indicates that between-

subject differences in sagittal trunk position is unlikely to be associated with alterations in lower limb kinematics.

In agreement with other studies, this current study found differences in lower limb kinetics between the FW and BW groups. Previous researchers found that a small decrease in the knee extensor moment in the FW in the early stance phase (Leteneur et al., 2009) and a small increase in the plantar flexor moment in the mid-stance phase during walking (Leteneur et al., 2009; Sato & Maitland, 2008), but the results did not show significant differences among the groups. These observations are consistent with our findings. However, although a previous study found no significant difference in peak hip extensor moment, they did observed that the duration of the hip moment was longer in the FW group compared to the BW group (Leteneur et al., 2009). In contrast, this current study observed a significant increase in peak hip extensor moment in the FW group. A possible reason for this discrepancy could be different characteristics of the participants or slight differences in the biomechanical calculations. To date there has been no previous study investigating the effect of natural trunk lean on muscle activation during walking.

7.5.3 Interpretation and implications of the findings

The findings presented in this chapter and the previous chapter shows that walking with an alteration in sagittal trunk position will affect the lower limb moments and muscle activation. The upper body segments accounts for approximately 65% of body mass (Dempster, 1955), thus a small change in upper body position could lead to an alteration in the position of COM and therefore the direction of the GRF vector. Alteration in the position and direction of the GRF vector, which would accompany a change in trunk lean, is the best way to understand why the changes in kinetics occur during walking. Figure 7-16 below illustrates the orientation of the GRF vector during the loading response phase of the gait cycle. During this period the GRF vector passes in front of the hip joint and behind the knee joint and ankle joint. With a change in upper body position, there will be a relative change of the GRF vector relative to the lower limb joints, and this will lead to a change in the joint moments.

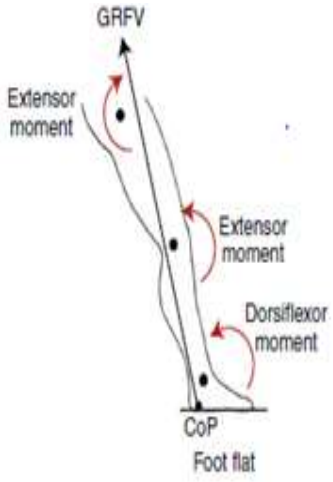


Figure 7-16 The ground reaction force vector relative to lower limb joint in the loading response phase.

Our results showed that the peak hip extensor moment significantly increased in the FW group. This could result from the anterior displacement of the GRF vector relative to the hip joint. In addition, results from the current study found small increases in plantar flexor moment during early stance phase (15-25%) in the FW leaners compared to the BW group. Although this result was non-significant it may have been a true effect of small magnitude which occurred due to the anterior shift of the COP relative to the ankle joint. This in turn would lead to an anterior movement of the GRF vector relative to the ankle joint. The result showed no differences in knee angle during walking between groups. This may explain the minimal change in knee moment which will be determined by the perpendicular difference from the GRF and the knee joint. Specifically, if the knee moves in the same way between the groups, then this distance is unlikely to change and therefore the moments are likely to be similar.

Muscles produce internal moments across joints during different periods of the gait cycle, which are balanced by external moments, resulting from the GRF. Therefore, changes in joint moments will be accompanied by corresponding changes in muscle activation. In the FW group the hip extensor moment was increased and there was a corresponding increase in hip extensor muscle (hamstring) activity. Similarly, the ankle plantar flexor moment was higher in the FW group and there was a corresponding increase in the activity of the gastrocnemius muscles (medial and lateral).

Data from chapter 4 showed that people with knee OA walk with higher hamstring muscle activity compared to matched healthy subjects. It is interesting to compare these data with the differences between the FW and BW groups observed in this study. This comparison is shown in Figure 7-17 below, in which the top plots show the BF and ST activation patterns for the OA and control data and the bottom plots shows the same data for the FW and the BW groups. Visual inspection of these data shows a lot of similarity in the BF patterns supporting the ideas that alterations in BF in people with knee OA are the result of an increased forward lean. For the ST muscle, although there is clearly increased muscle activity in both the FW and the OA groups, there is also a delay in the peak muscle activity in the OA group which is not apparent in the FW group. Taken together, these results suggest that some, but not all, of the differences in hamstring muscle patterns in people with knee OA could be the result of increased forward lean during walking.

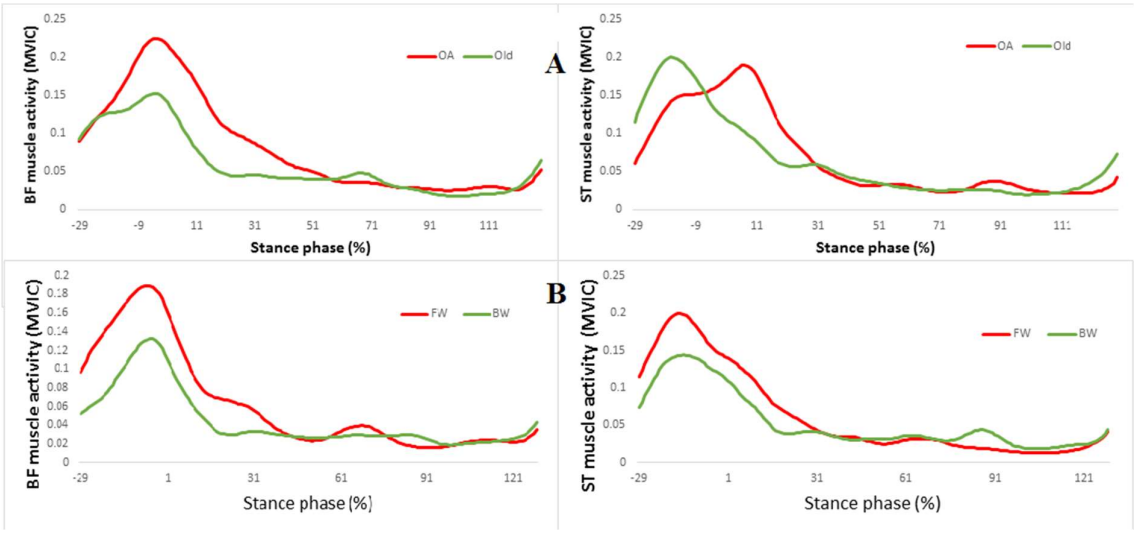


Figure 7-17 plots on the top (A) show the normalized hamstrings muscle activity (MVIC) in healthy (green) and knee OA (red). during walking over the stance phase. The plots below (B) show the differences in hamstrings muscle activity between the FW and BW groups while walking over the stance phase.

The differences in muscle co-contraction between people with knee OA and healthy subjects (chapter 4) are not consistent difference observed in this study between the FW and BW groups. Specifically, the data in this chapter did not show a significant difference in co-contraction between the FW and BW groups. However, the data in chapter 4 showed the people with knee OA walk with increase the hamstring-quadriceps muscle co-contraction. These contrasting findings can be explained from a consideration of the quadriceps activity in people with knee OA, which was observed to be higher approximately 30% compared to

healthy people but was very similar between the FW and BW groups. These results may indicate that, although change in hamstring activity are related to upper body position, the altered quadriceps muscle activity in knee OA could be the result of another disease-related mechanism, such as pain-related muscle guarding or to protect the knee from further degeneration (Astephen, Deluzio, Caldwell, Dunbar, et al., 2008; Childs et al., 2004; Hubley-Kozey et al., 2006; Rutherford et al., 2013).

In summary, the data presented in this chapter demonstrate clear similarities in muscle activation patterns between healthy people who walk with a 3-4 degree increase in trunk flexion and people with knee OA. As demonstrated earlier in this thesis, people with knee OA also walk with an increase in trunk flexion of a similar magnitude. Taken together these findings suggest that some of the previously observed alteration in lower limb muscle patterns, in people with knee OA, may be due to altered upper body position. This is important as it has been suggested by previous researchers, that the increased muscle activity in people with knee OA functions to either stabilities the joint in the presence of ligamentous instability (Childs et al., 2004) or is a direct response to pain (Hubley-Kozey et al., 2006). The data presented in this chapter suggests a completely different mechanisms and therefore could have wide reaching clinical implications for the management of knee OA. These are discussed in the next chapter.

7.5.4 Limitations

A number of important limitations need to be considered. Firstly, normalisation of EMG signals by MVIC can be problematic. However, a robust protocol with a 60 second rest between each contraction was applied and verbal encouragement provided. In addition, we used a standardised joint position to perform the MVIC on each muscle, as described in the method chapter (Rutherford, Hubley-Kozey, et al., 2011b). Furthermore, as all subjects were free of pain in this study, it is unlikely that there would have been any systematic differences in the MVICs between the FW and BW groups and therefore this aspect of the design should not have affected the findings and overall conclusion. In addition, it was difficult to measure the trunk inclination, as the spine is a multi-articulate structure and it is difficult to approximate as a single rigid segment. However, the optimal and minimal markers to define the movement of the thorax were used in this study (Armand et al., 2014). Another limitation

in this study was that the participants close to the median trunk angle were excluded in order to define the two groups. However, this decision was made based on the SEM of trunk angle measurement which approximately one degree, as showed in the test re-test reliability study (Appendix IX). Therefore, this aspect of the design was deemed necessary to ensure that participants were allocated to an appropriate group and would have ensured a robust analysis.

7.5.5 Conclusions

This study was designed to determine the biomechanical differences that are associated with natural variations in trunk inclination in healthy participants. The results showed that the peak hip extensor moment, both hamstring muscles and both gastrocnemius muscle activities were higher in the FW group compared to the BW group. In general, these findings provide support to the idea that alterations in hamstring activity, previously observed in people with knee OA may be the result of alterations in trunk inclination during walking. Therefore, it is necessary to investigate the biomechanical mechanisms which relate to change in sagittal trunk posture in both healthy individuals and those with knee OA. This is explored in the following chapter.

Chapter Eight

(Study Five)

What is the relationship between hip flexor muscle length and trunk inclination in healthy people and individuals with knee OA?

8.1 Introduction

Data from chapter 4 and previous research (Preece et al., 2018) demonstrated that patients with knee OA walk with approximately 3^{o} more in sagittal plane inclination of the trunk compared to matched healthy controls. In addition, higher hamstrings muscle activations with a corresponding increase in the medial and lateral hamstring-quadriceps co-contraction have been consistently observed in knee OA patients compared to matched healthy controls (Astephen, Deluzio, Caldwell, Dunbar, et al., 2008; Hortobagyi et al., 2005; Hubley-Kozey et al., 2009a). Interestingly, data presented in the previous chapters (5, 6 and 7) supports the idea that increases in trunk flexion may be part of a mechanisms which underlies the altered patterns of lower limb muscle activity observed in people with knee OA. This altered muscle activity is associated with increased joint loading (Brandon et al., 2014; Sritharan et al., 2016) and may accelerate the disease progression (Hodges et al., 2016a; Hubley-Kozey, Hatfield, et al., 2013). Therefore, there is a need to explore the possible biomechanical mechanisms which may underlie forward lean in people with knee OA.

As discussed in the literature review chapter, resting muscle length contributes to the alignment of the body segments. In the context of forward lean, it is important to understand potential imbalance of muscles surrounding the pelvis. Specifically, the balance of hip flexor and extensor muscles plays an important role in determining pelvic tilt in the sagittal plane in both standing and walking (Axelson & Hagbarth, 2001a). Muscle shortening of either the hip extensors/flexors may affect the sagittal plane orientation of pelvis and could lead to a corresponding effect on sagittal plane trunk orientation and/or lumbar lordosis (Shimada, 1996b).

Research has shown that muscle, maintained at a length that is either shorter or longer than its normal anatomical position, it can adapt to this position and become structurally longer or shorter (Proske et al., 1993; Williams, 1990; Williams & Goldspink, 1973, 1978). This is important in the context of people with knee OA who are known to spend long periods of time inactive and sitting (Lee et al., 2015; Sliepen et al., 2018; Wallis et al., 2013). In a sitting position, the hip flexor muscles are in a shortened position and if there is a structural adaptation of this muscle group, this could affect the sagittal orientation of the pelvis during standing and walking, with corresponding effects on trunk inclination.

To date, there has been minimal research investigating the link between hip flexor muscle length and gait biomechanics in patients with knee OA. Therefore, this chapter aimed to understand whether there was a difference in hip flexor length between people with knee OA and healthy people. The study also sought to understand the link between hip flexor muscle length and trunk inclination in walking and standing in both healthy people and those with knee OA. A secondary aim of this chapter was to explore differences in muscle patterns activation during walking between groups of healthy participants, grouped according to their hip flexor muscle length. The rationale behind this was that, if short hip flexors do underlie changes in trunk inclination, then there should be clear differences in muscle activation (muscle compensations) between people with different hip flexor lengths.

8.2 Research questions

This study aimed to answer the following questions:

Q 1 What are the differences of hip flexor muscle length between people with knee OA and healthy individuals?

Q 2 What is the relationship between hip flexor muscle length and trunk inclination in healthy people and individuals with knee OA in walking and standing?

Q 3 How do muscle patterns differ between healthy people grouped according to their hip flexor muscle length?

8.3 Method

The same data set, used in the previous chapters, and described in the methods chapter was used to address the three research questions above. Along with the data on normal walking for all 60 participants, the hip flexors muscle measurement (Modified Thomas test) was also used. A comparison design was used to address the first and third question and a correlational design to address the second question.

8.3.1 Sample and population

The sample for this study included 20 knee OA patients, 20 healthy older and 20 healthy young subjects, across both genders. The participants' characteristics are summarised in Table 8-1. Once again, the inclusion/exclusion criteria were given before (See section 3.2).

Table 8-1 Participants' characteristics for all groups: people with knee OA, healthy older and healthy young subjects. Values are the mean ± Standard Deviation (SD).

Variables	Knee OA	Old healthy	Young healthy
No. of subjects	20	20	20
Age (Years)	56.0 (8.7)	57.2 (8.7)	26.1 (6.8)
Height (M)	1.7 (.07)	1.7 (.06)	1.7 (.07)
Mass (Kg)	83.1 (14.4)	80 (11.3)	66.1 (8)
BMI (Kg/m^2)	28.7 (4.9)	27.4 (3.9)	22.2 (2.5)

8.3.2 Derivation of outcome measures

In the following paragraphs, the primary outcomes measures that used to answer the research questions listed above are explained in detail:

Trunk angle

As explained earlier in the thesis, trunk angle was tracked using markers on the jugular notch, second and eighth of thoracic vertebral and defined using markers on the greater trochanters and the acromions, for more detail see the literature review, section 3.6.4. For each subject, the ensemble average of the trunk angle (over stance phase) was calculated during walking. This trunk lean was then averaged across stance phase for each participant as used the walking trunk lean outcome for this study. In addition, in order to identify the trunk angle during standing, participants were asked to stand in a stationary position for 10 seconds whilst marker data was captured. An average trunk lean was calculated over this period and used as the standing trunk lean outcome for this study.

Hip flexor muscle length test

The hip flexor muscle length test (Modified Thomas test) was performed for all subjects for both lower limbs. With this test the tested leg was fully extended with the other knee held against up to the participants chest. The angle of the thigh was then measured by positioning a ruler on the line between the lateral aspect of the greater trochanteric and lateral femoral epicondyle of the knee joint (see section 3.5.3 for more details). Average (across both limbs) was then calculated for each participant and used as the outcome measure hip flexor length for this study.

Muscle activity

In order to be able to compare the muscle activity among the groups, all EMG activity was normalized by MVIC signal for each walking trials (See section 3.3.12). Then for each subject, the ensemble average curves for hamstrings, quadriceps and gastrocnemius muscles were calculated during normal walking. In order to understand the effect of hip flexor length (short and long) on lower limb EMG, average of two windows were analysed. The first window was the mean over the specific period of stance phase 10-20% which this period which corresponds to peak loading (see section 3.5). The second window was the mean over the specific period of stance phase used in chapter 7 which is based on the peak values of the EMG.

Defining the healthy groups based on hip flexor length

The third research question was designed to understand whether there is a difference in muscle activation patterns between healthy people who have different hip flexor muscle length. Similar to the design in the previous chapter, this allows us to understand the potential impact of differences in muscle length in people without pain. In order to address this question, it was necessary to divide the healthy subjects (both old and young) into two separate groups. The scatter diagram (Figure 8-1) shows how the subjects were divided into the two groups: short and long hip flexor length. The data were divided using the median, where the first half contains all values above the median (short hip flexor) and the other half contains values below the median (long hip flexor). The middle ten subjects were excluded in this study as they were within the standard error of measurement for the hip flexor length measurement which was approximately one degree (see section 3.5.3.1).

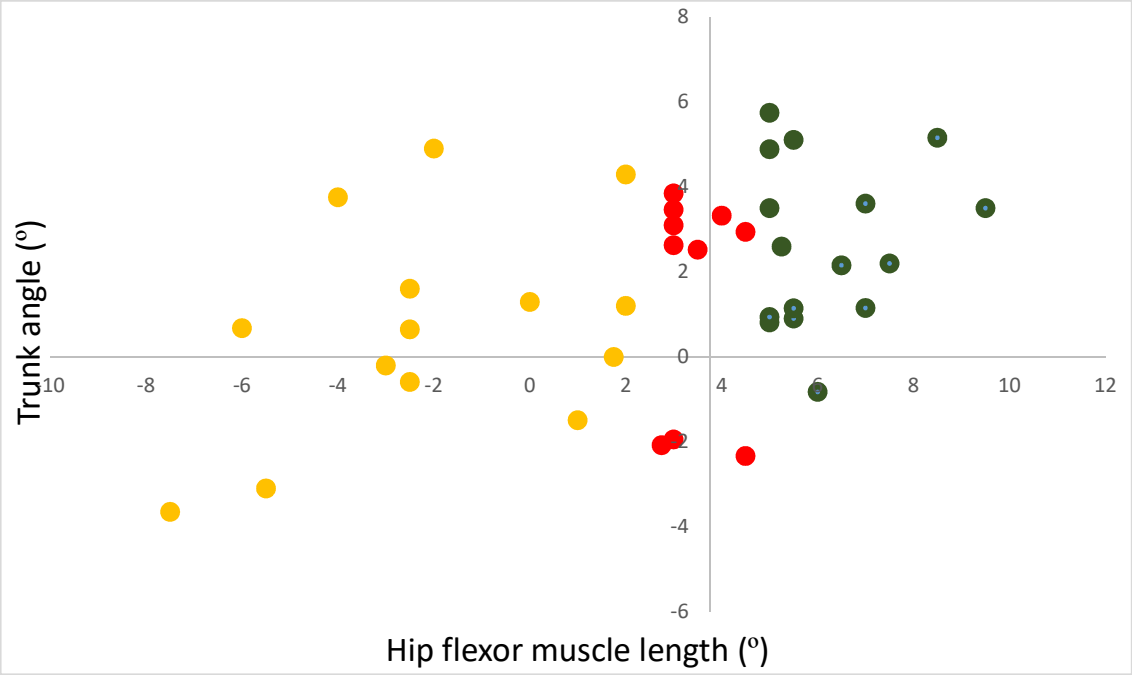


Figure 8-1 The scatter diagram shows the two groups and the excluded subjects. The green dotted is the short hip flexor, the yellow dotted is the long flexor length and the red dotted is middle values which excluded.

The table below shows the characteristics of the two groups defined based on hip flexor muscle length. Apart from hip flexor length, there was no significant differences in any of the variables.

Table 8-2 Participant's characteristics for the short and long hip flexor muscle in healthy groups. Values are the mean ± Standard Deviation (SD).

Variable	Short hip flexor	Long hip flexor
N. of subjects	16	14
Age (Years)	37.5 (16.69)	42.6 (14.4)
Height (M)	1.70 (.07)	1.69 (.07)
Mass (Kg)	69 (8.03)	75 (13.3)
BMI (Kg/m^2)	23.6 (2.2)	25.9 (4.3)
Hip flexor (°)	6.17 (1.34)	-2.05 (2.96)

8.3.3 Statistical analysis

Statistical analysis was performed using SPSS (version 24 for Windows) and Excel. In order to answer the first question ANOVA was used to determine if there were any significant differences between the group means. This test was explained in full in Chapter 4 (section 4.3.3). In order to answer the second question, a Person's correlation (r) test was used to determine the direction and strength of the relationship between the variables. For this test, Shapiro-Wilk's test (P >.05) was used to check that the data was normally distributed. The magnitude of Pearson correlation coefficient determines the strength of the correlation are set out in Table 8-3 (Cohen, 1988). In order to answer the last question, an independent sample t-test was performed to determine if differences existed between the two groups (short and long hip flexor length). This test was explained in full in chapter 7 (section 7.3.3).

Table 8-3 Correlation level (Cohen, 1988).

Coefficient value	Strength of association
.1 < r <.3	Small/ weak correlation
0.3 < r < .5	Medium/moderate correlation
***r* > .5**	Large/strong correlation

8.4 Results

8.4.1 Hip flexor muscle length in patients with knee OA and healthy groups

Table 8-4 shows the hip flexor muscle length between knee OA patients, the older and young healthy people. The Kruskal-Wallis test revealed that there was a difference between the groups (P = .004). The post hoc test revealed that there was only a statistically significant increase in hip flexor muscle length at the knee OA group compared to the older healthy group (Figure 8-2).

Table 8-4 The mean and SD of hip flexor muscle length (º) for knee OA, the older and young healthy subjects.

Variables	Knee OA	Old healthy	Young healthy	ANOVA P value
Hip flexor (º)	6.2 (4.2)	1.9 (3.7)	3.6 (4.1)	.004

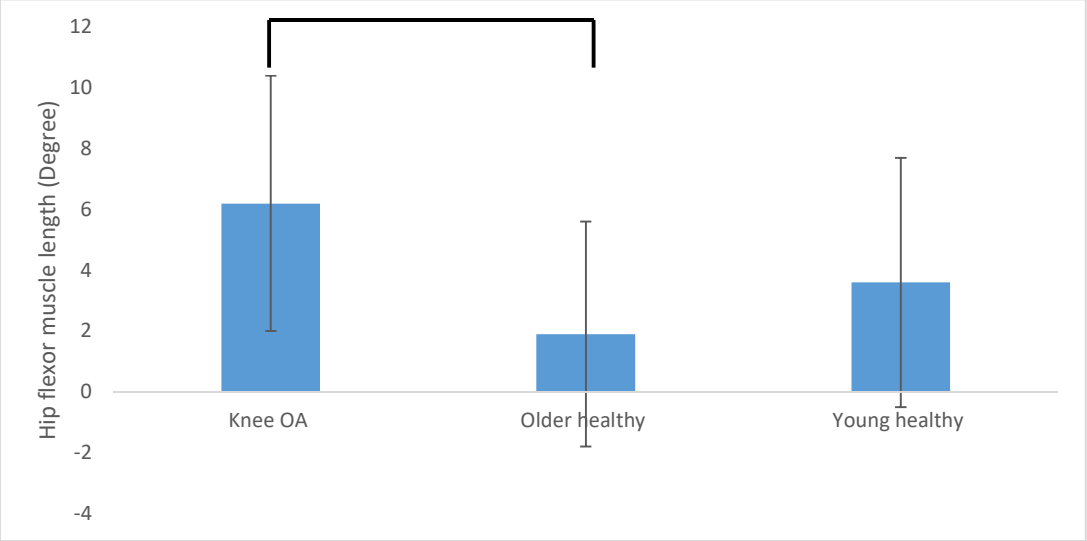


Figure 8-2 Hip flexor muscle length in people with knee OA, older and young healthy groups. The blue bars represent the mean across the 20 subjects and the vertical bars indicate the standard deviation. Horizontal lines indicate significant ($p<0.05$) pairwise differences.

8.4.2 Relationship between hip flexor muscle length and trunk inclination in OA patients

The results of the relationship between hip flexor length and trunk inclination during walking and standing in people with knee OA is shown in Figure 8-3 and Figure 8-4, respectively. Analysis showed that there was a strong positive correlation between the hip flexor length and trunk inclination in people with knee OA during walking (r = .67) while there was a weak correlation during standing (r = .26).

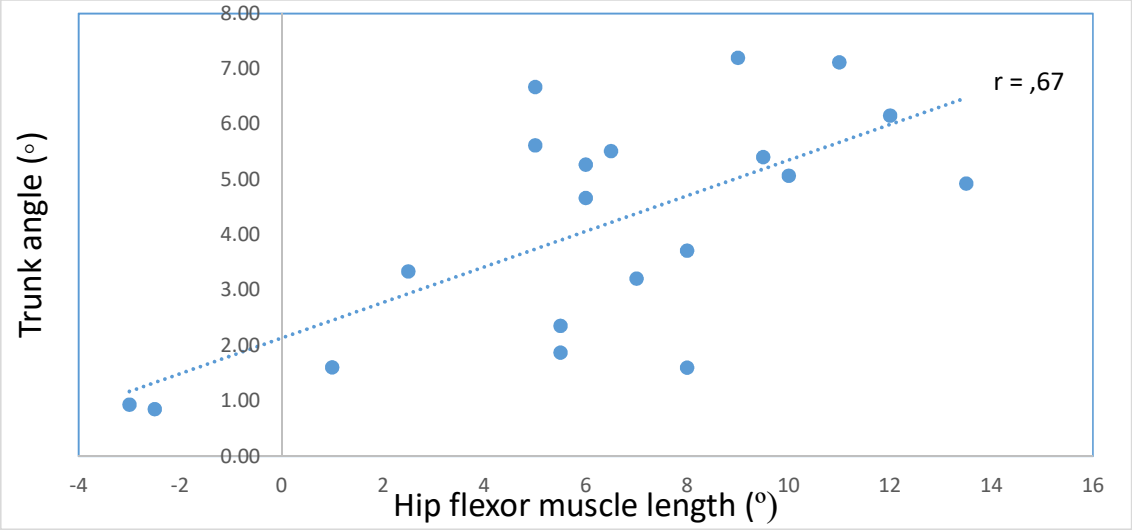


Figure 8-3 Correlation between hip flexor length and trunk inclination during walking in people with knee OA.

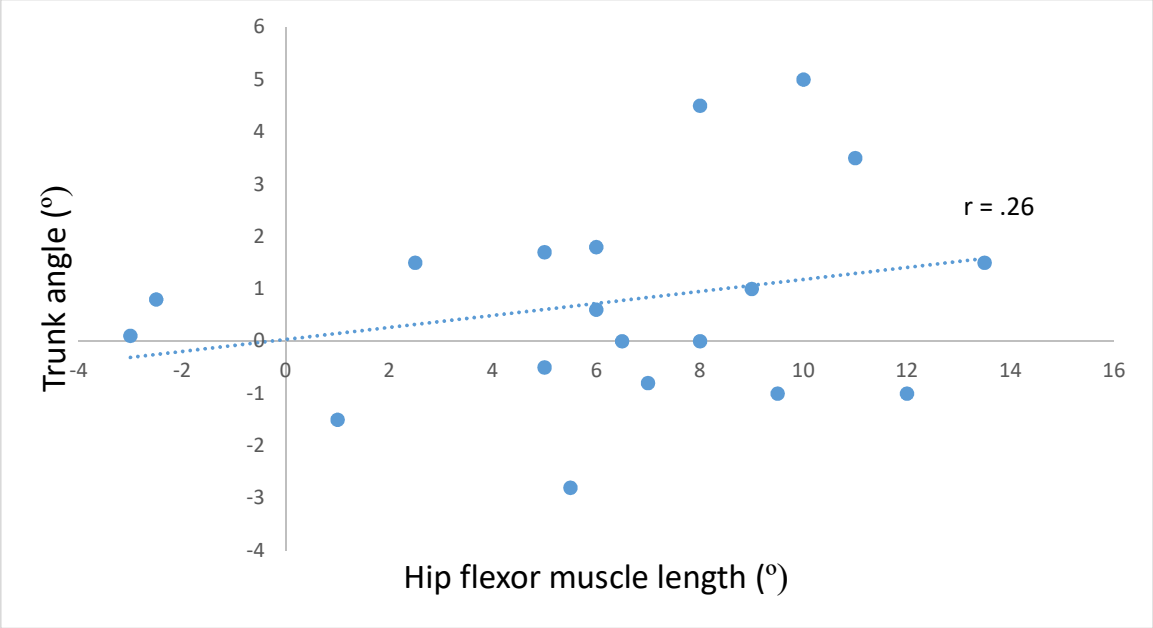


Figure 8-4 Correlation between hip flexor length and trunk inclination during standing in people with knee OA.

8.4.3 Relationship between hip flexor muscle length and trunk inclination in healthy older people

Figure 8-5 and Figure 8-6 show the relationship between hip flexor length and trunk inclination in healthy older individuals during walking and standing, respectively. Person's correlation test revealed that there was a moderate positive correlation between hip flexor length and trunk inclination in healthy older group during both walking (r= .45) and standing (r= .47).

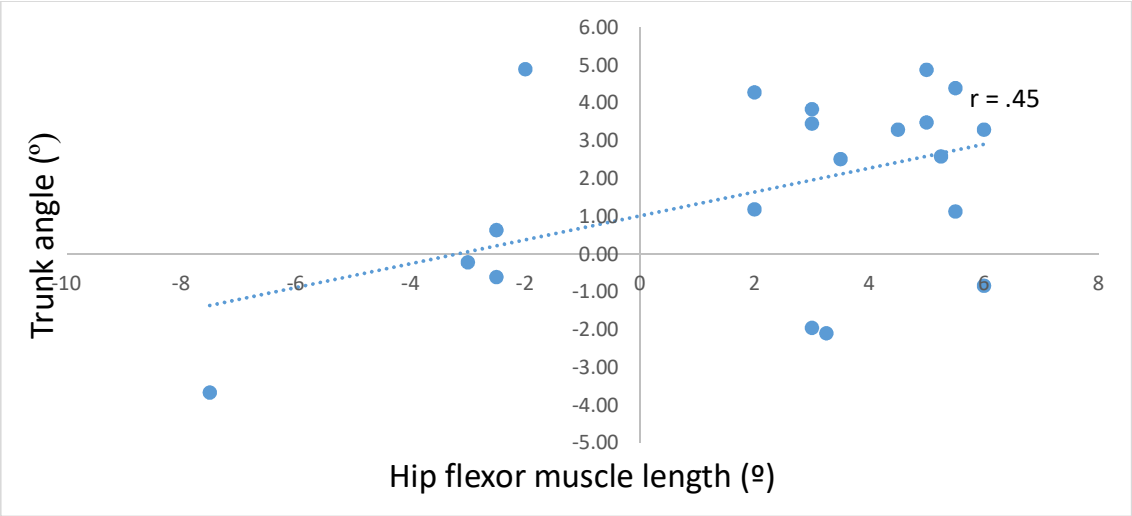


Figure 8-5 Correlation between hip flexor length and trunk inclination during walking in healthy older people.

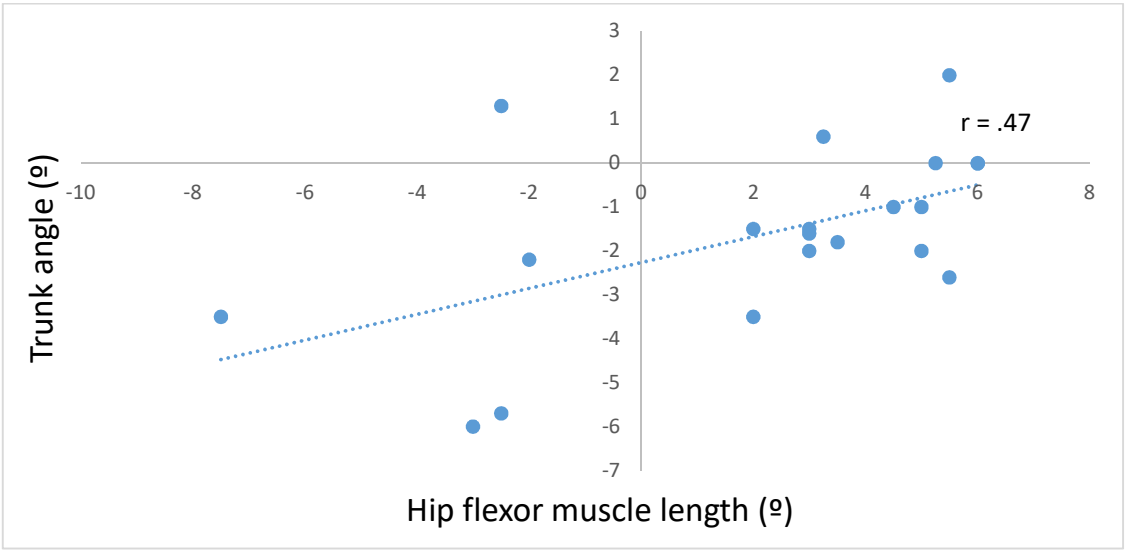


Figure 8-6 Correlation between hip flexor length and trunk inclination during standing in healthy older people.

8.4.4 Relationship between hip flexor muscle length and trunk inclination in healthy young people

The relationships between the hip flexor length and trunk inclination in healthy young subjects during walking and standing are shown out in Figure 8-7 and Figure 8-8, respectively. There was a strong positive correlation between hip flexor length and trunk inclination in healthy young during both walking (r=.53) and standing (r=.60).

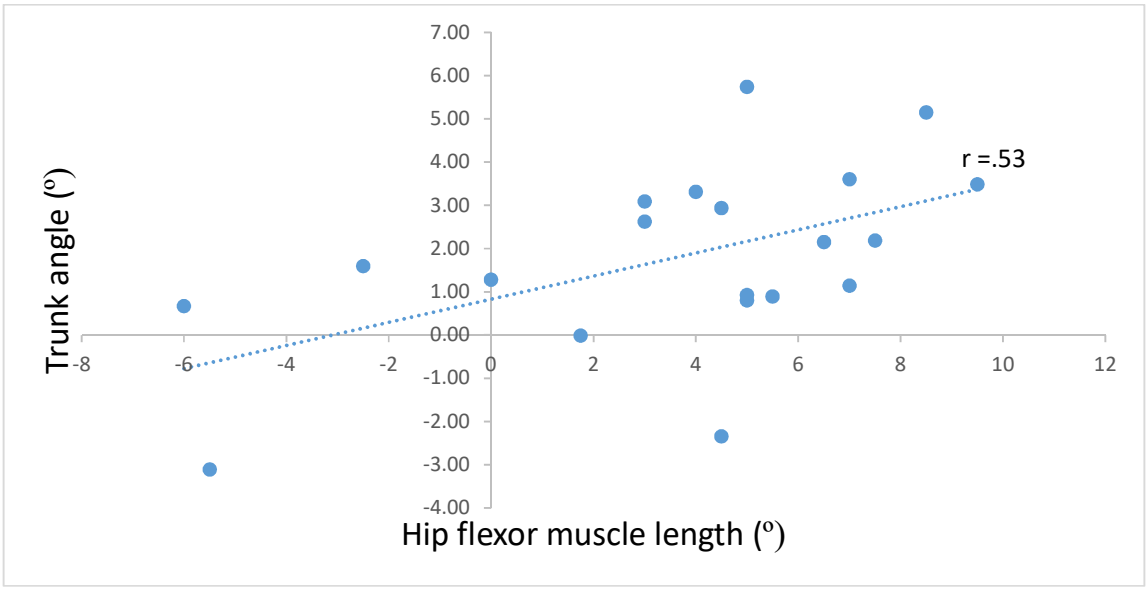


Figure 8-7 Correlation between hip flexor length and trunk inclination during walking in healthy young people.

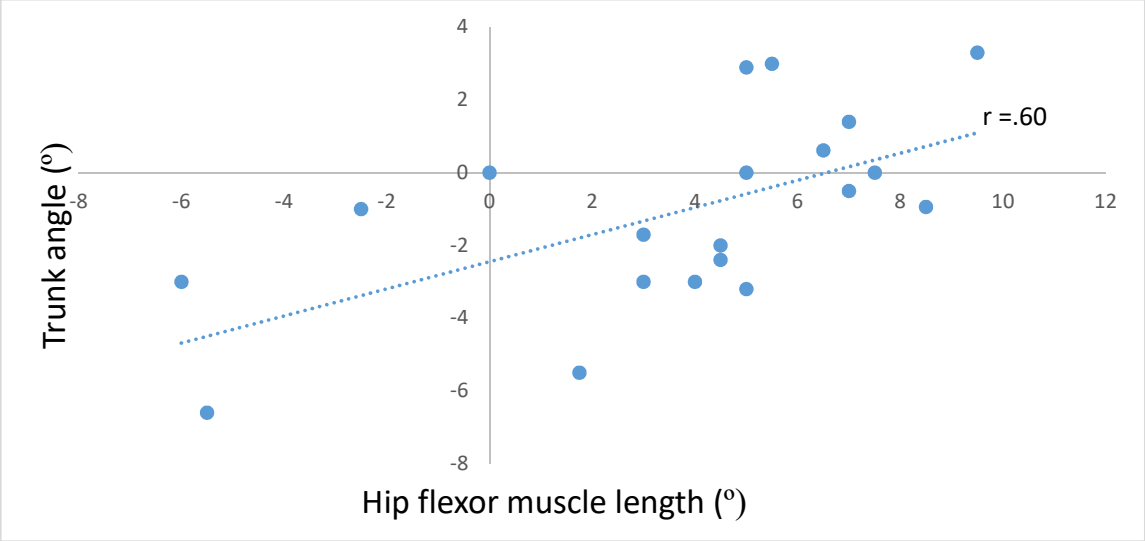


Figure 8-8 Correlation between hip flexor length and trunk inclination during standing in healthy young people.

8.4.5 Relationship between hip flexor muscle length and trunk inclination in the whole cohort

Figure 8-9 and Figure 8-10 show the same relationship for walking and standing. This correlation analysis showed that there was a strong positive correlation between hip flexor length and trunk inclination in both walking (r=.612) and standing (r=.553).

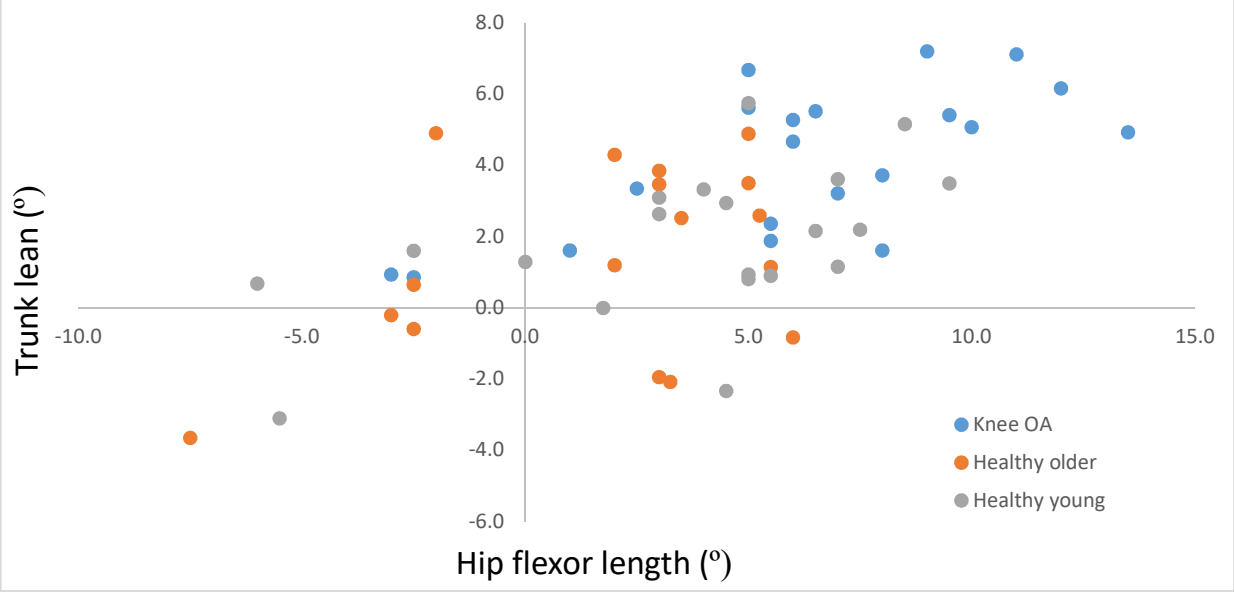


Figure 8-9 Correlation between hip flexor length and trunk inclination during walking in all people.

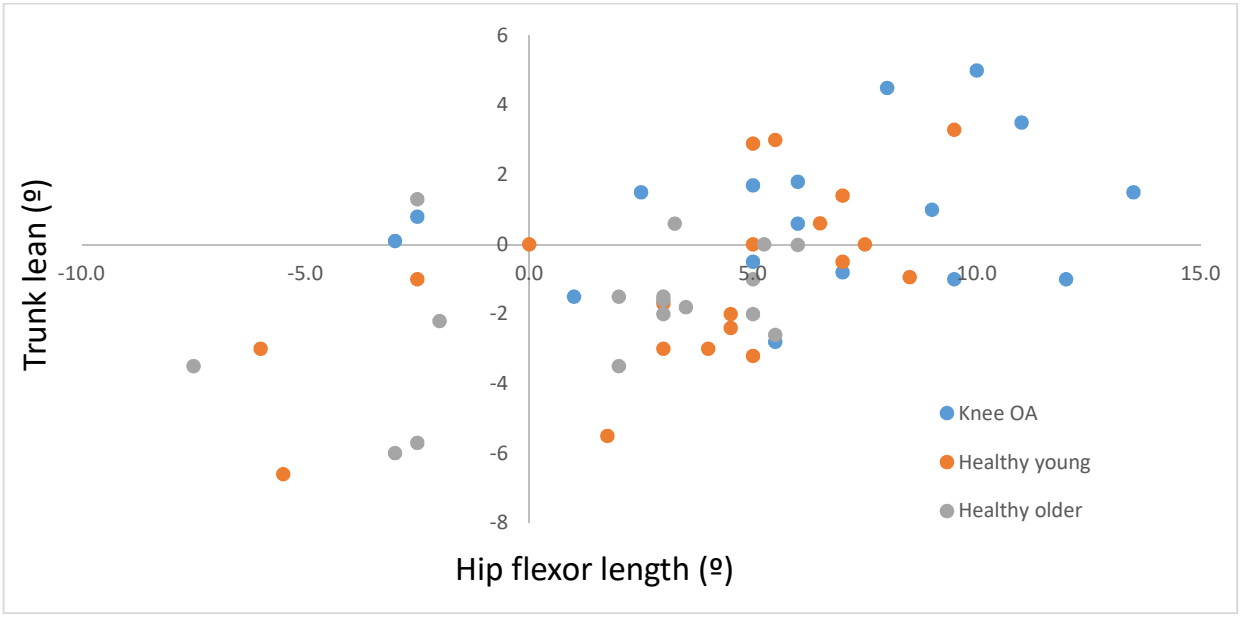


Figure 8-10 Correlation between hip flexor length and trunk inclination during standing in all people.

8.4.6 Effect of short and long hip flexor length on lower limb muscle activity

The plots in the sections below show the ensemble average data for the two groups (long and short hip flexors). It should be noted that the EMG data from patients with knee OA have also been included visual references (for later discussion) but these data were not used for statistical analysis.

Semitendinosus (ST)

Ensemble average profiles the two groups (short/long hip flexor length) along with the knee OA group are shown in Figure 8-11. These plots illustrate clearly a distinct increase in ST activity in the short hip flexor group at approximately 10% of stance phase. Interestingly, this characteristic is also observed in the OA group who also showed prolonged activity across the early stance phase. Despite these visual differences, the mean EMG activity across both windows of 10-20% and -29-0% showed that no differences between the short and long hip flexor groups. These statistical results are presented in Table 8-5.

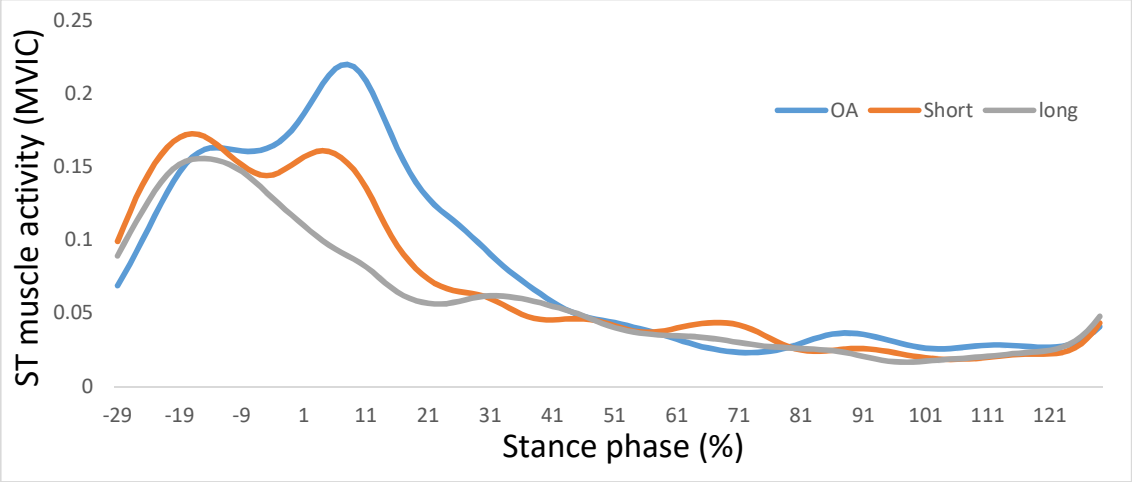


Figure 8-11 The ensemble average curves of ST muscle activity during walking for healthy (short and long hip flexor length) and knee OA group.

Biceps Femoris (BF)

Figure 8-12 illustrate the differences in BF muscle activity between the short and long hip flexor groups during walking. Interestingly, the group with the shorter hip flexors appeared to have delayed activity of the BF muscle. However, the activity in this group still appeared to be lower in magnitude than in the OA group. Although there was an difference of approximately 30% in BF muscle activity across 10-20% stance in the short hip flexor group this difference did not reach significance (P = .18). No difference was observed across the period -29-0% (P=.25).

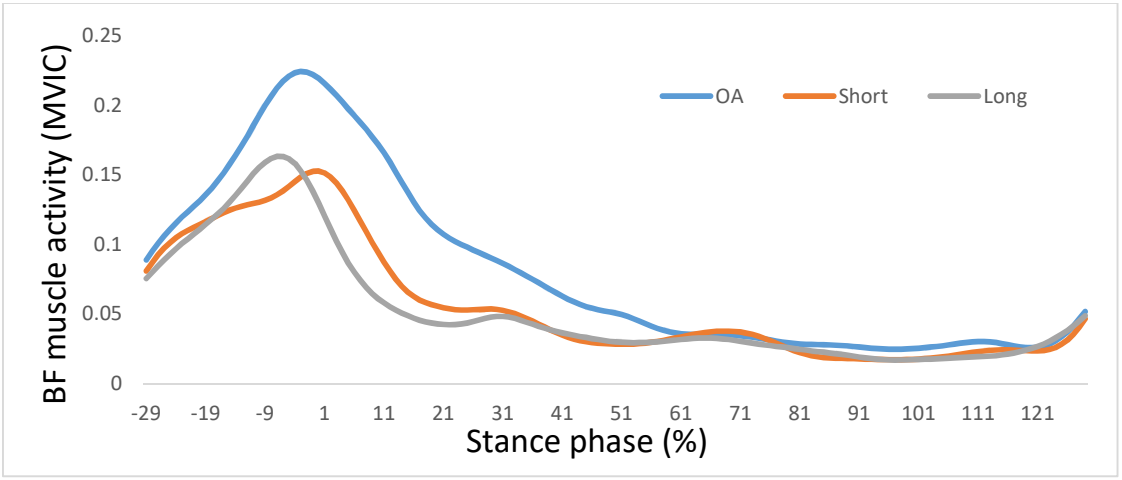


Figure 8-12 The ensemble average curves of BF muscle activity during walking for healthy (short and long hip flexor length) and knee OA group.

Vastus medialis oblique (VMO)

The pattern of VMO muscle activity in people with knee OA, short and long hip flexors are presented in Figure 8-13. It is clear that VMO activity was very similar between the short and long hip flexor groups. However, individuals with OA had high VMO activity across the stance phase (Figure 8-13). No differences was found between the two groups during either periods (Table 8-5).

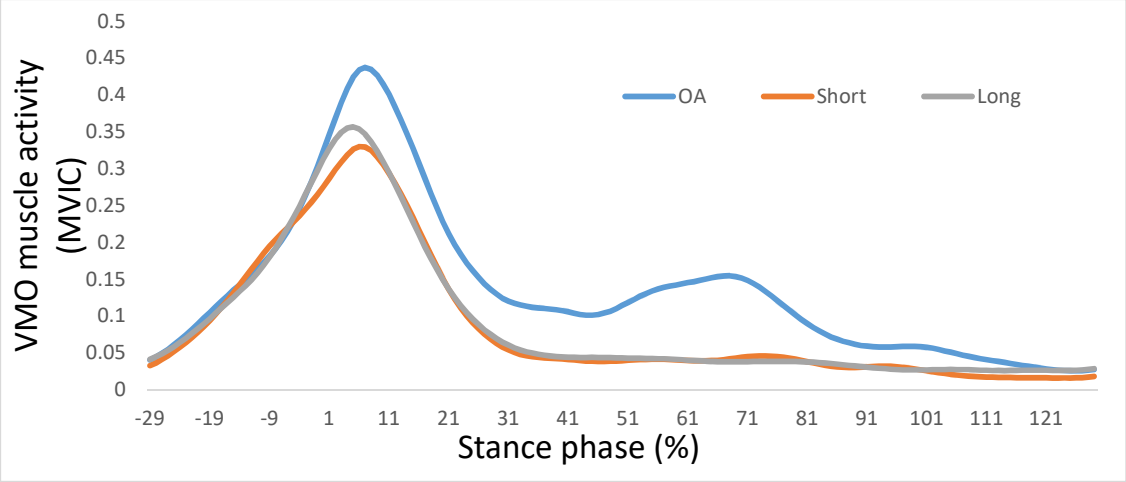


Figure 8-13 The ensemble average curves of VMO muscle activity during walking for healthy (short and long hip flexor length) and knee OA group.

Vastus lateralis oblique (VLO)

The ensemble average curves in Figure 8-14 illustrate the characteristic pattern of VLO across the stance phase of walking. The plot illustrates the differences between the groups, with minimal difference in activity between the short and long hip flexor length groups and an overall higher activity in knee OA group. The analysis showed no differences between the short and long hip flexor groups across either the 10-20% or the -20-10% period.

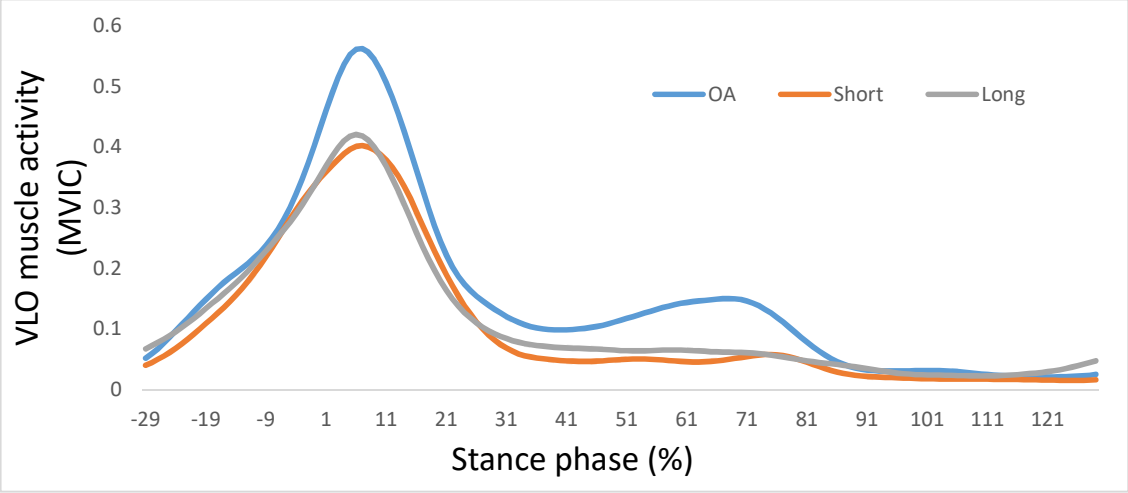


Figure 8-14 The ensemble average curves of VLO muscle activity during walking for healthy (short and long hip flexor length) and knee OA group.

Medial gastrocnemius (MG)

The plots below show the ensemble average of the normalised MG activity for both the healthy (short and long hip flexor length) groups and the knee OA group (Figure 8-15). This plot illustrates clearly that MG activity was lower in the knee OA group for almost all of stance phase. Nevertheless, there was a little difference between the short and long hip flexor groups. No statistical differences were observed between the short and long hip flexor groups in either periods (Table 8-5).

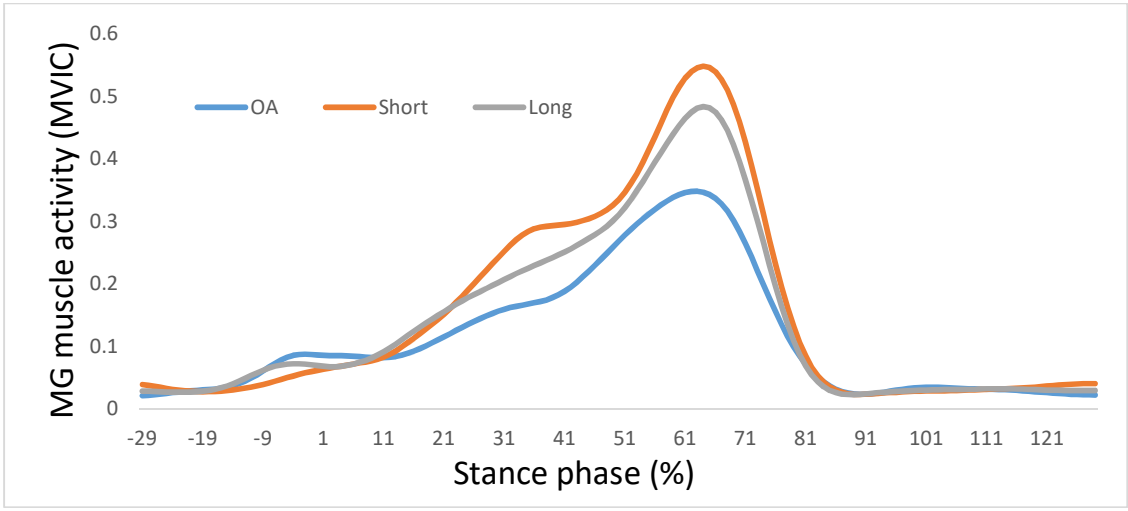


Figure 8-15 The ensemble average curves of MG muscle activity during walking for healthy (short and long hip flexor length) and knee OA group.

Lateral gastrocnemius (LG)

Figure 8-16 shows the LG activity in people with knee OA, and the healthy groups with short and long hip flexors during walking. As shown in Figure 8-16, LG activity was higher in the knee OA group over the early and mid-stance, with no differences in the patterns between the healthy groups. Analysis found no significant differences between the short and long hip flexor length across either of the two time windows (10-20% or 55-85%).

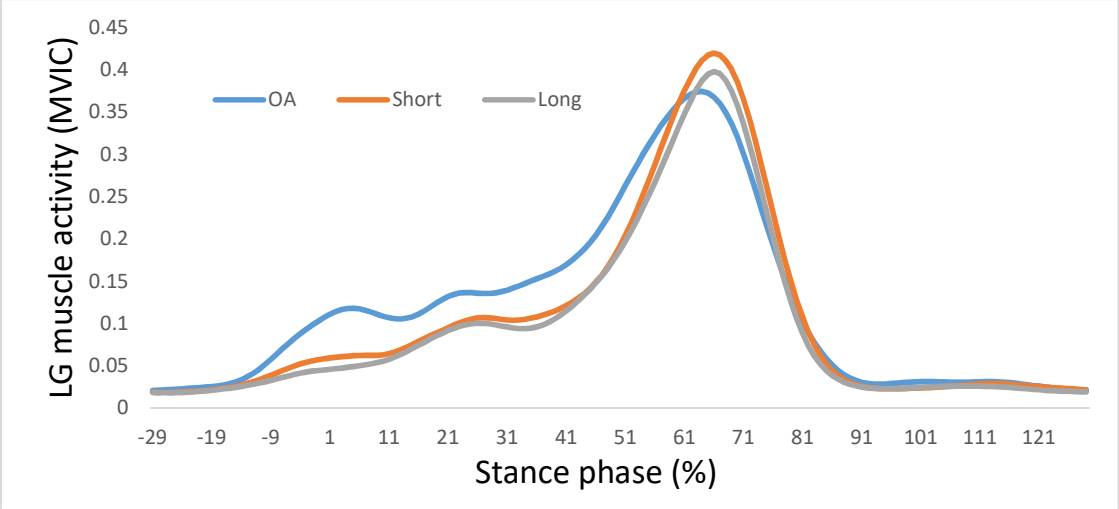


Figure 8-16 The ensemble average curves of LG muscle activity during walking for healthy (short and long hip flexor length) and knee OA group.

Table 8-5 Result summary of the differences in the EMG (MVIC: proportion of the MVIC) between the two groups (short and long hip flexor) over 10-20%. Values are the mean ± Standard Deviation (SD).

EMG	**10-20% period**		
	Short hip flexor	**Long hip flexor**	**P**
MG	.10 (.04)	.11 (.09)	**.76**
LG	.07 (.04)	.07 (.03)	**.74**
VMO	.23 (.19)	.21 (.11)	**.80**
VLO	.30 (.30)	.28 (.13)	**.77**
ST	.10 (.09)	.07 (.04)	**.21**
BF	.06 (.04)	.05 (.02)	**.18**

Table 8-6 Result summary of the differences in the EMG (MVIC: proportion of the MVIC) between the two groups (short and long hip flexor) over specific window. Values are the mean ± Standard Deviation (SD).

EMG (Average window)	Short hip flexor	Long hip flexor	P
MG (55-85%)	.35 (.15)	.30 (.07)	**.33**
LG (55-85%)	.28 (.13)	.25 (.06)	**.54**
VMO (-20-10%)	.20 (.17)	.22 (.12)	**.96**
VLO (-20-10%)	.27 (.22)	.28 (.13)	**.86**
ST (-29-0%)	.15 (.06)	.14 (.08)	**.70**
BF (-29-0%)	.12 (.05)	.12 (.04)	**.81**

8.5 Discussion

8.5.1 Overview of the result

The last study in this thesis was designed to understand the difference in hip flexor length between people with knee OA and healthy controls. In addition, the relationship between hip flexor muscle length and trunk inclination during walking and standing was investigated in both healthy and OA groups. The data showed that hip flexor length was shorter in patients with knee OA compared to the older healthy subjects. In addition, results showed that there were a strong to moderate correlation between hip flexor length and trunk inclination in both patients with knee OA and healthy individual during walking and standing. However, result revealed that there was a weaker correlation in patients with knee OA during standing. A further aim of this study was to understand the differences in muscle activation between two groups of healthy with distinctly different hip flexor muscle lengths. Although there were no statistically significant differences, the data showed a trend for a second peak in ST activity

around 10% stance which was similar to the profile observed in the people with knee OA. However, there were minimal differences in the other muscle activation patterns. To date, there has been no previous research investigating hip flexor muscle length in people with knee OA and linking hip flexor muscle length to trunk inclination in either people with knee OA or healthy people. Therefore, a broad discussion of this area has been included and parallels drawn to related studies wherever possible.

8.5.2 Interpretation and implications of the findings

This study found that the hip flexor muscles were significant shorter in patients with knee OA compared to the older healthy participants. Similarly previous studies showed that shortening of the hip flexor muscle length is common complication of neurological or musculoskeletal conditions (Attias et al., 2016). Specifically, it has been suggested that a restriction in hip flexor muscle length might be a risk factor in patients with low back pain (Kolber & Fiebert, 2005). In addition, Mills et al. (2015) studied the effect of shortening the hip flexor muscle length on lower limb biomechanics in adult female subjects during double-leg squat. They concluded that people with shortening of the hip flexor muscles could limit the range of hip extension and suggested that this might lead to increased risk of a lower limb biomechanics injury, for example hamstring muscle injury (Mills et al., 2015). Taken together, these studies demonstrated that shortening in hip flexor muscle length could impair human gait and daily living activities.

In addition, this study found some evidence that older healthy people had longer hip flexors compared to young healthy people, however importantly this result was not significant. Nevertheless, it is interested to speculate on why this may be case. There is evidence that shorter hip flexor muscles may be associated with musculoskeletal problems, such as low back pain (Attias et al., 2016). Given that people with low back pain or musculoskeletal problem were excluded from this study, this may have led to the exclusion of people with short hip flexors (and therefore at risk of musculoskeletal pain). Although younger people were also excluded who had musculoskeletal pain, it possible that younger people have increased tolerance of musculoskeletal and therefore could have shorter hip flexors but no pain. This might explain why the younger healthy people appeared to have shorter hip flexor muscles compared with the older group. This study appears to be the first study to provide evidence

that the hip flexor muscle is shorter in patients with knee OA. Therefore, these findings might motivate other researchers to investigate whether current clinical approaches for hip flexor length might be appropriate for patients with knee OA.

The physiological mechanism that could underlie change in hip flexor muscle length in people with knee OA is likely to relate to physiological changes within the muscle. Several lines of evidence suggest that if muscles are frequently maintained at a length that is either shorter or longer than that needed for optimal posture, they can adapt to this position (Gossman et al., 1982; Williams & Goldspink, 1978). The mechanism behind this has been linked to a reduction/increase in the number of sarcomeres in series in the muscle (Williams, 1990; Williams & Goldspink, 1973, 1978). As explained earlier, people with knee OA tend to lead a sedentary lifestyle, spending more time sitting and less time active (Lee et al., 2015; Sliepen et al., 2018; Wallis et al., 2013). Results in this chapter showed that a positive relationship between hip flexor length and trunk inclination, demonstrating a potential link between trunk flexion, altered lower limb muscle patterns, which could explain the leaning forward in those with knee OA compared to the older healthy participants. Further research is required to fully understand whether the finding here of shortening hip flexors is the result of prolonged sitting or whether it is an adaptation to the disease. However, it would appear feasible that inactivity is a contributing factor to hip flexor changes. If this is the case, then the current clinical guidance to remain active (Sliepen et al., 2018) is very relevant.

The data indicated a strong correlation between hip flexor muscle length and trunk inclination during walking in people with knee OA. However, a weak correlation was observed in the same group during standing. This difference might be explained by compensations in other parts of the body to the shortening of the hip flexor muscles. Specifically, shortening of the hip flexor muscles will influence the sagittal plane orientation of the pelvic (anterior pelvic tilt) as was noted by (Kagaya et al., 2003). Other research suggested that during a restriction in the hip flexor length could affect the lumber lordosis (Shimada, 1996a). Therefore, the finding of a lower correlation in standing might indicate more variability in the lumbar compensation than occurs in walking in people with knee OA.

The results in this chapter showed a moderate to strong relationship between hip flexor muscle length and trunk inclination in both people with knee OA and the healthy groups. This

is consistent with Krol, Polak, Szczygiel, Wojcik, and Gleb (2017) who studied the relationship between hip flexor muscle length and pelvic tilt in adults with low back pain using a Modified Thomas test. They observed a positive relationship between the hip flexor muscle length and anterior pelvic tilt; however, this result was not significant. Interestingly, but contrary to findings in this current study, no relationship was observed between hip flexor muscle length and pelvic inclination in healthy adult people in a study by Youdas, Garrett, Harmsen, Suman, and Carey (1996). This lack of a correlation could reflect the difficulty in measuring pelvic tilt which can be confounded by inter-subject variation in bony anatomy (Preece et al., 2008). Nevertheless, the data in this current study demonstrate a clear link between shortening of the hip flexor muscles and forward lean and this finding warrants further investigation.

The final part of this study showed a trend toward increased hamstring activity in the short hip flexor group. However, no significant differences were observed across the period of interest (10-20% of stance phase). This contrasts with the findings in the previous study (Chapter 7), which demonstrated clear differences in hamstring patterns between two groups of healthy people with distinct differences in sagittal trunk lean. In order to interpret this, it is important to note that although strong, the correlations between trunk inclination and hip flexor muscle length were not perfect. Correlations of 0.6-0.7 suggest that no more than 50% of the variance in trunk inclination can be explained by variability in hip flexor length. Given the clearer differences in the previous chapter, it would appear that increased forward lean is the main driver of increased hamstring patterns and that a shortening of the hip flexors predisposes an individual to have increased forward lean. However, as discussed above, this link is not straightforward as there can be possible compensations in the lumbar spine. Nevertheless, the data here are to some degree consistent with a study by Mills et al. (2015) who observed a trend towards increased biceps femoris muscle activity in a group of female soccer players with short hip flexor muscles during double-leg squat. Similar to this study, statistical differences were not observed and this may again indicate the need to look upper body position to better understand muscle differences.

Although there were no statistical differences in ST activity between the groups with short/long hip flexors, Figure 8-11 shows a trend for ST muscle activity to be prolonged in people with short hip flexors. Interestingly this trend was observed in the people with knee OA but was not observed as clearly between two healthy groups with different trunk leans

(previous chapter, Figure 7-11). This subtle difference may indicate a different mechanism whereby a short hip flexor may lead to compensatory increased hip extensor activity which is not the direct result of increased forward lean. Further work is required to explore this idea. Whatever the mechanism, the findings of this study support further investigation into the use of hip flexor stretching exercises in people with knee OA which may, by reducing forward lean, lead to a reduction in hamstring activation during walking.

8.5.3 Limitation

In this investigation there are a number of limitations which should be discussed. First, the modified Thomas test was used to provide an indication of muscle length, however, this test cannot be used to obtain a direct measure of hip flexor muscle length. Nevertheless, previous investigation has found that this test is valid (Vigotsky et al., 2016) and reliable (Kim & Ha, 2015; Wakefield, Halls, Difilippo, & Cottrell, 2015) as a measure of hip flexor muscle length. In addition, a test-retest reliability of the modified Thomas test was conducted and the result showed excellent reliability between days (see section 3.5.3.1). The robust nature of this measurement and the lack of any other non-invasive technique for measuring hip flexor length, support the use of this approach in our design.

Another source of uncertainty is that, in order to define the two groups (short and long hip flexor) for the final research question, 10 subjects with a middle range were excluded. This band was based on the reliability of the modified Thomas test which had a SEM of 1.2^{o}. Although this approach reduced the number of participants which could be used to address the final research question, it was a necessary step to properly define the groups. Unlike with the second research question, a correlational design was not deemed appropriate given the inherent variability in EMG. However, despite showing some visual differences, no statistically significant differences were found in hamstring activity between the groups with short/long hip flexors. It is possible that this lack of a difference reflects the small sample size and therefore further work is required to explore these ideas further on a larger cohort.

The final limitation relates specifically to the anatomical configuration of the three primary hip flexor muscles: iliopsoas, rectus femoris and adductor longus. With the modified Thomas test used here, no attempt was made to differentiate between the three muscles and to

identify if shortness was present in one, two or all three of the muscles. It is possible that different effects on trunk position and/or lower limb biomechanics may correspond to shortening of the different component muscles. However, the main aim of this study was to understand, in general, the relationship between the hip flexor muscle length and trunk inclination. Further work should now explore the potential links between specific differences hip flexor length and trunk position/lower limb biomechanics.

8.5.4 Conclusion and clinical relevance

This is the first study to demonstrate that people with knee OA have short hip flexor muscles when compared to age-matched controls. The data also support the idea that short of hip flexor muscles will lead to increased forward lean both in patients with knee OA and also healthy individual during walking and standing. However, there was no direct link between hip flexor muscle length and muscle patterns and walking, suggesting that alterations in hamstring patterns are primarily the result of alterations in upper body position. Previous studies demonstrated different type of exercise that could help patients with knee OA. These include strengthening exercises, Tai Chi exercises, general stretching exercise and manual therapy (Anwer et al., 2018; Bartholdy et al., 2017; Bennell et al., 2016; Fransen et al., 2015). It is possible that current interventions for managing knee OA are not effective because they do not directly target the knee joint loading. Therefore further investigations are needed to assess the potential effectiveness of clinical programmes, such as stretching the hip flexor muscle in patients with knee OA which could facilitate and improve trunk position during walking and decrease sagittal trunk lean. If successful, this may reduce co-contraction and lead to decrease the joint loading and pain for people with knee OA.

Chapter Nine

Final discussion and future work

9.1 Introduction

The aim of the work presented in this thesis was to understand the potential effect of trunk flexion on lower limb kinetics, muscle activation and muscle co-contraction in people with knee OA and healthy participants during walking. Moreover, this thesis aimed to explore the biomechanical mechanisms which relate to increased trunk flexion in both healthy individuals and those with knee OA. This has been achieved with the five studies. In this chapter, a summary and conclusion for the studies conducted throughout the thesis are presented. Furthermore, several recommendations for the future work are suggested.

9.2 Summary

Study one:

The first study was designed to understand the key biomechanical differences between people with knee OA and healthy controls during walking. The main findings were that people with knee OA walk with an increased sagittal plane inclination of the trunk. In addition, patients with knee OA walked with higher hamstrings and quadriceps muscle activations along with a corresponding increase in hamstring-quadriceps co-contraction compared to the healthy groups.

Study two:

This study aimed to understand the effect of increased/decreased trunk flexion (-5^{o}, NW, +5^{o} and +10^{o}) on lower limb biomechanics during walking in young healthy subjects. The data indicated that, in healthy young people, increasing trunk inclination leads to corresponding increases in the hip extensor moment, the ankle plantar flexor moment, an associated increase in hamstring and gastrocnemius activity and an increase in hamstring-quadricep co-contraction.

Study three:

This study aimed to fully understand how increasing/decreasing trunk lean (-5º, NW and +5º) would affect lower limb biomechanics in people with knee OA and matched healthy controls. The results showed similar effects to study two, demonstrating the effect of increasing trunk lean was similar to that observed in the young healthy people. However, interestingly, although decreasing trunk lean lead to a decrease in hamstring activity in the younger people, this effect was not observed in the people with knee OA or the older healthy group.

Study four:

This study aimed to understand the effect of inter-subject variability in natural trunk inclination on lower limb biomechanics during walking in healthy subjects. This study showed the peak hip extensor moment, hamstring and gastrocnemius muscle activation were higher in healthy people who habitually walk with a forward lean compared to people who walk with less trunk inclination. Taken together, the data presented in studies 2, 3 and 4 provide strong evidence that some aspects of the altered lower limb moments and muscle activation patterns observed in people with knee OA could be the result of increased trunk lean. Specifically, the increased trunk inclination in people with OA might explain increased hamstring activity, increased hip moments and go some way to explaining increased co-contraction. however, it is unlikely to explain increased quadriceps activity.

Study five:

This study aimed to explore the biomechanical mechanisms which could underlie differences in forward lean during walking. Specifically, a hypothesis related to hip flexor muscle length and trunk inclination was explored. In addition, this study aimed to quantify possible differences in muscle activation between two groups of healthy with distinctly different hip flexor muscle lengths. This study showed that the people with knee OA have shorter hip flexor muscles compared to healthy people and also that there was a relatively strong correlation between the hip flexor muscle length and trunk inclination during walking. However, although there was some evidence that people with short hip flexors had increased hamstring activity, this did not reach statistical significance.

9.3 Thesis novelty

Figure 9-1 illustrates a flow diagram of a new model for OA progression which incorporates the idea that a sedentary lifestyle could lead to muscle length changes which subsequently lead to changes in trunk flexion and associated changes in muscle activation. This model represents a multifactorial approach to OA development in which both sedentary lifestyle patterns, along with knee pain, can precipitate changes in trunk flexion and muscle activation which may lead to joint degeneration. At the top of the model is the idea that sedentary lifestyles (prolonged sitting) leads to a shortening of the hip flexor muscles. Short hip flexors then create a forward lean (increased sagittal trunk flexion) which leads to increased knee muscle activity and co-contraction. This increased muscle activity increases loading on the knee joint which then accelerates disease progression. However, in parallel with this linear model, it is possible that increased loading on the joint could trigger increased pain which in turn could trigger yet more muscle activity, creating a vicious cycle. The evidence (both from the literature and from this thesis) supporting this model is discussed in the sections below.

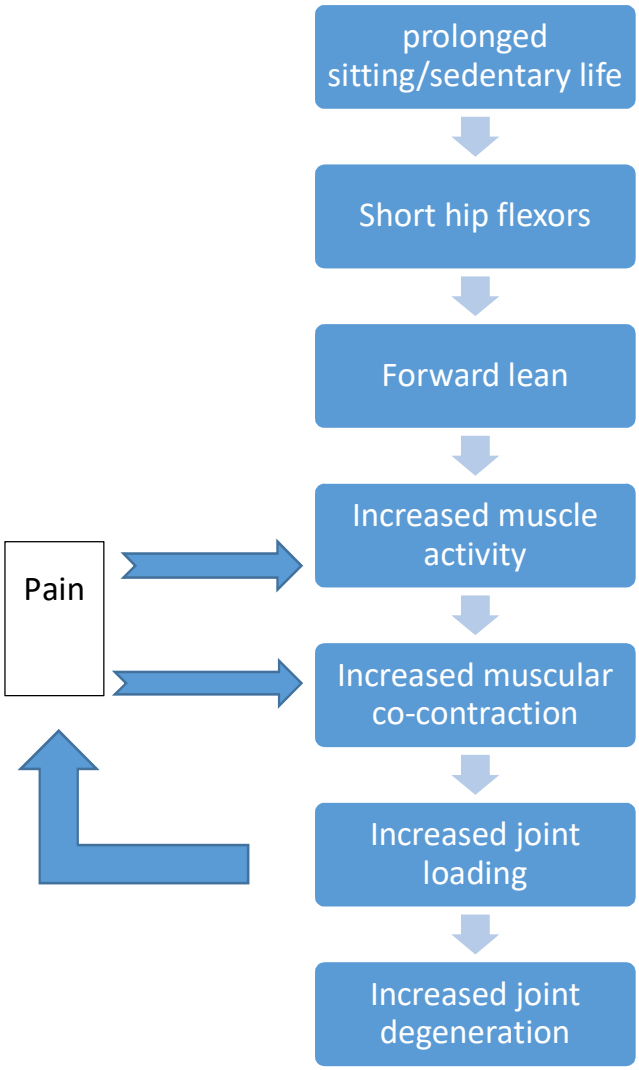


Figure 9-1 Flow diagram of new model to explain OA progression based on sedentary lifestyle.

Previous studies have observed that people with knee OA spend more their time sitting and are less active, with a sedentary lifestyle (Lee et al., 2015; Sliepen et al., 2018; Wallis et al., 2013). In a sitting position, the hip flexor muscles are in shortened position and there is some evidence that muscles, maintained in a shortened position, will adapt and retain a lower length during movement (Gossman et al., 1982; Knutson & Owens Jr, 2003; Williams & Goldspink, 1978). Such a shortening of the hip flexor muscles will lead to an anterior tilt of the pelvis and a corresponding forward lean. Evidence for this idea was presented in chapter 8 which showed shorter hip flexors in people with knee OA and a positive relationship between hip flexor length and trunk inclination during walking, both in people with knee OA

and also in healthy people. Further evidence supporting this idea was presented in chapter 4 (Study 1) and in previous research (Preece et al., 2018) showing that people with knee OA walk with an increase in the sagittal plane inclination of the trunk of approximately 3º. Taken together these findings support the idea that people with knee OA have short hip flexors and that they walk with an increase in trunk inclination.

The next step in the model is the link between forward lean and increased muscle activity. A central part of this thesis was dedicated to this idea and evidence was provided to show that hamstring muscle patterns and co-contraction will increase when heathy people (Chapter 5) and people with knee OA (Chapter 6) are instructed to walk with an increase in natural trunk inclination. It is interesting to compare the data from the healthy older people to the data from the people with knee OA. **Error! Reference source not found.** illustrates hamstring muscle activity patterns during normal walking for people with knee OA (red) and for older healthy (NW, black). Also shown are the muscle patterns when the older healthy walk with a 5° increase in their trunk angle (green). Importantly, there is a pronounced change in the healthy patterns, so that become similar to those typical knee OA, when they are instructed to walk with increased trunk lean. For example there was a 175% increase in semitendinosus activity and a shift in the peak in semitendinosus activity to later in stance, matching the profile observed in the group with knee OA. These findings demonstrate a clear link between forward lean and muscle activity/contraction.

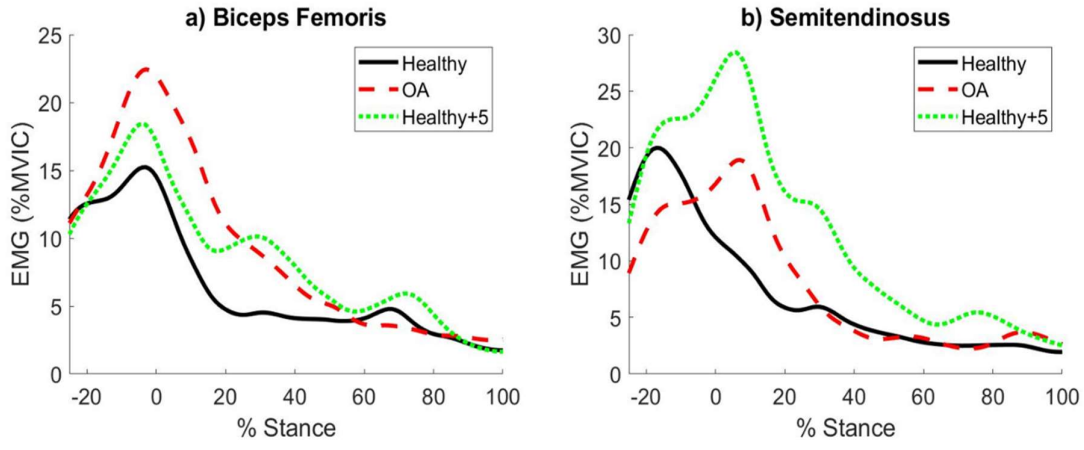


Figure 9-2 Biceps femoris (A) and Semitendinosus (B) muscle activity during walking in people with knee OA (red), older healthy (NW, black) and older healthy (NW+ 5°, green).

Further evidence to support the link between forward lean and increased muscle activity was provided in chapter 7. These data showed clear differences in hamstring and gastrocnemius patterns between groups of healthy people who habitually walk with a difference in trunk angle. This shows that patterns of muscle activation, characteristic of people with knee OA, can occur in health people and therefore and not simply a result of pain and/or structural abnormalities. However, it is also important to acknowledge that increased pain can often trigger increased muscle activity (Wilson et al., 2011). This idea is captured in the model shown in Figure 9-1.

Numerous studies have reported that patients with knee OA walk with increased knee muscle co-contraction (Heiden et al., 2009; Lewek et al., 2004a; Winby et al., 2013). To date, numerous ideas have been proposed to explain the increased muscular co-contraction. For example it has been hypothesised to control the knee adduction moment during walking (Lewek et al., 2004a), to increase stability (Childs et al., 2004) or as a localised muscular response to pain (Hubley-Kozey et al., 2006). However, the data in this thesis supports the idea that it could be the result of an altered upper body position. Regardless of the cause, there is now strong evidence to support the final steps in the proposed model that altered muscle patterns will increase joint loading (Brandon et al., 2014; Sritharan et al., 2016) and accelerate cartilage loss (Hodges et al., 2016a; Hubley-Kozey, Hatfield, et al., 2013).

9.4 Implication for clinical practice

Given the model proposed above, there is a need to investigate interventions that can decrease sedentary activity patterns for example walking, cycling, and swimming in people with knee OA. This thesis showed a relatively strong link between hip flexor muscle length and trunk lean and therefore, further research is required to fully understand whether the finding of shortening hip flexors is the result of prolonged sitting or whether it could be some form of adaptation to knee OA. In addition, therapeutic approaches such as a programme of tailored stretching exercises for the hip flexor muscle may facilitate and improve trunk position during walking and then decrease the sagittal trunk lean in people with knee OA.

There is a clear need to investigate further intervention which can effectively improve upper body posture (decrease trunk lean) during walking. If successful, this could may reduce co-contraction and lead to clinical benefits for people with knee OA. Interestingly, the findings of this thesis do not support that simply instructing people with knee OA to decrease trunk lean during walking may not change muscle activation patterns. Specifically, our data showed that when people with knee OA were instructed to walk 5°with less trunk lean, the decrease in muscular co-contraction was small and non-significant. Therefore, other interventions are required that could decrease trunk lean in those with knee OA. If successful, such interventions may reduce knee muscle co-contraction which may lead to a corresponding reduction in the compressive forces at the knee and improvement in symptoms.

9.5 Global thesis limitations

A number of important limitations need to be considered. Firstly, this thesis did not examine the gait differences between the affected and unaffected limb in people with knee OA. Only the affected limb was evaluated in this thesis. However, previous research has investigated the biomechanical differences between the ipsilateral and contralateral limbs in individuals with knee OA (Chan, Smith, Kirtley, & Tsang, 2005; Gustafso et al., 2019; Metcalfe, Andersson, Goodfellow, & Thorstensson, 2012). These studies typically find a consistent pattern of altered muscle activity between the affected and unaffected limb which has led researchers to conclude that the altered gait characteristics in the affected leg may leads to compensatory changes in the contralateral limb. However, the ideas explored in this thesis challenge this notion and suggest that the consistency between the affected and unaffected limb could be the result of altered upper body position during walking. However, future work, investigating both limbs in people with knee OA is required to explore the idea further.

Another limitation of the current thesis was that the older healthy participants were matched for BMI & age to the group with knee OA, but no matching or measurement of physical activity was undertaken. Recent studies suggest that people with knee OA spend more their time sitting and are less active, with a sedentary lifestyle (Lee et al., 2015; Sliepen et al., 2018; Wallis et al., 2013). This could be the result of increased knee pain or, as suggested above, the sedentary lifestyle pattern, may have precipitated muscular changes which lead to the onset of knee OA. Whatever the reason, it would be pragmatically, very difficult to match

between the OA and healthy groups for physical activity patterns. Furthermore, to my best of my knowledge, no study in people with knee OA has matched healthy people and those with knee OA in the context of physical activity.

A final limitation of this thesis was that the impact of physical activity and/or trunk strength were not investigated in the context of upper body position and/or lower limb activity patterns. However, this is first study to investigate the impact of position of the trunk on knee muscle activation patterns in people with knee osteoarthritis. Therefore, it was necessary to maintain a focus on the most important factor and lay the ground for future work which could look at secondary factors. External moments, and therefore muscle activation patterns during gait, are more strongly influence by the position and direction ground reaction force vector than the maximal strength of the trunk muscles. Therefore, the decision to focus on trunk position rather than strength was made. Nevertheless, it is acknowledged that future work could investigate the impact of trunk muscle strength on muscle activation patterns.

9.6 Future work

The findings of this thesis raise several questions, which should be addressed in future studies, based on the need to improve upper body position during walking in people with knee OA. Firstly, further investigations are needed to assess the potential effectiveness of clinical programmes, such as virtual reality biofeedback training, to facilitate and improve trunk position during walking. In addition, trunk, abdomen and lower extremity muscle strengthening exercises and/or stretching exercises that could lead improved upper body posture during walking people with knee OA should be investigated. Most importantly, the finding presented in chapter 8 motivates future clinical research which should focus on stretching the hip flexor muscle in patients with knee OA to understand whether this could bring about long term changes in hip flexor muscle length and whether this would bring about a corresponding decrease in trunk inclination and reduction in co-contraction.

References

Aagaard, P., Simonsen, E., Andersen, J., Magnusson, S., Bojsen-Møller, F., & Dyhre-Poulsen, P. (2000). Antagonist muscle coactivation during isokinetic knee extension. *Scandinavian Journal of Medicine & Science in Sports, 10*(2), 58-67.

Al-Zahrani, K. S., & Bakheit, A. M. O. (2002). A study of the gait characteristics of patients with chronic osteoarthritis of the knee. *Disability and Rehabilitation, 24*(5), 275-280. doi:10.1080/09638280110087098

Allison, G. T. (2003). Trunk muscle onset detection technique for EMG signals with ECG artefact. *Journal of Electromyography and Kinesiology, 13*(3), 209-216. doi:10.1016/s1050-6411(03)00019-1

Alliston, T., Hernandez, C. J., Findlay, D. M., Felson, D. T., & Kennedy, O. D. (2018). Bone marrow lesions in osteoarthritis: what lies beneath. *Journal of Orthopaedic Research®, 36*(7), 1818-1825.

Alnahdi, A. H., Zeni, J. A., & Snyder-Mackler, L. (2012). Muscle impairments in patients with knee osteoarthritis. *Sports health, 4*(4), 284-292.

Altman, R., Asch, E., Bloch, D., Bole, G., Borenstein, D., Brandt, K., . . . Wolfe, F. (1986). DEVELOPMENT OF CRITERIA FOR THE CLASSIFICATION AND REPORTING OF OSTEOARTHRITIS - CLASSIFICATION OF OSTEOARTHRITIS OF THE KNEE. *Arthritis and Rheumatism, 29*(8), 1039-1049. doi:10.1002/art.1780290816

Andriacchi, T. P. (1994). Dynamics of knee malalignment. *The Orthopedic clinics of North America, 25*(3), 395-403.

Andriacchi, T. P., & Mundermann, A. (2006). The role of ambulatory mechanics in the initiation and progression of knee osteoarthritis. *Current opinion in rheumatology, 18*(5), 514-518. doi:10.1097/01.bor.0000240365.16842.4e

Andriacchi, T. P., Mundermann, A., Smith, R. L., Alexander, E. J., Dyrby, C. O., & Koo, S. (2004). A framework for the in vivo pathomechanics of osteoarthritis at the knee. *Annals of Biomedical Engineering, 32*(3), 447-457. doi:10.1023/b:abme.0000017541.82498.37

Anwer, S., Alghadir, A., Zafar, H., & Brismée, J.-M. (2018). Effects of orthopaedic manual therapy in knee osteoarthritis: a systematic review and meta-analysis. *Physiotherapy, 104*(3), 264-276. doi:10.1016/j.physio.2018.05.003

Arazpour, M., Bani, M. A., Maleki, M., Ghomshe, F. T., Kashani, R. V., & Hutchins, S. W. (2013). Comparison of the efficacy of laterally wedged insoles and bespoke unloader knee orthoses in treating medial compartment knee osteoarthritis. *Prosthetics and Orthotics International, 37*(1), 50-57. doi:10.1177/0309364612447094

Arden, N., & Nevitt, M. C. (2006a). Osteoarthritis: epidemiology. *Best practice & research Clinical rheumatology, 20*(1), 3-25.

Arden, N., & Nevitt, M. C. (2006b). Osteoarthritis: Epidemiology. *Best Practice & Research in Clinical Rheumatology, 20*(1), 3-25. doi:10.1016/j.berth.2005.09.007

Armand, S., Sangeux, M., & Baker, R. (2014). Optimal markers' placement on the thorax for clinical gait analysis. *Gait & Posture, 39*(1), 147-153.

Arsenault, A., Winter, D., Marteniuk, R., & Hayes, K. (1986). How many strides are required for the analysis of electromyographic data in gait? *Scandinavian Journal of Rehabilitation Medicine, 18*(3), 133-135.

Arthritis Care. (2017). Living with arthritis. Retrieved from https://www.arthritiscare.org.uk/living-with-arthritis

Arthritis Research UK. (2016, October 12, 2016). Retrieved from http://www.arthritisresearchuk.org/

Astephen, J. L., Deluzio, K. J., Caldwell, G. E., & Dunbar, M. J. (2008). Biomechanical changes at the hip, knee, and ankle joints during gait are associated with knee osteoarthritis severity. *Journal of Orthopaedic Research, 26*(3), 332-341. doi:10.1002/jor.20496

Astephen, J. L., Deluzio, K. J., Caldwell, G. E., Dunbar, M. J., & Hubley-Kozey, C. L. (2008). Gait and neuromuscular pattern changes are associated with differences in knee osteoarthritis severity levels. *J Biomech, 41*(4), 868-876. doi:10.1016/j.jbiomech.2007.10.016

Astephen Wilson, J. L., Stanish, W. D., & Hubley-Kozey, C. L. (2017). Asymptomatic and symptomatic individuals with the same radiographic evidence of knee osteoarthritis walk with different knee moments and muscle activity. *Journal of Orthopaedic Research, 35*(8), 1661-1670.

Attias, M., Chevalley, O., Bonnefoy-Mazure, A., De Coulon, G., Cheze, L., & Armand, S. (2016). Effects of contracture on gait kinematics: A systematic review. *Clinical Biomechanics, 33*, 103-110. doi:10.1016/j.clinbiomech.2016.02.017

Aujla, R. S., & Esler, C. N. (2017). Total knee arthroplasty for osteoarthritis in patients less than fifty-five years of age: a systematic review. *The Journal of arthroplasty, 32*(8), 2598-2603. e2591.

Axelson, H. W., & Hagbarth, K. E. (2001a). Human motor control consequences of thixotropic changes in muscular short-range stiffness. *Journal of Physiology-London, 535*(1), 279-288. doi:10.1111/j.1469-7793.2001.00279.x

Axelson, H. W., & Hagbarth, K. E. (2001b). Human motor control consequences of thixotropic changes in muscular short-range stiffness. *The Journal of Physiology, 535*(1), 279-288.

Bailey, R. A. (2005). Designing experiments and analyzing data: a model comparison perspective, 2nd edition. *Journal of the Royal Statistical Society Series a-Statistics in Society, 168*, 634-635. doi:10.1111/j.1467-985X.2005.00368_9.x

Baker, R. (2006). Gait analysis methods in rehabilitation. *Journal of Neuroengineering and Rehabilitation, 3*. doi:10.1186/1743-0003-3-4

Baliunas, A. J., Hurwitz, D. E., Ryals, A. B., Karrar, A., Case, J. P., Block, J. A., & Andriacchi, T. P. (2002). Increased knee joint loads during walking are present in subjects with knee osteoarthritis. *Osteoarthritis and Cartilage, 10*(7), 573-579. doi:10.1053/joca.2002.0797

Ball, N., & Scurr, J. (2010). An assessment of the reliability and standardisation of tests used to elicit reference muscular actions for electromyographical normalisation. *Journal of Electromyography and Kinesiology, 20*(1), 81-88.

Baratta, R., Solomonow, M., Zhou, B., Letson, D., Chuinard, R., & D'ambrosia, R. (1988). Muscular coactivation: the role of the antagonist musculature in maintaining knee stability. *The American journal of sports medicine, 16*(2), 113-122.

Barbour, K. E., Helmick, C. G., Boring, M., & Brady, T. J. (2017). Vital Signs: Prevalence of Doctor-Diagnosed Arthritis and Arthritis-Attributable Activity Limitation - United States, 2013-2015. *Mmwr-Morbidity and Mortality Weekly Report, 66*(9), 246-253.

Barrios, J. A., Butler, R. J., Crenshaw, J. R., Royer, T. D., & Davis, I. S. (2013). Mechanical effectiveness of lateral foot wedging in medial knee osteoarthritis after 1 year of wear. *Journal of Orthopaedic Research, 31*(5), 659-664. doi:10.1002/jor.22252

Bartel, D. (1991). Unicompartmental arthritis: biomechanics and treatment alternatives. *Instructional course lectures, 41*, 73-76.

Bartholdy, C., Juhl, C., Christensen, R., Lund, H., Zhang, W. Y., & Henriksen, M. (2017). The role of muscle strengthening in exercise therapy for knee osteoarthritis: A systematic review and meta-regression analysis of randomized trials. *Seminars in Arthritis and Rheumatism, 47*(1), 9-21. doi:10.1016/j.semarthrit.2017.03.007

Basmajian, J. V., & De Luca, C. J. (1985). *Muscles alive: their functions revealed by electromyography*: Williams & Wilkins.

Bates, D., Mächler, M., Bolker, B., & Walker, S. (2014). Fitting linear mixed-effects models using lme4. *arXiv preprint arXiv:1406.5823*.

Bauer, D. C., Hunter, D. J., Abramson, S. B., Attur, M., Corr, M., Felson, D., . . . Kraus, V. B. (2006). Classification of osteoarthritis biomarkers: a proposed approach. *Osteoarthritis and Cartilage, 14*(8), 723-727. doi:10.1016/j.joca.2006.04.001

Bellamy, N., Buchanan, W. W., Goldsmith, C. H., Campbell, J., & Stitt, L. W. (1988). Validation study of WOMAC: a health status instrument for measuring clinically important patient relevant outcomes to antirheumatic drug therapy in patients with osteoarthritis of the hip or knee. *The Journal of rheumatology, 15*(12), 1833-1840.

Bennell, K. L., Bowles, K. A., Wang, Y. Y., Cicuttini, F. M., Davies-Tuck, M., & Hinman, R. S. (2011). Higher dynamic medial knee load predicts greater cartilage loss over 12 months in medial knee osteoarthritis. *Annals of the Rheumatic Diseases, 70*(10), 1770-1774. doi:10.1136/ard.2010.147082

Bennell, K. L., Hall, M., & Hinman, R. S. (2016). Osteoarthritis year in review 2015: rehabilitation and outcomes. *Osteoarthritis and Cartilage, 24*(1), 58-70. doi:https://doi.org/10.1016/j.joca.2015.07.028

Bennell, K. L., & Hinman, R. S. (2011). A review of the clinical evidence for exercise in osteoarthritis of the hip and knee. *Journal of Science and Medicine in Sport, 14*(1), 4-9.

Benoit, D. L., Lamontagne, M., Cerulli, G., & Liti, A. (2003). The clinical significance of electromyography normalisation techniques in subjects with anterior cruciate ligament injury during treadmill walking. *Gait & Posture, 18*(2), 56-63. doi:10.1016/s0966-6362(02)00194-7

Bijlsma, J. W. J., & Knahr, K. (2007). Strategies for the prevention and management of osteoarthritis of the hip and knee. *Best Practice & Research in Clinical Rheumatology, 21*(1), 59-76. doi:10.1016/j.berh.2006.08.013

Birmingham, T. B., Hunt, M. A., Jones, I. C., Jenkyn, T. R., & Giffin, J. R. (2007). Test-retest reliability of the peak knee adduction moment during walking in patients with medial compartment knee Osteoarthritis. *Arthritis & Rheumatism-Arthritis Care & Research, 57*(6), 1012-1017. doi:10.1002/art.22899

Bombardier, C., Hawker, G., & Mosher, D. (2016). *The impact of arthritis in Canada: today and over the next 30 years*: Arthritis Alliance of Canada.

Boonen, A., & Severens, J. L. (2011). The burden of illness of rheumatoid arthritis. *Clinical Rheumatology, 30*, S3-S8. doi:10.1007/s10067-010-1634-9

Boren, K., Conrey, C., Le Coguic, J., Paprocki, L., Voight, M., & Robinson, T. K. (2011). Electromyographic analysis of gluteus medius and gluteus maximus during rehabilitation exercises. *International journal of sports physical therapy, 6*(3), 206.

Boyer, K. A., Johnson, R. T., Banks, J. J., Jewell, C., & Hafer, J. F. (2017). Systematic review and meta-analysis of gait mechanics in young and older adults. *Experimental Gerontology, 95*, 63-70. doi:10.1016/j.exger.2017.05.005

Brandon, S. C. E., Miller, R. H., Thelen, D. G., & Deluzio, K. J. (2014). Selective lateral muscle activation in moderate medial knee osteoarthritis subjects does not unload medial knee condyle. *Journal of Biomechanics, 47*(6), 1409-1415. doi:10.1016/j.jbiomech.2014.01.038

Brandt, K. D., Dieppe, P., & Radin, E. (2009). Etiopathogenesis of Osteoarthritis. *Medical Clinics of North America, 93*(1), 1-+. doi:10.1016/j.mcna.2008.08.009

Brandt, K. D., Dieppe, P., & Radin, E. L. (2008). Etiopathogenesis of osteoarthritis. *Rheumatic Disease Clinics of North America, 34*(3), 531-559.

Briem, K., Ramsey, D. K., Newcomb, W., Rudolph, K. S., & Snyder-Mackler, L. (2007). Effects of the amount of valgus correction for medial compartment knee osteoarthritis on clinical outcome, knee kinetics and muscle co-contraction after opening wedge high tibial osteotomy. *Journal of Orthopaedic Research, 25*(3), 311-318. doi:10.1002/jor.20326

Buckwalter, J. A., & Lohmander, S. (1994). OPERATIVE TREATMENT OF OSTEOARTHROSIS - CURRENT PRACTICE AND FUTURE-DEVELOPMENT. *Journal of Bone and Joint Surgery-American Volume, 76A*(9), 1405-1418.

Buckwalter, J. A., & Mankin, H. J. (1998). *Articular cartilage: Degeneration and osteoarthritis, repair, regeneration, and transplantation* (Vol. 47).

Buckwalter, J. A., Mankin, H. J., & Grodzinsky, A. J. (2005). Articular cartilage and osteoarthritis. *Instructional Course Lectures-American Academy of Orthopaedic Surgeons, 54*, 465.

Buckwalter, J. A., Saltzman, C., & Brown, T. (2004). The impact of osteoarthritis - Implications for research. *Clinical Orthopaedics and Related Research*(427), S6-S15. doi:10.1097/01.blo.0000143938.30681.9d

Burden, A. (2010). How should we normalize electromyograms obtained from healthy participants? What we have learned from over 25years of research. *Journal of Electromyography and Kinesiology, 20*(6), 1023-1035. doi:https://doi.org/10.1016/j.jelekin.2010.07.004

Burden, A. M., Trew, M., & Baltzopoulos, V. (2003). Normalisation of gait EMGs: a re-examination. *Journal of Electromyography and Kinesiology, 13*(6), 519-532. doi:10.1016/s1050-6411(03)00082-8

Burke, D., Hagbarth, K.-E., Löfstedt, L., & Wallin, B. G. (1976). The responses of human muscle spindle endings to vibration during isometric contraction. *The Journal of Physiology, 261*(3), 695-711.

Cappozzo, A., Catani, F., Leardini, A., Benedetti, M., & Della Croce, U. (1996). Position and orientation in space of bones during movement: experimental artefacts. *Clinical Biomechanics, 11*(2), 90-100.

Cappozzo, A., Della Croce, U., Leardini, A., & Chiari, L. (2005). Human movement analysis using stereophotogrammetry - Part 1: theoretical background. *Gait & Posture, 21*(2), 186-196. doi:10.1016/j.gaitpost.2004.01.010

Cejudo, A., de Baranda, P. S., Ayala, F., & Santonja, F. (2015). Test-retest reliability of seven common clinical tests for assessing lower extremity muscle flexibility in futsal and handball players. *Physical Therapy in Sport, 16*(2), 107-113. doi:10.1016/j.ptsp.2014.05.004

Cereatti, A., Camomilla, V., Vannozzi, G., & Cappozzo, A. (2007). Propagation of the hip joint centre location error to the estimate of femur vs pelvis orientation using a constrained or an unconstrained approach. *Journal of Biomechanics, 40*(6), 1228-1234.

Chan, G. N., Smith, A. W., Kirtley, C., & Tsang, W. W. (2005). Changes in knee moments with contralateral versus ipsilateral cane usage in females with knee osteoarthritis. *Clinical Biomechanics, 20*(4), 396-404.

Chang, A. H., Moisio, K. C., Chmiel, J. S., Eckstein, F., Guermazi, A., Prasad, P. V., . . . Sharma, L. (2015). External knee adduction and flexion moments during gait and medial tibiofemoral disease progression in knee osteoarthritis. *Osteoarthritis and Cartilage, 23*(7), 1099-1106. doi:10.1016/j.joca.2015.02.005

Chapman, G. J., Parkes, M. J., Forsythe, L., Felson, D., & Jones, R. (2015). Ankle motion influences the external knee adduction moment and may predict who will respond to lateral wedge

insoles?: an ancillary analysis from the SILK trial. *Osteoarthritis and Cartilage, 23*(8), 1316-1322.

Chehab, E. F., Favre, J., Erhart-Hledik, J. C., & Andriacchi, T. P. (2014). Baseline knee adduction and flexion moments during walking are both associated with 5 year cartilage changes in patients with medial knee osteoarthritis. *Osteoarthritis and Cartilage, 22*(11), 1833-1839. doi:10.1016/j.joca.2014.08.009

Chen, A., Gupte, C., Akhtar, K., Smith, P., & Cobb, J. (2012). The global economic cost of osteoarthritis: how the UK compares. *Arthritis, 2012*.

Chen, C. T., Burton-Wurster, N., Lust, G., Bank, R. A., & Tekoppele, J. M. (1999). Compositional and metabolic changes in damaged cartilage are peak-stress, stress-rate, and loading-duration dependent. *Journal of Orthopaedic Research, 17*(6), 870-879. doi:10.1002/jor.1100170612

Chesworth, B. M., Mahomed, N. N., Bourne, R. B., & Davis, A. M. (2008). Willingness to go through surgery again validated the WOMAC clinically important difference from THR/TKR surgery. *Journal of clinical epidemiology, 61*(9), 907-918.

Childs, J. D., Sparto, P. J., Fitzgerald, G. K., Bizzini, M., & Irrgang, J. J. (2004). Alterations in lower extremity movement and muscle activation patterns in individuals with knee osteoarthritis. *Clinical Biomechanics, 19*(1), 44-49. doi:10.1016/j.clinbiomech.2003.08.007

Clapis, P. A., Davis, S. M., & Davis, R. O. (2008). Reliability of inclinometer and goniometric measurements of hip extension flexibility using the modified Thomas test. *Physiotherapy Theory and Practice, 24*(2), 135-141. doi:10.1080/09593980701378256

Cohen, J. (1988). Statistical power analysis for the behavioral sciences 2nd edn: Erlbaum Associates, Hillsdale.

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*(1), 155.

Cole, G., Nigg, B., Ronsky, J., & Yeadon, M. (1993). Application of the joint coordinate system to three-dimensional joint attitude and movement representation: a standardization proposal. *Journal of biomechanical engineering, 115*(4A), 344-349.

Conaghan, P. G., Dickson, J., & Grant, R. L. (2008). Guidelines: care and management of osteoarthritis in adults: summary of NICE guidance. *BMJ: British Medical Journal, 336*(7642), 502.

Conaghan, P. G., Kloppenburg, M., Schett, G., Bijlsma, J. W. J., & Comm, E. O. A. H. (2014). Osteoarthritis research priorities: a report from a EULAR ad hoc expert committee. *Annals of the Rheumatic Diseases, 73*(8), 1442-1445. doi:10.1136/annrheumdis-2013-204660

Cooper, C., Snow, S., McAlindon, T. E., Kellingray, S., Stuart, B., Coggon, D., & Dieppe, P. A. (2000). Risk factors for the incidence and progression of radiographic knee osteoarthritis. *Arthritis and Rheumatism, 43*(5), 995-1000. doi:10.1002/1529-0131(200005)43:5<995::aid-anr6>3.0.co;2-1

Cram, J. R. (1998). *Introduction to surface electromyography*: Aspen Publishers.

Culliford, D. J., Maskell, J., Kiran, A., Judge, A., Javaid, M. K., Cooper, C., & Arden, N. K. (2012). The lifetime risk of total hip and knee arthroplasty: results from the UK general practice research database. *Osteoarthritis and Cartilage, 20*(6), 519-524. doi:10.1016/j.joca.2012.02.636

Czaprowski, D., Stoliński, Ł., Tyrakowski, M., Kozinoga, M., & Kotwicki, T. (2018). Non-structural misalignments of body posture in the sagittal plane. *Scoliosis and spinal disorders, 13*(1), 6.

da Silva, R. R., Santos, A. A. M., Júnior, J. d. S. C., & Matos, M. A. (2014). Quality of life after total knee arthroplasty: systematic review. *Revista Brasileira de Ortopedia (English Edition), 49*(5), 520-527.

Davis, R. B., Ounpuu, S., Tyburski, D., & Gage, J. R. (1991). A gait analysis data collection and reduction technique. *Human Movement Science, 10*(5), 575-587.

Davis, R. B., Õunpuu, S., Tyburski, D., & Gage, J. R. (1991). A gait analysis data collection and reduction technique. *Human Movement Science, 10*(5), 575-587. doi:https://doi.org/10.1016/0167-9457(91)90046-Z

Dearborn, J., Eakin, C., & Skinner, H. (1996). Medial compartment arthrosis of the knee. *American journal of orthopedics (Belle Mead, NJ), 25*(1), 18-26.

Delaney, S., Worsley, P., Warner, M., Taylor, M., & Stokes, M. (2010). ASSESSING CONTRACTILE ABILITY OF THE QUADRICEPS MUSCLE USING ULTRASOUND IMAGING. *Muscle & Nerve, 42*(4), 530-538. doi:10.1002/mus.21725

Dempster, W. T. (1955). *Space requirements of the seated operator, geometrical, kinematic, and mechanical aspects of the body with special reference to the limbs*. Retrieved from

Denegar, C. R., & Ball, D. W. (1993). Assessing reliability and precision of measurement: an introduction to intraclass correlation and standard error of measurement. *Journal of Sport Rehabilitation, 2*(1), 35-42.

Deshpande, B. R., Katz, J. N., Solomon, D. H., Yelin, E. H., Hunter, D. J., Messier, S. P., . . . Losina, E. (2016). Number of persons with symptomatic knee osteoarthritis in the US: impact of race and ethnicity, age, sex, and obesity. *Arthritis Care & Research, 68*(12), 1743-1750.

Deyle, G. D., Henderson, N. E., Matekel, R. L., Ryder, M. G., Garber, M. B., & Allison, S. C. (2000). Effectiveness of manual physical therapy and exercise in osteoarthritis of the knee. A randomized, controlled trial. *Annals of internal medicine, 132*(3), 173-181.

DiBonaventura, M. D., Gupta, S., McDonald, M., Sadosky, A., Pettitt, D., & Silverman, S. (2012). Impact of self-rated osteoarthritis severity in an employed population: Cross-sectional analysis of data from the national health and wellness survey. *Health and Quality of Life Outcomes, 10*. doi:10.1186/1477-7525-10-30

Dieppe, P. A., & Lohmander, L. S. (2005). Pathogenesis and management of pain in osteoarthritis. *The Lancet, 365*(9463), 965-973. doi:10.1016/s0140-6736(05)71086-2

Dixon, S. J., Hinman, R. S., Creaby, M. W., Kemp, G., & Crossley, K. M. (2010). Knee Joint Stiffness During Walking in Knee Osteoarthritis. *Arthritis Care & Research, 62*(1), 38-44. doi:10.1002/acr.20012

Doorenbosch, C. A., Harlaar, J., Roebroeck, M. E., & Lankhorst, G. J. (1994). Two strategies of transferring from sit-to-stand; the activation of monoarticular and biarticular muscles. *Journal of Biomechanics, 27*(11), 1299-1307.

Doorenbosch, C. A. M., & Harlaar, J. (2003). A clinically applicable EMG-force model to quantify active stabilization of the knee after a lesion of the anterior cruciate ligament. *Clinical Biomechanics, 18*(2), 142-149. doi:10.1016/s0268-0033(02)00183-3

Dougados, M., Gueguen, A., Nguyen, M., Thiesce, A., Listrat, V., Jacob, L., . . . Amor, B. (1992). LONGITUDINAL RADIOLOGIC EVALUATION OF OSTEOARTHRITIS OF THE KNEE. *Journal of Rheumatology, 19*(3), 378-384.

Duffell, L. D., Jordan, S. J., Cobb, J. P., & McGregor, A. H. (2017). Gait adaptations with aging in healthy participants and people with knee joint osteoarthritis. *Gait & Posture, 57*, 246-251. doi:10.1016/j.gaitpost.2017.06.015

Edd, S. N., Favre, J., Blazek, K., Omoumi, P., Asay, J. L., & Andriacchi, T. P. (2017). Altered gait mechanics and elevated serum pro-inflammatory cytokines in asymptomatic patients with

MRI evidence of knee cartilage loss. *Osteoarthritis and Cartilage, 25*(6), 899-906. doi:https://doi.org/10.1016/j.joca.2016.12.029

Erhart-Hledik, J. C., Favre, J., & Andriacchi, T. P. (2015). New insight in the relationship between regional patterns of knee cartilage thickness, osteoarthritis disease severity, and gait mechanics. *Journal of Biomechanics, 48*(14), 3868-3875. doi:10.1016/j.jbiomech.2015.09.033

Escobar, A., Quintana, J., Bilbao, A., Arostegui, I., Lafuente, I., & Vidaurreta, I. (2007). Responsiveness and clinically important differences for the WOMAC and SF-36 after total knee replacement. *Osteoarthritis and Cartilage, 15*(3), 273-280.

Farina, D., & Mesin, L. (2005). Sensitivity of surface EMG-based conduction velocity estimates to local tissue in-homogeneities - influence of the number of channels and inter-channel distance. *Journal of Neuroscience Methods, 142*(1), 83-89. doi:10.1016/j.jneumeth.2004.07.011

Felson, D. T. (1990). THE EPIDEMIOLOGY OF KNEE OSTEOARTHRITIS - RESULTS FROM THE FRAMINGHAM OSTEOARTHRITIS STUDY. *Seminars in Arthritis and Rheumatism, 20*(3), 42-50. doi:10.1016/0049-0172(90)90046-i

Felson, D. T. (2006). Osteoarthritis of the knee. *New England Journal of Medicine, 354*(8), 841-848.

Felson, D. T., Anderson, J. J., Naimark, A., Walker, A. M., & Meenan, R. F. (1988). Obesity and knee osteoarthritis: the Framingham Study. *Annals of internal medicine, 109*(1), 18-24.

Felson, D. T., Chaisson, C. E., Hill, C. L., Totterman, S. M., Gale, M. E., Skinner, K. M., . . . Gale, D. R. (2001). The association of bone marrow lesions with pain in knee osteoarthritis. *Annals of internal medicine, 134*(7), 541-549.

Felson, D. T., Lawrence, R. C., Dieppe, P. A., Hirsch, R., Helmick, C. G., Jordan, J. M., . . . Fries, J. F. (2000). Osteoarthritis: New Insights. Part 1: The Disease and Its Risk Factors. *Annals of internal medicine, 133*(8), 635-646. doi:10.7326/0003-4819-133-8-200010170-00016

Felson, D. T., McLaughlin, S., Goggins, J., LaValley, M. P., Gale, M. E., Totterman, S., . . . Gale, D. (2003). Bone marrow edema and its relation to progression of knee osteoarthritis. *Annals of internal medicine, 139*(5_Part_1), 330-336.

Felson, D. T., & Nevitt, M. C. (1998). The effects of estrogen on osteoarthritis. *Current opinion in rheumatology, 10*(3), 269-272. doi:10.1097/00002281-199805000-00019

Felson, D. T., Zhang, Y., Anthony, J. M., Naimark, A., & Anderson, J. J. (1992). Weight loss reduces the risk for symptomatic knee osteoarthritis in women: the Framingham Study. *Annals of internal medicine, 116*(7), 535-539.

Field, A. (2013). *Discovering statistics using IBM SPSS statistics*: sage.

Foley, S., Lord, S. R., Srikanth, V., Cooley, H., & Jones, G. (2006). Falls risk is associated with pain and dysfunction but not radiographic osteoarthritis in older adults: Tasmanian Older Adult Cohort study. *Osteoarthritis and Cartilage, 14*(6), 533-539.

Fransen, M., McConnell, S., Harmer, A. R., Van der Esch, M., Simic, M., & Bennell, K. L. (2015). Exercise for osteoarthritis of the knee. *Cochrane database of systematic reviews*(1).

Gandevia, S., McCloskey, D., & Burke, D. (1992). Kinaesthetic signals and muscle contraction. *Trends in neurosciences, 15*(2), 62-65.

Gok, H. (2003). Kinetic and kinematic characteristics of gait in patients with medial knee arthrosis. *Acta Orthopaedica Scandinavica, 74*(3), 369-370.

Gossman, M. R., Sahrmann, S. A., & Rose, S. J. (1982). Review of length-associated changes in muscle: experimental evidence and clinical implications. *Physical therapy, 62*(12), 1799-1808.

Grasso, R., Zago, M., & Lacquaniti, F. (2000). Interactions between posture and locomotion: Motor patterns in humans walking with bent posture versus erect posture. *Journal of Neurophysiology, 83*(1), 288-300.

Green, D., Noble, P., Ahuero, J., & Birdsall, H. (2006). Cellular events leading to chondrocyte death after cartilage impact injury. *Arthritis & Rheumatology, 54*(5), 1509-1517.

Guccione, A. A., Felson, D. T., Anderson, J. J., Anthony, J. M., Zhang, Y. Q., Wilson, P. W. F., . . . Kannel, W. B. (1994). THE EFFECTS OF SPECIFIC MEDICAL CONDITIONS ON THE FUNCTIONAL LIMITATIONS OF ELDERS IN THE FRAMINGHAM-STUDY. *American Journal of Public Health, 84*(3), 351-358. doi:10.2105/ajph.84.3.351

Gustafso, J. A., Anderton, W., Sowa, G. A., Piva, S. R., & Farrokhi, S. (2019). Dynamic knee joint stiffness and contralateral knee joint loading during prolonged walking in patients with unilateral knee osteoarthritis. *Gait & Posture, 68*, 44-49. doi:10.1016/j.gaitpost.2018.10.032

Gutierrez, E. M., Bartonek, Å., Haglund-Åkerlind, Y., & Saraste, H. (2003). Centre of mass motion during gait in persons with myelomeningocele. *Gait & Posture, 18*(2), 37-46.

Halaki, M., & Gi, K. (2012). Normalization of EMG Signals: To Normalize or Not to Normalize and What to Normalize to? doi:10.5772/49957

Hallett, M. (1993). Physiology of basal ganglia disorders: an overview. *Canadian Journal of Neurological Sciences, 20*(3), 177-183.

Hannan, M. T., Felson, D. T., & Pincus, T. (2000). Analysis of the discordance between radiographic changes and knee pain in osteoarthritis of the knee. *Journal of Rheumatology, 27*(6), 1513-1517.

HANSSON, G.-Å., STRÖMBERG, U., LARSSON, B., OHLSSON, K., BALOGH, I., & MORITZ, U. (1992). Electromyographic fatigue in neck/shoulder muscles and endurance in women with repetitive work. *Ergonomics, 35*(11), 1341-1352.

Hart, D. J., Doyle, D. V., & Spector, T. D. (1999). Incidence and risk factors for radiographic knee osteoarthritis in middle-aged women. *Arthritis Rheum, 42*(1), 17-24.

Harvey, D. (1998). Assessment of the flexibility of elite athletes using the modified Thomas test. *British Journal of Sports Medicine, 32*(1), 68-70.

Hassan, B. S., Mockett, S., & Doherty, M. (2001). Static postural sway, proprioception, and maximal voluntary quadriceps contraction in patients with knee osteoarthritis and normal control subjects. *Annals of the Rheumatic Diseases, 60*(6), 612-618. doi:10.1136/ard.60.6.612

Hatze, H. (1974). The meaning of the term 'biomechanics'. *Journal of Biomechanics, 7*(2), 189-190.

Hayes, C. W., Jamadar, D. A., Welch, G. W., Jannausch, M. L., Lachance, L. L., Capul, D. C., & Sowers, M. R. (2005). Osteoarthritis of the knee: comparison of MR imaging findings with radiographic severity measurements and pain in middle-aged women. *Radiology, 237*(3), 998-1007.

Heidari, B. (2011). Knee osteoarthritis prevalence, risk factors, pathogenesis and features: Part I. *Caspian journal of internal medicine, 2*(2), 205-212.

Heiden, T. L., Lloyd, D. G., & Ackland, T. R. (2009). Knee joint kinematics, kinetics and muscle co-contraction in knee osteoarthritis patient gait. *Clin Biomech (Bristol, Avon), 24*(10), 833-841. doi:10.1016/j.clinbiomech.2009.08.005

Hermens, H. J., Freriks, B., Disselhorst-Klug, C., & Rau, G. (2000). Development of recommendations for SEMG sensors and sensor placement procedures. *Journal of Electromyography and Kinesiology, 10*(5), 361-374. doi:10.1016/s1050-6411(00)00027-4

Hermens, H. J., Freriks, B., Merletti, R., Stegeman, D., Blok, J., Rau, G., . . . Hägg, G. (1999). European recommendations for surface electromyography. *Roessingh research and development, 8*(2), 13-54.

Hinman, R. S., Hunt, M. A., Simic, M., & Bennell, K. L. (2013). Exercise, gait retraining, footwear and insoles for knee osteoarthritis. *Current Physical Medicine and Rehabilitation Reports, 1*(1), 21-28.

Hirokawa, S., Solomonow, M., Luo, Z., Lu, Y., & D'ambrosia, R. (1991). Muscular co-contraction and control of knee stability. *Journal of Electromyography and Kinesiology, 1*(3), 199-208.

Hodges, P. W., van den Hoorn, W., Wrigley, T. V., Hinman, R. S., Bowles, K.-A., Cicuttini, F., . . . Bennell, K. (2016a). Increased duration of co-contraction of medial knee muscles is associated with greater progression of knee osteoarthritis. *Manual Therapy, 21*, 151-158. doi:10.1016/j.math.2015.07.004

Hodges, P. W., van den Hoorn, W., Wrigley, T. V., Hinman, R. S., Bowles, K. A., Cicuttini, F., . . . Bennell, K. (2016b). Increased duration of co-contraction of medial knee muscles is associated with greater progression of knee osteoarthritis. *Manual Therapy, 21*, 151-158. doi:10.1016/j.math.2015.07.004

Hoehn, K., & Marieb, E. N. (2007). *Human anatomy & physiology*: Benjamin Cummings.

Hori, N., Newton, R. U., Kawamori, N., McGuigan, M. R., Kraemer, W. J., & Nosaka, K. (2009). RELIABILITY OF PERFORMANCE MEASUREMENTS DERIVED FROM GROUND REACTION FORCE DATA DURING COUNTERMOVEMENT JUMP AND THE INFLUENCE OF SAMPLING FREQUENCY. *Journal of Strength and Conditioning Research, 23*(3), 874-882. doi:10.1519/JSC.0b013e3181a00ca2

Hortobagyi, T., Westerkamp, L., Beam, S., Moody, J., Garry, J., Holbert, D., & DeVita, P. (2005). Altered hamstring-quadriceps muscle balance in patients with knee osteoarthritis. *Clin Biomech (Bristol, Avon), 20*(1), 97-104. doi:10.1016/j.clinbiomech.2004.08.004

Houston, M. E., Norman, R. W., & Froese, E. A. (1988). Mechanical measures during maximal velocity knee extension exercise and their relation to fibre composition of the human vastus lateralis muscle. *European Journal of Applied Physiology and Occupational Physiology, 58*(1-2), 1-7.

Hsu, A.-L., Tang, P.-F., & Jan, M.-H. (2002). Test-retest reliability of isokinetic muscle strength of the lower extremities in patients with stroke. *Archives of Physical Medicine and Rehabilitation, 83*(8), 1130-1137.

Huang, S.-C., Wei, I. P., Chien, H.-L., Wang, T.-M., Liu, Y.-H., Chen, H.-L., . . . Lin, J.-G. (2008). Effects of severity of degeneration on gait patterns in patients with medial knee osteoarthritis. *Medical Engineering & Physics, 30*(8), 997-1003. doi:10.1016/j.medengphy.2008.02.006

Hubley-Kozey, C. L., Deluzio, K. J., Landry, S. C., McNutt, J. S., & Stanish, W. D. (2006). Neuromuscular alterations during walking in persons with moderate knee osteoarthritis. *Journal of Electromyography and Kinesiology, 16*(4), 365-378. doi:10.1016/j.jelekin.2005.07.014

Hubley-Kozey, C. L., Hatfield, G., & Stanish, W. D. (2013). Muscle activation differences during walking between those with moderate knee osteoarthritis who progress to total knee arthroplasty and those that do not: a follow up study. *Osteoarthritis and Cartilage, 21*, S38-S38.

Hubley-Kozey, C. L., Hatfield, G. L., Wilson, J. L. A., & Dunbar, M. J. (2010). Alterations in neuromuscular patterns between pre and one-year post-total knee arthroplasty. *Clinical Biomechanics, 25*(10), 995-1002. doi:10.1016/j.clinbiomech.2010.07.008

Hubley-Kozey, C. L., Hill, N. A., Rutherford, D. J., Dunbar, M. J., & Stanish, W. D. (2009a). Co-activation differences in lower limb muscles between asymptomatic controls and those with varying degrees of knee osteoarthritis during walking. *Clinical Biomechanics, 24*(5), 407-414. doi:10.1016/j.clinbiomech.2009.02.005

Hubley-Kozey, C. L., Hill, N. A., Rutherford, D. J., Dunbar, M. J., & Stanish, W. D. (2009b). Co-activation differences in lower limb muscles between asymptomatic controls and those with varying degrees of knee osteoarthritis during walking (vol 24, pg 407, 2009). *Clinical Biomechanics, 24*(6), 529-529. doi:10.1016/j.clinbiomech.2009.04.010

Hubley-Kozey, C. L., Robbins, S. M., Rutherford, D. J., & Stanish, W. D. (2013). Reliability of surface electromyographic recordings during walking in individuals with knee osteoarthritis. *Journal of Electromyography and Kinesiology, 23*(2), 334-341. doi:10.1016/j.jelekin.2012.12.002

Huch, K., Kuettner, K. E., & Dieppe, P. (1997). Osteoarthritis in ankle and knee joints. *Seminars in Arthritis and Rheumatism, 26*(4), 667-674. doi:10.1016/s0049-0172(97)80002-9

Hughes, A. J., Daniel, S. E., Kilford, L., & Lees, A. J. (1992). Accuracy of clinical diagnosis of idiopathic Parkinson's disease: a clinico-pathological study of 100 cases. *Journal of Neurology, Neurosurgery & Psychiatry, 55*(3), 181-184.

Hunter, D. J., McDougall, J. J., & Keefe, F. J. (2008). The symptoms of osteoarthritis and the genesis of pain. *Rheumatic diseases clinics of North America, 34*(3), 623-643. doi:10.1016/j.rdc.2008.05.004

Hunter, D. J., Schofield, D., & Callander, E. (2014). The individual and socioeconomic impact of osteoarthritis. *Nature Reviews Rheumatology, 10*(7), 437-441. doi:10.1038/nrrheum.2014.44

Hurley, M. V., Scott, D. L., Rees, J., & Newham, D. J. (1997). Sensorimotor changes and functional performance in patients with knee osteoarthritis. *Annals of the Rheumatic Diseases, 56*(11), 641-648. doi:10.1136/ard.56.11.641

Ishihara, M., Ohmiya, N., Nakamura, M., Funasaka, K., Miyahara, R., Ohno, E., . . . Goto, H. (2014). Risk factors of symptomatic NSAID-induced small intestinal injury and diaphragm disease. *Alimentary Pharmacology & Therapeutics, 40*(5), 538-547. doi:10.1111/apt.12858

Jacobson, W. C., Gabel, R. H., & Brand, R. A. (1995). Surface vs. fine-wire electrode ensemble-averaged signals during gait. *Journal of Electromyography and Kinesiology, 5*(1), 37-44.

Jansen, M. J., Viechtbauer, W., Lenssen, A. F., Hendriks, E. J. M., & de Bie, R. A. (2011). Strength training alone, exercise therapy alone, and exercise therapy with passive manual mobilisation each reduce pain and disability in people with knee osteoarthritis: a systematic review. *Journal of Physiotherapy, 57*(1), 11-20.

Jensen, C., Vasseljen, O., & Westgaard, R. H. (1993). The influence of electrode position on bipolar surface electromyogram recordings of the upper trapezius muscle. *European Journal of Applied Physiology and Occupational Physiology, 67*(3), 266-273.

Jinks, C., Jordan, K., & Croft, P. (2002). Measuring the population impact of knee pain and disability with the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC). *Pain, 100*(1-2), 55-64.

Johnson, F., Leitl, S., & Waugh, W. (1980). The distribution of load across the knee. A comparison of static and dynamic measurements. *Bone & Joint Journal, 62*(3), 346-349.

Johnson, V. L., & Hunter, D. J. (2014). The epidemiology of osteoarthritis. *Best Practice & Research in Clinical Rheumatology, 28*(1), 5-15. doi:10.1016/j.berh.2014.01.004

Jordan, J. M., Helmick, C. G., Renner, J. B., Luta, G., Dragomir, A. D., Woodard, J., . . . Hochberg, M. C. (2007). Prevalence of knee symptoms and radiographic and symptomatic knee osteoarthritis in African Americans and Caucasians: The Johnston County Osteoarthritis project. *Journal of Rheumatology, 34*(1), 172-180.

Jordan, K. M., Arden, N. K., Doherty, M., Bannwarth, B., Bijlsma, J. W. J., Dieppe, P., . . . Dougados, M. (2003). EULAR Recommendations 2003: an evidence based approach to the management of knee osteoarthritis: Report of a Task Force of the Standing Committee for International Clinical Studies Including Therapeutic Trials (ESCISIT). *Annals of the Rheumatic Diseases, 62*(12), 1145-1155. doi:10.1136/ard.2003.011742

Kadaba, M. P., Ramakrishnan, H. K., & Wootten, M. E. (1990). MEASUREMENT OF LOWER-EXTREMITY KINEMATICS DURING LEVEL WALKING. *Journal of Orthopaedic Research, 8*(3), 383-392. doi:10.1002/jor.1100080310

Kagaya, H., Ito, S., Iwami, T., Obinata, G., & Shimada, Y. (2003). A computer simulation of human walking in persons with joint contractures. *Tohoku Journal of Experimental Medicine, 200*(1), 31-37. doi:10.1620/tjem.200.31

Kaufman, K. R., Hughes, C., Morrey, B. F., Morrey, M., & An, K. N. (2001). Gait characteristics of patients with knee osteoarthritis. *Journal of Biomechanics, 34*(7), 907-915. doi:10.1016/s0021-9290(01)00036-7

Kaufman, K. R., & Sutherland, D. H. (2006). Kinematics of normal human walking. *Human walking, 3*, 33-52.

Kellgren, J. H., & Lawrence, J. S. (1957). RADIOLOGICAL ASSESSMENT OF OSTEO-ARTHROSIS. *Annals of the Rheumatic Diseases, 16*(4), 494-502. doi:10.1136/ard.16.4.494

Kendall, H. O., Kendall, F. P., & Wadsworth, G. E. (1973). Muscles, Testing and function. *American Journal of Physical Medicine & Rehabilitation, 52*(1), 43.

Khan, M., Evaniew, N., Bedi, A., Ayeni, O. R., & Bhandari, M. (2014). Arthroscopic surgery for degenerative tears of the meniscus: a systematic review and meta-analysis. *Canadian Medical Association Journal, 186*(14), 1057-1064.

Kidd, B. (2012). Mechanisms of pain in osteoarthritis. *HSS journal, 8*(1), 26-28.

Kidd, B. L. (2006). Osteoarthritis and joint pain. *Pain, 123*(1-2), 6-9. doi:10.1016/j.pain.2006.04.009

Kim, G. M., & Ha, S. M. (2015). Reliability of the modified Thomas test using a lumbo-plevic stabilization. *Journal of physical therapy science, 27*(2), 447-449.

Kim, S. (2008). Changes in surgical loads and economic burden of hip and knee replacements in the US: 1997–2004. *Arthritis Care & Research, 59*(4), 481-488.

Kim, W. Y., Richards, J., Jones, R. K., & Hegab, A. (2004). A new biomechanical model for the functional assessment of knee osteoarthritis. *Knee, 11*(3), 225-231. doi:10.1016/s0968-0160(03)00068-1

Kirk, R. E. (2013). Experimental Design: Procedures for the Behavioral Sciences. 1995. *Brooks/Cole, Pacific Grove, CA*.

Kleissen, R. F. M., Buurke, J. H., Harlaar, J., & Zilvold, G. (1998). Electromyography in the biomechanical analysis of human movement and its clinical application. *Gait & Posture, 8*(2), 143-158. doi:10.1016/s0966-6362(98)00025-3

Kluger, D., Major, M. J., Fatone, S., & Gard, S. A. (2014). The effect of trunk flexion on lower-limb kinetics of able-bodied gait. *Human Movement Science, 33*, 395-403. doi:10.1016/j.humov.2013.12.006

Knutson, G. A., & Owens Jr, E. F. (2003). Active and passive characteristics of muscle tone and their relationship to models of subluxation/joint dysfunction: Part I. *the Journal of the canadian chiropractic Association, 47*(3), 168.

Kolber, M. J., & Fiebert, I. M. (2005). Addressing flexibility of the rectus femoris in the athlete with low back pain. *Strength and Conditioning Journal, 27*(5), 66-73. doi:10.1519/00126548-200510000-00012

Konrad, A., Gad, M., & Tilp, M. (2015). Effect of PNF stretching training on the properties of human muscle and tendon structures. *Scandinavian Journal of Medicine & Science in Sports, 25*(3), 346-355.

Konrad, P. (2005). The abc of emg. *A practical introduction to kinesiological electromyography, 1*, 30-35.

Koo, T. K., & Li, M. Y. (2016). A guideline of selecting and reporting intraclass correlation coefficients for reliability research. *Journal of chiropractic medicine, 15*(2), 155-163.

Krol, A., Polak, M., Szczygiel, E., Wojcik, P., & Gleb, K. (2017). Relationship between mechanical factors and pelvic tilt in adults with and without low back pain. *Journal of Back and Musculoskeletal Rehabilitation, 30*(4), 699-705. doi:10.3233/bmr-140177

Kyllerman, M. (1982). Dyskinetic cerebral palsy. *Acta Paediatrica, 71*(4), 551-558.

Landry, S. C., McKean, K. A., Hubley-Kozey, C. L., Stanish, W. D., & Deluzio, K. J. (2007). Knee biomechanics of moderate OA patients measured during gait at a self-selected and fast walking speed. *Journal of Biomechanics, 40*(8), 1754-1761. doi:10.1016/j.jbiomech.2006.08.010

Lawrence, R. C., Felson, D. T., Helmick, C. G., Arnold, L. M., Choi, H., Deyo, R. A., . . . Hunder, G. G. (2008). Estimates of the prevalence of arthritis and other rheumatic conditions in the United States: Part II. *Arthritis & Rheumatism, 58*(1), 26-35.

Leardini, G., Salaffi, F., Caporali, R., Canesi, B., Rovati, L., Montanelli, R., & Italian Grp Study Costs, A. (2004). Direct and indirect costs of osteoarthritis of the knee. *Clinical and Experimental Rheumatology, 22*(6), 699-706.

Ledingham, J., Regan, M., Jones, A., & Doherty, M. (1993). RADIOGRAPHIC PATTERNS AND ASSOCIATIONS OF OSTEOARTHRITIS OF THE KNEE IN PATIENTS REFERRED TO HOSPITAL. *Annals of the Rheumatic Diseases, 52*(7), 520-526. doi:10.1136/ard.52.7.520

Lee, J., Chang, R. W., Ehrlich-Jones, L., Kwoh, C. K., Nevitt, M., Semanik, P. A., . . . Dunlop, D. D. (2015). Sedentary Behavior and Physical Function: Objective Evidence From the Osteoarthritis Initiative. *Arthritis Care & Research, 67*(3), 366-373. doi:10.1002/acr.22432

Lee, J., & Schmid-Schönbein, G. W. (1995). Biomechanics of skeletal muscle capillaries: hemodynamic resistance, endothelial distensibility, and pseudopod formation. *Annals of Biomedical Engineering, 23*(3), 226-246.

Lehman, G. J., & McGill, S. M. (1999). The importance of normalization in the interpretation of surface electromyography: a proof of principle. *Journal of Manipulative & Physiological Therapeutics, 22*(7), 444-446.

Leteneur, S., Gillet, C., Sadeghi, H., Allard, P., & Barbier, F. (2009). Effect of trunk inclination on lower limb joint and lumbar moments in able men during the stance phase of gait. *Clin Biomech (Bristol, Avon), 24*(2), 190-195. doi:10.1016/j.clinbiomech.2008.10.005

Levine, D., Richards, J., Whittle, M., & Whittle, M. (2012). *Whittle's gait analysis*. Edinburgh: Churchill Livingstone Elsevier.

Lewek, M. D., Ramsey, D. K., Snyder-Mackler, L., & Rudolph, K. S. (2005). Knee stabilization in patients with medial compartment knee osteoarthritis. *Arthritis Rheum, 52*(9), 2845-2853. doi:10.1002/art.21237

Lewek, M. D., Rudolph, K. S., & Snyder-Mackler, L. (2004a). Control of frontal plane knee laxity during gait in patients with medial compartment knee osteoarthritis. *Osteoarthritis and Cartilage, 12*(9), 745-751. doi:10.1016/j.joca.2004.05.005

Lewek, M. D., Rudolph, K. S., & Snyder-Mackler, L. (2004b). Quadriceps femoris muscle weakness and activation failure in patients with symptomatic knee osteoarthritis. *Journal of Orthopaedic Research, 22*(1), 110-115. doi:10.1016/s0736-0266(03)00154-2

Lewek, M. D., Scholz, J., Rudolph, K. S., & Snyder-Mackler, L. (2006). Stride-to-stride variability of knee motion in patients with knee osteoarthritis. *Gait & Posture, 23*(4), 505-511. doi:10.1016/j.gaitpost.2005.06.003

Lewis, C. L., & Sahrmann, S. A. (2015). Effect of posture on hip angles and moments during gait. *Man Ther, 20*(1), 176-182. doi:10.1016/j.math.2014.08.007

Litwic, A., Edwards, M. H., Dennison, E. M., & Cooper, C. (2013). Epidemiology and burden of osteoarthritis. *British Medical Bulletin, 105*(1), 185-199. doi:10.1093/bmb/lds038

Liu, Y. H., Wang, T. M., Wei, I. P., Lu, T. W., Hong, S. W., & Kuo, C. C. (2014). Effects of bilateral medial knee osteoarthritis on intra- and inter-limb contributions to body support during gait. *Journal of Biomechanics, 47*(2), 445-450. doi:10.1016/j.jbiomech.2013.11.001

Lopez-Olivo, M. A., Ingleshwar, A., Volk, R. J., Jibaja-Weiss, M., Barbo, A., Saag, K., . . . Suarez-Almazor, M. E. (2018). Development and pilot testing of multimedia patient education tools for patients with knee osteoarthritis, osteoporosis, and rheumatoid arthritis. *Arthritis Care & Research, 70*(2), 213-220.

Madden, E. G., Kean, C. O., Wrigley, T. V., Bennell, K. L., & Hinman, R. S. (2015). Effect of Rocker-Soled Shoes on Parameters of Knee Joint Load in Knee Osteoarthritis. *Medicine and Science in Sports and Exercise, 47*(1), 128-135. doi:10.1249/mss.0000000000000384

Madden, E. G., Kean, C. O., Wrigley, T. V., Bennell, K. L., & Hinman, R. S. (2017). How do rocker-soled shoes influence the knee adduction moment in people with knee osteoarthritis? An analysis of biomechanical mechanisms. *Journal of Biomechanics, 57*, 62-68. doi:10.1016/j.jbiomech.2017.03.030

Manal, K., Gardinier, E., Buchanan, T. S., & Snyder-Mackler, L. (2015). A more informed evaluation of medial compartment loading: the combined use of the knee adduction and flexor moments. *Osteoarthritis and Cartilage, 23*(7), 1107-1111. doi:10.1016/j.joca.2015.02.779

March, L. M., & Bachmeier, C. J. M. (1997). Economics of osteoarthritis: a global perspective. *Baillieres Clinical Rheumatology, 11*(4), 817-834. doi:10.1016/s0950-3579(97)80011-8

Mascarin, N. C., Vancini, R. L., dos Santos Andrade, M., de Paiva Magalhães, E., de Lira, C. A. B., & Coimbra, I. B. (2012). Effects of kinesiotherapy, ultrasound and electrotherapy in management of bilateral knee osteoarthritis: prospective clinical trial. *Bmc Musculoskeletal Disorders, 13*(1), 182.

Maxwell, S. E. (1980). Pairwise multiple comparisons in repeated measures designs. *Journal of Educational Statistics, 5*(3), 269-287.

Mazzuca, S. A., Brandt, K. D., Katz, B. P., Ding, Y., Lane, K. A., & Buckwalter, K. A. (2007). Risk factors for early radiographic changes of tibiofemoral osteoarthritis. *Annals of the Rheumatic Diseases, 66*(3), 394-399. doi:10.1136/ard.2006.055905

McAlindon, T. E., Felson, D. T., Zhang, Y., Hannan, M. T., Aliabadi, P., Weissman, B., . . . Jacques, P. (1996). Relation of dietary intake and serum levels of vitamin D to progression of osteoarthritis of the knee among participants in the Framingham Study. *Annals of internal medicine, 125*(5), 353-359.

McGill, S. M. (1991). Electromyographic activity of the abdominal and low back musculature during the generation of isometric and dynamic axial trunk torque: implications for lumbar mechanics. *Journal of Orthopaedic Research, 9*(1), 91-103.

McNair, P. J., Depledge, J., Brettkelly, M., & Stanley, S. N. (1996). Verbal encouragement: Effects on maximum effort voluntary muscle action. *British Journal of Sports Medicine, 30*(3), 243-245. doi:10.1136/bjsm.30.3.243

Messier, S. P., DeVita, P., Cowan, R. E., Seay, J., Young, H. C., & Marsh, A. P. (2005). Do older adults with knee osteoarthritis place greater loads on the knee during gait? A preliminary study. *Archives of Physical Medicine and Rehabilitation, 86*(4), 703-709.

Metcalfe, A., Stewart, C., Postans, N., Dodds, A., Holt, C. A., & Roberts, A. (2013). The effect of osteoarthritis of the knee on the biomechanics of other joints in the lower limbs. *The bone & joint journal, 95*(3), 348-353.

Metcalfe, A. J., Andersson, M. L., Goodfellow, R., & Thorstensson, C. A. (2012). Is knee osteoarthritis a symmetrical disease? Analysis of a 12 year prospective cohort study. *Bmc Musculoskeletal Disorders, 13*(1), 153.

Meyer, A. J., D'Lima, D. D., Besier, T. F., Lloyd, D. G., Colwell, C. W., Jr., & Fregly, B. J. (2013). Are external knee load and EMG measures accurate indicators of internal knee contact forces during gait? *J Orthop Res, 31*(6), 921-929. doi:10.1002/jor.22304

Mezghani, N., Billard, D., Ouakrim, Y., Fuentes, A., Hagemeister, N., & De Guise, J. A. (2017). Biomechanical analysis to characterize the impact of knee osteoarthritis on hip, knee, and ankle kinematics. *Journal of Biomedical Engineering and Informatics, 3*(2), 36.

Michael, J. W. P., Schluter-Brust, K. U., & Eysel, P. (2010). The Epidemiology, Etiology, Diagnosis, and Treatment of Osteoarthritis of the Knee. *Deutsches Arzteblatt International, 107*(9), 152-U139. doi:10.3238/arztebl.2010.0152

Mileusnic, M. P., Brown, I. E., Lan, N., & Loeb, G. E. (2006). Mathematical models of proprioceptors. I. Control and transduction in the muscle spindle. *Journal of Neurophysiology, 96*(4), 1772-1788.

Mills, K., Hunt, M. A., Leigh, R., & Ferber, R. (2013). A systematic review and meta-analysis of lower limb neuromuscular alterations associated with knee osteoarthritis during level walking. *Clinical Biomechanics, 28*(7), 713-724. doi:10.1016/j.clinbiomech.2013.07.008

Mills, M., Frank, B., Goto, S., Blackburn, T., Cates, S., Clark, M., . . . Padua, D. (2015). EFFECT OF RESTRICTED HIP FLEXOR MUSCLE LENGTH ON HIP EXTENSOR MUSCLE ACTIVITY AND LOWER EXTREMITY BIOMECHANICS IN COLLEGE-AGED FEMALE SOCCER PLAYERS. *International journal of sports physical therapy, 10*(7), 946-954.

Mink, J. W. (1996). The basal ganglia: focused selection and inhibition of competing motor programs. *Progress in neurobiology, 50*(4), 381-425.

Miyazaki, T., Wada, M., Kawahara, H., Sato, M., Baba, H., & Shimada, S. (2002). Dynamic load at baseline can predict radiographic disease progression in medial compartment knee osteoarthritis. *Annals of the Rheumatic Diseases, 61*(7), 617-622. doi:10.1136/ard.61.7.617

Moon, K. W., Kim, J., Kim, J. H., Song, R., Lee, E. Y., Song, Y. W., & Lee, E. B. (2011). Risk factors for acute kidney injury by non-steroidal anti-inflammatory drugs in patients with hyperuricaemia. *Rheumatology, 50*(12), 2278-2282. doi:10.1093/rheumatology/ker286

Moreside, J. M., & McGill, S. M. (2011). Quantifying normal 3D hip ROM in healthy young adult males with clinical and laboratory tools: Hip mobility restrictions appear to be plane-specific. *Clinical Biomechanics, 26*(8), 824-829. doi:10.1016/j.clinbiomech.2011.03.015

Morrison, J. (1970). The mechanics of the knee joint in relation to normal walking. *Journal of Biomechanics, 3*(1), 51-61.

Mow, V. C., Holmes, M. H., & Lai, W. M. (1984). FLUID TRANSPORT AND MECHANICAL-PROPERTIES OF ARTICULAR-CARTILAGE - A REVIEW. *Journal of Biomechanics, 17*(5), 377-394. doi:10.1016/0021-9290(84)90031-9

Moyer, R. F., Birmingham, T. B., Bryant, D. M., Giffin, J. R., Marriott, K. A., & Leitch, K. M. (2015). Biomechanical effects of valgus knee bracing: a systematic review and meta-analysis. *Osteoarthritis and Cartilage, 23*(2), 178-188. doi:10.1016/j.joca.2014.11.018

Mundermann, A., Dyrby, C. O., & Andriacchi, T. P. (2005). Secondary gait changes in patients with medial compartment knee osteoarthritis - Increased load at the ankle, knee, and hip during walking. *Arthritis and Rheumatism, 52*(9), 2835-2844. doi:10.1002/art.21262

Mundermann, A., Dyrby, C. O., Hurwitz, D. E., Sharma, L., & Andriacchi, T. P. (2004a). Potential strategies to reduce medial compartment loading in patients with knee osteoarthritis of varying severity - Reduced walking speed. *Arthritis and Rheumatism, 50*(4), 1172-1178. doi:10.1002/art.20132

Mundermann, A., Dyrby, C. O., Hurwitz, D. E., Sharma, L., & Andriacchi, T. P. (2004b). Potential strategies to reduce medial compartment loading in patients with knee osteoarthritis of varying severity - Reduced walking speed (vol 50, pg 1172, 2004). *Arthritis and Rheumatism, 50*(12), 4073-4073. doi:10.1002/art.588

Murray, C. J., Vos, T., Lozano, R., Naghavi, M., Flaxman, A. D., Michaud, C., . . . Memish, Z. A. (2012). Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet, 380*(9859), 2197-2223. doi:10.1016/S0140-6736(12)61689-4

Murray, M. P., Drought, A. B., & Kory, R. C. (1964). WALKING PATTERNS OF NORMAL MEN. *Journal of Bone and Joint Surgery-American Volume, 46*(2), 335-360.

Mutch, L., Alberman, E., Hagberg, B., Kodama, K., & Perat, M. V. (1992). Cerebral palsy epidemiology: where are we now and where are we going? *Developmental Medicine & Child Neurology, 34*(6), 547-551.

Nelson, A., Renner, J., Golightly, Y., Schwartz, T., Kraus, V., Helmick, C., & Jordan, J. (2012). Differences in multi-joint symptomatic osteoarthritis phenotypes by race and gender: the Johnston County osteoarthritis project. *Osteoarthritis and Cartilage, 20*, S156.

Nelson, A. E., Braga, L., Renner, J. B., Atashili, J., Woodard, J., Hochberg, M. C., . . . Jordan, J. M. (2010). Characterization of individual radiographic features of hip osteoarthritis in African American and White women and men: the Johnston County Osteoarthritis Project. *Arthritis Care & Research, 62*(2), 190-197.

Neogi, T., & Zhang, Y. (2013). Epidemiology of OA. *Rheumatic diseases clinics of North America, 39*(1), 1.

Nguyen, T. C., & Baker, R. (2004). Two methods of calculating thorax kinematics in children with myelomeningocele. *Clinical Biomechanics, 19*(10), 1060-1065.

Nordesjö, L.-O., Nordgren, B., Wigren, A., & Kolstad, K. (1983). Isometric strength and endurance in patients with severe rheumatoid arthritis or osteoarthrosis in the knee joints. *Scandinavian Journal of Rheumatology, 12*(2), 152-156.

Nordin, M., & Frankel, V. H. (2001). *Basic biomechanics of the musculoskeletal system*: Lippincott Williams & Wilkins.

Nyland, J., Kuzemchek, S., Parks, M., & Caborn, D. N. M. (2004). Femoral anteversion influences vastus medialis and gluteus medius EMG amplitude: composite hip abductor EMG amplitude ratios during isometric combined hip abduction-external rotation. *Journal of Electromyography and Kinesiology, 14*(2), 255-261. doi:10.1016/s1050-6411(03)00078-6

O'Sullivan, S. B., Schmitz, T. J., & Fulk, G. (2013). *Physical rehabilitation*: FA Davis.

Obeso, J. A., Rodriguez-Oroz, M. C., Rodriguez, M., Lanciego, J. L., Artieda, J., Gonzalo, N., & Olanow, C. W. (2000). Pathophysiology of the basal ganglia in Parkinson's disease. *Trends in neurosciences, 23*, S8-S19.

Pandy, M. G., & Andriacchi, T. P. (2010). Muscle and Joint Function in Human Locomotion. In M. L. Yarmush, J. S. Duncan, & M. L. Gray (Eds.), *Annual Review of Biomedical Engineering, Vol 12* (Vol. 12, pp. 401-433).

Payton, C. J., & Burden, A. (2017). *Biomechanical evaluation of movement in sport and exercise: the British Association of Sport and Exercise Sciences guide*: Routledge.

Peat, G., McCarney, R., & Croft, P. (2001). Knee pain and osteoarthritis in older adults: a review of community burden and current use of primary health care. *Annals of the Rheumatic Diseases, 60*(2), 91-97. doi:10.1136/ard.60.2.91

Peeler, J. D., & Anderson, J. E. (2008). Reliability limits of the modified Thomas test for assessing rectus femoris muscle flexibility about the knee joint. *Journal of Athletic Training, 43*(5), 470-476. doi:10.4085/1062-6050-43.5.470

Perry, J. (1998). The contribution of dynamic electromyography to gait analysis. *Journal of Rehabilitation Research and Development, 33*.

Perry, J. (2010). Gait analysis : normal and pathological function. In J. M. Burnfield & L. M. Cabico (Eds.), (2nd ed. / Jacquelin Perry, Judith M. Burnfield ; illustrated by Lydia M. Cabico. ed.). Thorofare, N.J.: Thorofare, N.J. : Slack.

Perry, J., & Davids, J. R. (1992). Gait analysis: normal and pathological function. *Journal of Pediatric Orthopaedics, 12*(6), 815.

Peter, W. F. H., Jansen, M. J., Hurkmans, E. J., Bloo, H., Dekker-Bakker, L. M. M. C. J., Dilling, R. G., . . . Vlieland, T. P. M. V. (2011). PHYSIOTHERAPY IN HIP AND KNEE OSTEOARTHRITIS: DEVELOPMENT OF A PRACTICE GUIDELINE CONCERNING INITIAL ASSESSMENT. TREATMENT AND EVALUATION. *Acta Reumatologica Portuguesa, 36*(3), 268-281.

Pottie, P., Presle, N., Terlain, B., Netter, P., Mainard, D., & Berenbaum, F. (2006). Obesity and osteoarthritis: more complex than predicted! : BMJ Publishing Group Ltd.

Preece, S. J., Algarni, A., & Jones, R. K. (2018). Trunk flexion during walking in people with knee osteoarthritis. *Gait Posture.*(Under review).

Preece, S. J., Jones, R. K., Brown, C. A., Cacciatore, T. W., & Jones, A. K. (2016). Reductions in co-contraction following neuromuscular re-education in people with knee osteoarthritis. *BMC Musculoskelet Disord, 17*(1), 372. doi:10.1186/s12891-016-1209-2

Preece, S. J., Willan, P., Nester, C. J., Graham-Smith, P., Herrington, L., & Bowker, P. (2008). Variation in pelvic morphology may prevent the identification of anterior pelvic tilt. *Journal of Manual & Manipulative Therapy, 16*(2), 113-117.

Prince, F., Winter, D. A., Stergiou, P., & Walt, S. E. (1994). ANTICIPATORY CONTROL OF UPPER-BODY BALANCE DURING HUMAN LOCOMOTION. *Gait & Posture, 2*(1), 19-25. doi:10.1016/0966-6362(94)90013-2

Pritzker, K. P. (2003). Pathology of osteoarthritis. *Osteoarthritis, 2*, 49-58.

Prodromos, C. C., Andriacchi, T., & Galante, J. (1985a). A relationship between gait and clinical changes following high tibial osteotomy. *The Journal of bone and joint surgery. American volume, 67*(8), 1188-1194.

Prodromos, C. C., Andriacchi, T. P., & Galante, J. O. (1985b). A RELATIONSHIP BETWEEN GAIT AND CLINICAL CHANGES FOLLOWING HIGH TIBIAL OSTEOTOMY. *Journal of Bone and Joint Surgery-American Volume, 67A*(8), 1188-1194. doi:10.2106/00004623-198567080-00007

Proske, U., & Morgan, D. L. (1999). Do cross-bridges contribute to the tension during stretch of passive muscle? *Journal of Muscle Research & Cell Motility, 20*(5-6), 433-442.

Proske, U., Morgan, D. L., & Gregory, J. E. (1993). Thixotropy in skeletal muscle and in muscle spindles: a review. *Progress in neurobiology, 41*(6), 705-721.

Radin, E. L., Martin, R. B., Burr, D. B., Caterson, B., Boyd, R. D., & Goodwin, C. (1984). Effects of mechanical loading on the tissues of the rabbit knee. *Journal of Orthopaedic Research, 2*(3), 221-234.

Ramsey, D. K., Briem, K., Axe, M. J., & Snyder-Mackler, L. (2007). A mechanical theory for the effectiveness of bracing for medial compartment osteoarthritis of the knee. *J Bone Joint Surg Am, 89*(11), 2398-2407. doi:10.2106/JBJS.F.01136

Ramsey, D. K., Snyder-Mackler, L., Lewek, M., Newcomb, W., & Rudolph, K. S. (2007). Effect of Anatomic realignment on muscle function during gait in patients with medial compartment knee osteoarthritis. *Arthritis & Rheumatism-Arthritis Care & Research, 57*(3), 389-397. doi:10.1002/art.22608

Rau, G., Disselhorst-Klug, C., & Schmidt, R. (2000). Movement biomechanics goes upwards: from the leg to the arm. *Journal of Biomechanics, 33*(10), 1207-1216. doi:10.1016/s0021-9290(00)00062-2

Reize, P., Müller, O., Motzny, S., & Wülker, N. (2006). Prediction of the location of the centre of rotation of the hip joint external landmarks. *Zeitschrift Fur Orthopadie Und Ihre Grenzgebiete, 144*(5), 492-496.

Richardson, J. T. E. (2011). Eta squared and partial eta squared as measures of effect size in educational research. *Educational Research Review, 6*(2), 135-147. doi:https://doi.org/10.1016/j.edurev.2010.12.001

Roos, E. M. (2005). Joint injury causes knee osteoarthritis in young adults. *Current opinion in rheumatology, 17*(2), 195-200. doi:10.1097/01.bor.0000151406.64393.00

Rosenbaum, P., Paneth, N., Leviton, A., Goldstein, M., Bax, M., Damiano, D., . . . Jacobsson, B. (2007). A report: the definition and classification of cerebral palsy April 2006. *Dev Med Child Neurol Suppl, 109*(suppl 109), 8-14.

Rudolph, K. S., Schmitt, L. C., & Lewek, M. D. (2007). Age-related changes in strength, joint laxity, and walking patterns: are they related to knee osteoarthritis? *Physical therapy, 87*(11), 1422.

Ruiz, D., Koenig, L., Dall, T. M., Gallo, P., Narzikul, A., Parvizi, J., & Tongue, J. (2013). The Direct and Indirect Costs to Society of Treatment for End-Stage Knee Osteoarthritis. *Journal of Bone and Joint Surgery-American Volume, 95A*(16), 1473-1480. doi:10.2106/jbjs.l.01488

Rutherford, Hubley-Kozey, C. L., & Stanish, W. D. (2013). Changes in knee joint muscle activation patterns during walking associated with increased structural severity in knee osteoarthritis. *Journal of Electromyography and Kinesiology, 23*(3), 704-711. doi:10.1016/j.jelekin.2013.01.003

Rutherford, D., Baker, M., Wong, I., & Stanish, W. (2017). The effect of age and knee osteoarthritis on muscle activation patterns and knee joint biomechanics during dual belt treadmill gait. *Journal of Electromyography and Kinesiology, 34*, 58-64. doi:https://doi.org/10.1016/j.jelekin.2017.04.001

Rutherford, D. J., Hubley-Kozey, C. L., & Stanish, W. D. (2011a). Maximal voluntary isometric contraction exercises: A methodological investigation in moderate knee osteoarthritis. *Journal of Electromyography and Kinesiology, 21*(1), 154-160. doi:10.1016/j.jelekin.2010.09.004

Rutherford, D. J., Hubley-Kozey, C. L., & Stanish, W. D. (2011b). Maximal voluntary isometric contraction exercises: a methodological investigation in moderate knee osteoarthritis. *J Electromyogr Kinesiol, 21*(1), 154-160. doi:10.1016/j.jelekin.2010.09.004

Rutherford, D. J., Hubley-Kozey, C. L., Stanish, W. D., & Dunbar, M. J. (2011). Neuromuscular alterations exist with knee osteoarthritis presence and severity despite walking velocity similarities. *Clin Biomech (Bristol, Avon), 26*(4), 377-383. doi:10.1016/j.clinbiomech.2010.11.018

Ryser, L., Wright, B. D., Aeschlimann, A., Mariacher-Gehler, S., & Stucki, G. (1999). A new look at the Western Ontario and McMaster Universities Osteoarthritis Index using Rasch analysis. *Arthritis Care & Research, 12*(5), 331-335.

Saha, D., Gard, S., & Fatone, S. (2008). The effect of trunk flexion on able-bodied gait. *Gait Posture, 27*(4), 653-660. doi:10.1016/j.gaitpost.2007.08.009

Sahrmann, S. (2002). *Diagnosis and treatment of movement impairment syndromes*: Elsevier Health Sciences.

Salaffi, F., Carotti, M., & Grassi, W. (2005). Health-related quality of life in patients with hip or knee osteoarthritis: comparison of generic and disease-specific instruments. *Clinical Rheumatology, 24*(1), 29-37.

Sandell, L. J. (2012). Etiology of osteoarthritis: genetics and synovial joint development. *Nature Reviews Rheumatology, 8*(2), 77-89. doi:10.1038/nrrheum.2011.199

Santo, A. S., Roper, J. L., Dufek, J. S., & Mercer, J. A. (2012). Rocker-bottom, profile-type shoes do not increase lower extremity muscle activity or energy cost of walking. *The Journal of Strength & Conditioning Research, 26*(9), 2426-2431.

Sato, H., & Maitland, M. E. (2008). Relationship between forward trunk lean during walking and musculoskeletal functions for females. *Journal of Mechanics in Medicine and Biology, 8*(4), 459-471.

Schipplein, O. D., & Andriacchi, T. P. (1991). INTERACTION BETWEEN ACTIVE AND PASSIVE KNEE STABILIZERS DURING LEVELWALKING. *Journal of Orthopaedic Research, 9*(1), 113-119. doi:10.1002/jor.1100090114

Schloemer, S. A., Thompson, J. A., Silder, A., Thelen, D. G., & Siston, R. A. (2017). Age-Related Differences in Gait Kinematics, Kinetics, and Muscle Function: A Principal Component Analysis. *Annals of Biomedical Engineering, 45*(3), 695-710. doi:10.1007/s10439-016-1713-4

Schmitt, L. C., & Rudolph, K. S. (2008). Muscle stabilization strategies in people with medial knee osteoarthritis: the effect of instability. *J Orthop Res, 26*(9), 1180-1185. doi:10.1002/jor.20619

Schmitz, A., Silder, A., Heiderscheit, B., Mahoney, J., & Thelen, D. G. (2009). Differences in lower-extremity muscular activation during walking between healthy older and young adults. *Journal of Electromyography and Kinesiology, 19*(6), 1085-1091. doi:10.1016/j.jelekin.2008.10.008

Schneider, E., & Chao, E. (1983). Fourier analysis of ground reaction forces in normals and patients with knee joint disease. *Journal of Biomechanics, 16*(8), 591-601.

Schnitzer, T. J., Popovich, J. M., Andersson, G. B. J., & Andriacchi, T. P. (1993). EFFECT OF PIROXICAM ON GAIT IN PATIENTS WITH OSTEOARTHRITIS OF THE KNEE. *Arthritis and Rheumatism, 36*(9), 1207-1213. doi:10.1002/art.1780360905

Schwartz, M. H., Trost, J. P., & Wervey, R. A. (2004). Measurement and management of errors in quantitative gait data. *Gait & Posture, 20*(2), 196-203.

Selistre, L. F. A., Mattiello, S. M., Nakagawa, T. H., Goncalves, G. H., Petrella, M., & Jones, R. K. (2017). The relationship between external knee moments and muscle co-activation in subjects with medial knee osteoarthritis. *Journal of Electromyography and Kinesiology, 33*, 64-72. doi:10.1016/j.jelekin.2017.01.007

Shah, M. R., Kaplan, K. M., Meislin, R. J., & Bosco III, J. A. (2007). Articular cartilage restoration of the knee. *BULLETIN-HOSPITAL FOR JOINT DISEASES NEW YORK, 65*(1), 51.

Sharma, L., Hurwitz, D. E., Thonar, E., Sum, J. A., Lenz, M. E., Dunlop, D. D., . . . Andriacchi, T. P. (1998). Knee adduction moment, serum hyaluronan level, and disease severity in medial tibiofemoral osteoarthritis. *Arthritis and Rheumatism, 41*(7), 1233-1240. doi:10.1002/1529-0131(199807)41:7<1233::aid-art14>3.0.co;2-l

Sharma, L., Song, J., Felson, D. T., Cahue, S., Shamiyeh, E., & Dunlop, D. D. (2001). The role of knee alignment in disease progression and functional decline in knee osteoarthritis. *Jama-Journal of the American Medical Association, 286*(2), 188-195. doi:10.1001/jama.286.2.188

Shimada, T. (1996a). Factors affecting appearance patterns of hip-flexion contractures and their effects on postural and gait abnormalities. *Kobe Journal of Medical Sciences, 42*(4), 271-290.

Shimada, T. (1996b). Factors affecting appearance patterns of hip-flexion contractures and their effects on postural and gait abnormalities. *The Kobe journal of medical sciences, 42*(4), 271-290.

Silvers, W. M., & Dolny, D. G. (2011). Comparison and reproducibility of sEMG during manual muscle testing on land and in water. *Journal of Electromyography and Kinesiology, 21*(1), 95-101. doi:10.1016/j.jelekin.2010.05.004

Silverwood, V., Blagojevic-Bucknall, M., Jinks, C., Jordan, J. L., Protheroe, J., & Jordan, K. P. (2015a). Current evidence on risk factors for knee osteoarthritis in older adults: a systematic review and meta-analysis. *Osteoarthritis Cartilage, 23*(4), 507-515. doi:10.1016/j.joca.2014.11.019

Silverwood, V., Blagojevic-Bucknall, M., Jinks, C., Jordan, J. L., Protheroe, J., & Jordan, K. P. (2015b). Current evidence on risk factors for knee osteoarthritis in older adults: a systematic review and meta-analysis. *Osteoarthritis and Cartilage, 23*(4), 507-515. doi:10.1016/j.joca.2014.11.019

Skou, S. T., Roos, E. M., Laursen, M. B., Rathleff, M. S., Arendt-Nielsen, L., Simonsen, O., & Rasmussen, S. (2015). A randomized, controlled trial of total knee replacement. *New England Journal of Medicine, 373*(17), 1597-1606.

Sled, E. A., Khoja, L., Deluzio, K. J., Olney, S. J., & Culham, E. G. (2010). Effect of a Home Program of Hip Abductor Exercises on Knee Joint Loading, Strength, Function, and Pain in People With Knee Osteoarthritis: A Clinical Trial. *Physical therapy, 90*(6), 895-904. doi:10.2522/ptj.20090294

Slemenda, C., Brandt, K. D., Heilman, D. K., Mazzuca, S., Braunstein, E. M., Katz, B. P., & Wolinsky, F. D. (1997). Quadriceps weakness and osteoarthritis of the knee. *Annals of Internal Medicine, 127*(2), 97-&.

Sliepen, M., Mauricio, E., Lipperts, M., Grimm, B., & Rosenbaum, D. (2018). Objective assessment of physical activity and sedentary behaviour in knee osteoarthritis patients - beyond daily steps and total sedentary time. *Bmc Musculoskeletal Disorders, 19*, 10. doi:10.1186/s12891-018-1980-3

Smith, A. J., Lloyd, D. G., & Wood, D. J. (2004). Pre-surgery knee joint loading patterns during walking predict the presence and severity of anterior knee pain after total knee arthroplasty. *Journal of Orthopaedic Research, 22*(2), 260-266. doi:10.1016/s0736-0266(03)00184-0

Sobhani, S., Hijmans, J., van den Heuvel, E., Zwerver, J., Dekker, R., & Postema, K. (2013). Biomechanics of slow running and walking with a rocker shoe. *Gait & Posture, 38*(4), 998-1004. doi:10.1016/j.gaitpost.2013.05.008

Soderberg, G. L., & Knutson, L. M. (2000). A guide for use and interpretation of kinesiologic electromyographic data. *Physical therapy, 80*(5), 485-498.

Sorensen, R. R., Jorgensen, M. G., Rasmussen, S., & Skou, S. T. (2014). Impaired postural balance in the morning in patients with knee osteoarthritis. *Gait & Posture, 39*(4), 1040-1044. doi:10.1016/j.gaitpost.2014.01.002

Sowers, M. R., & Karvonen-Gutierrez, C. A. (2010). The evolving role of obesity in knee osteoarthritis. *Current opinion in rheumatology, 22*(5), 533-537. doi:10.1097/BOR.0b013e32833b4682

Spector, T. D., Hart, D. J., & Doyle, D. V. (1994). INCIDENCE AND PROGRESSION OF OSTEOARTHRITIS IN WOMEN WITH UNILATERAL KNEE DISEASE IN THE GENERAL-POPULATION - THE EFFECT OF OBESITY. *Annals of the Rheumatic Diseases, 53*(9), 565-568. doi:10.1136/ard.53.9.565

Srikanth, V. K., Fryer, J. L., Zhai, G., Winzenberg, T. M., Hosmer, D., & Jones, G. (2005). A meta-analysis of sex differences prevalence, incidence and severity of osteoarthritis. *Osteoarthritis and Cartilage, 13*(9), 769-781. doi:10.1016/j.joca.2005.04.014

Sritharan, P., Lin, Y. C., Richardson, S. E., Crossley, K. M., Birmingham, T. B., & Pandy, M. G. (2016). Musculoskeletal loading in the symptomatic and asymptomatic knees of middle-aged osteoarthritis patients. *Journal of Orthopaedic Research*.

Steultjens, M., Dekker, J., & van der Esch, M. (2006). The pros and cons of muscle co-contraction in osteoarthritis of the knee: comment on the article by Lewek et al. *Arthritis and Rheumatism, 54*(4), 1354-1354. doi:10.1002/art.21781

Stoppiello, L. A., Mapp, P. I., Wilson, D., Hill, R., Scammell, B. E., & Walsh, D. A. (2014). Structural Associations of Symptomatic Knee Osteoarthritis. *Arthritis & Rheumatology, 66*(11), 3018-3027. doi:10.1002/art.38778

Sun, H. B. (2010). Mechanical loading, cartilage degradation, and arthritis. *Annals of the New York Academy of Sciences, 1211*(1), 37-50.

Team, R. C. (2017). R: A language and environment for statistical computing.

Thoma, L. M., McNally, M. P., Chaudhari, A. M., Flanigan, D. C., Best, T. M., Siston, R. A., & Schmitt, L. C. (2016). Muscle co-contraction during gait in individuals with articular cartilage defects in the knee. *Gait & Posture, 48*, 68-73. doi:10.1016/j.gaitpost.2016.04.021

Thorstensson, A., Nilsson, J., Carlson, H., & ZOMLEFER, M. R. (1984). Trunk movements in human locomotion. *Acta Physiologica Scandinavica, 121*(1), 9-22.

Thorstensson, C. A., Petersson, I., Jacobsson, L., Boegård, T., & Roos, E. (2004). Reduced functional performance in the lower extremity predicted radiographic knee osteoarthritis five years later. *Annals of the Rheumatic Diseases, 63*(4), 402-407.

Trepczynski, A., Kutzner, I., Schwachmeyer, V., Heller, M. O., Pfitzner, T., & Duda, G. N. (2018). Impact of antagonistic muscle co-contraction on in vivo knee contact forces. *Journal of Neuroengineering and Rehabilitation, 15*(1), 101.

Turcot, K., Sagawa, Y., Hoffmeyer, P., Suva, D., & Armand, S. (2015). Multi-joint postural behavior in patients with knee osteoarthritis. *Knee, 22*(6), 517-521. doi:10.1016/j.knee.2014.09.001

Vad, V. B., & Dysart, S. H. (2012). Managing Knee Osteoarthritis: Systemic Pharmacotherapy and Intra-Articular Treatments. *The Journal of Musculoskeletal Medicine (Online)*.

van der Stouwe, A. M. M., Toxopeus, C. M., de Jong, B. M., Yavuz, P., Valsan, G., Conway, B. A., . . . Maurits, N. M. (2015). Muscle co-activity tuning in Parkinsonian hand movement: disease-specific changes at behavioral and cerebral level. *Frontiers in Human Neuroscience, 9*, 437. doi:10.3389/fnhum.2015.00437

van Ingen Schenau, G., Boots, P., De Groot, G., Snackers, R., & Van Woensel, W. (1992). The constrained control of force and position in multi-joint movements. *Neuroscience, 46*(1), 197-207.

van Raaij, T. M., Reijman, M., Brouwer, R. W., Bierma-Zeinstra, S. M., & Verhaar, J. A. (2010). Medial knee osteoarthritis treated by insoles or braces: a randomized trial. *Clinical Orthopaedics and Related Research®, 468*(7), 1926-1932.

Vigotsky, A. D., Lehman, G. J., Beardsley, C., Contreras, B., Chung, B., & Feser, E. H. (2016). The modified Thomas test is not a valid measure of hip extension unless pelvic tilt is controlled. *Peerj, 4*. doi:10.7717/peerj.2325

Vos, T., Flaxman, A. D., Naghavi, M., Lozano, R., Michaud, C., Ezzati, M., . . . Murray, C. J. L. (2012). Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet, 380*(9859), 2163-2196.

Wakefield, C. B., Halls, A., Difilippo, N., & Cottrell, G. T. (2015). Reliability of Goniometric and Trigonometric Techniques for Measuring Hip-Extension Range of Motion Using the Modified Thomas Test. *Journal of Athletic Training, 50*(5), 460-466. doi:10.4085/1062-6050-50.2.05

Wallis, J., Webster, K., Levinger, P., & Taylor, N. (2013). What proportion of people with hip and knee osteoarthritis meet physical activity guidelines? A systematic review and meta-analysis. *Osteoarthritis and Cartilage, 21*(11), 1648-1659.

Wang, J.-W., Kuo, K., Andriacchi, T., & Galante, J. (1990). The influence of walking mechanics and time on the results of proximal tibial osteotomy. *The Journal of bone and joint surgery. American volume, 72*(6), 905-909.

Wang, Y., Simpson, J. A., Wluka, A. E., Teichtahl, A. J., English, D. R., Giles, G. G., . . . Cicuttini, F. M. (2009). Relationship between body adiposity measures and risk of primary knee and hip replacement for osteoarthritis: a prospective cohort study. *Arthritis research & therapy, 11*(2), R31.

Watt, I., & Doherty, M. (2003). Plain radiographic features of osteoarthritis. *Osteoarthritis, 2*, 211-225.

Westfall, P. H., Tobias, R. D., & Wolfinger, R. D. (2011). *Multiple comparisons and multiple tests using SAS*: SAS Institute.

Williams, P. (1990). Use of intermittent stretch in the prevention of serial sarcomere loss in immobilised muscle. *Annals of the Rheumatic Diseases, 49*(5), 316.

Williams, P. E., & Goldspink, G. (1973). The effect of immobilization on the longitudinal growth of striated muscle fibres. *Journal of Anatomy, 116*(Pt 1), 45.

Williams, P. E., & Goldspink, G. (1978). Changes in sarcomere length and physiological properties in immobilized muscle. *Journal of Anatomy, 127*(Pt 3), 459.

Wilson, J. L. A., Deluzio, K. J., Dunbar, M. J., Caldwell, G. E., & Hubey-Kozey, C. L. (2011). The association between knee joint biomechanics and neuromuscular control and moderate knee osteoarthritis radiographic and pain severity. *Osteoarthritis and Cartilage, 19*(2), 186-193. doi:10.1016/j.joca.2010.10.020

Winby, C. R., Gerus, P., Kirk, T. B., & Lloyd, D. G. (2013). Correlation between EMG-based co-activation measures and medial and lateral compartment loads of the knee during gait. *Clinical Biomechanics, 28*(9), 1014-1019. doi:http://dx.doi.org/10.1016/j.clinbiomech.2013.09.006

Winby, C. R., Lloyd, D. G., Besier, T. F., & Kirk, T. B. (2009). Muscle and external load contribution to knee joint contact loads during normal gait. *Journal of Biomechanics, 42*(14), 2294-2300. doi:10.1016/j.jbiomech.2009.06.019

Winkel, J., & Jørgensen, K. (1991). Significance of skin temperature changes in surface electromyography. *European Journal of Applied Physiology and Occupational Physiology, 63*(5), 345-348.

Winter, D., Fuglevand, A., & Archer, S. (1994a). Crosstalk in surface electromyography: theoretical and practical estimates. *Journal of Electromyography and Kinesiology, 4*(1), 15-26.

Winter, D. A. (1990). *BIOMECHANICS AND MOTOR CONTROL OF HUMAN MOVEMENT SECOND EDITION*.

Winter, D. A. (2009). *Biomechanics and motor control of human movement*: John Wiley & Sons.

Winter, D. A., Fuglevand, A. J., & Archer, S. E. (1994b). CROSSTALK IN SURFACE ELECTROMYOGRAPHY - THEORETICAL AND PRACTICAL ESTIMATES. *Journal of Electromyography and Kinesiology, 4*(1), 15-26. doi:10.1016/1050-6411(94)90023-x

Winter, D. A., & Yack, H. J. (1987). EMG PROFILES DURING NORMAL HUMAN WALKING - STRIDE-TO-STRIDE AND INTER-SUBJECT VARIABILITY. *Electroencephalography and Clinical Neurophysiology, 67*(5), 402-411. doi:10.1016/0013-4694(87)90003-4

Wise, B. L., Niu, J. B., Yang, M., Lane, N. E., Harvey, W., Felson, D. T., . . . Multictr Osteoarthritis, M. G. (2012). Patterns of Compartment Involvement in Tibiofemoral Osteoarthritis in Men and Women and in Whites and African Americans. *Arthritis Care & Research, 64*(6), 847-852. doi:10.1002/acr.21606

Yang, J., & Winter, D. (1983). Electromyography reliability in maximal and submaximal isometric contractions. *Archives of Physical Medicine and Rehabilitation, 64*(9), 417-420.

Yang, K. G. A., Saris, D. B. F., Dhert, W. J. A., & Verbout, A. J. (2004). Osteoarthritis of the knee: current treatment options and future directions. *Current Orthopaedics, 18*(4), 311-320. doi:10.1016/j.cuor.2004.04.005

Ylinen, J. (2008). *Stretching Therapy: for sport and manual therapies*: Elsevier Health Sciences.

Youdas, J. W., Garrett, T. R., Harmsen, S., Suman, V. J., & Carey, J. R. (1996). Lumbar lordosis and pelvic inclination of asymptomatic adults. *Physical therapy, 76*(10), 1066-1081.

Young, W., Clothier, P., Otago, L., Bruce, L., & Liddell, D. (2003). Relationship between a modified Thomas test and leg range of motion in Australian-Rules football kicking. *Journal of Sport Rehabilitation, 12*(4), 343-350.

Yusuf, E., Kortekaas, M. C., Watt, I., Huizinga, T. W., & Kloppenburg, M. (2011). Do knee abnormalities visualised on MRI explain knee pain in knee osteoarthritis? A systematic review. *Annals of the Rheumatic Diseases, 70*(1), 60-67.

Zeni, J. A., & Higginson, J. S. (2009). Differences in gait parameters between healthy subjects and persons with moderate and severe knee osteoarthritis: a result of altered walking speed? *Clinical Biomechanics, 24*(4), 372-378.

Zeni, J. A., Rudolph, K., & Higginson, J. S. (2010a). Alterations in quadriceps and hamstrings coordination in persons with medial compartment knee osteoarthritis. *Journal of Electromyography and Kinesiology, 20*(1), 148-154. doi:10.1016/j.jelekin.2008.12.003

Zeni, J. A., Rudolph, K., & Higginson, J. S. (2010b). Alterations in quadriceps and hamstrings coordination in persons with medial compartment knee osteoarthritis. *J Electromyogr Kinesiol, 20*(1), 148-154. doi:10.1016/j.jelekin.2008.12.003

Zhang, F. F., Driban, J. B., Lo, G. H., Price, L. L., Booth, S., Eaton, C. B., . . . McAlindon, T. E. (2014). Vitamin D Deficiency Is Associated with Progression of Knee Osteoarthritis. *Journal of Nutrition, 144*(12), 2002-2008. doi:10.3945/jn.114.193227

Zhang, W., Doherty, M., Arden, N., Bannwarth, B., Bijlsma, J., Gunther, K.-P., . . . Kaklamanis, P. (2005). EULAR evidence based recommendations for the management of hip osteoarthritis: report of a task force of the EULAR Standing Committee for International Clinical Studies Including Therapeutics (ESCISIT). *Annals of the Rheumatic Diseases, 64*(5), 669-681.

Zhang, Y., & Jordan, J. M. (2010). Epidemiology of osteoarthritis. *Clin Geriatr Med, 26*(3), 355-369. doi:10.1016/j.cger.2010.03.001

Zhou, S., Lawson, D. L., Morrison, W. E., & Fairweather, I. (1995). Electromechanical delay in isometric muscle contractions evoked by voluntary, reflex and electrical stimulation. *European Journal of Applied Physiology and Occupational Physiology, 70*(2), 138-145.

Appendices

Appendix I

University of Salford's Ethical approval

University of Salford MANCHESTER

Research, Innovation and Academic Engagement Ethical Approval Panel

Research Centres Support Team
G0.3 Joule House
University of Salford
M5 4WT

T +44(0)161 295 2280

www.salford.ac.uk/

24 March 2017

Dear Wael,

RE: ETHICS APPLICATION–HSR1617-98–'The influence of trunk inclination on lower limb moments and muscle activation.'

Based on the information you provided I am pleased to inform you that application HSR1617-98 has been approved.

If there are any changes to the project and/or its methodology, then please inform the Panel as soon as possible by contacting Health-ResearchEthics@salford.ac.uk

Yours sincerely,

Appendix II

IRAS's Ethical approval

NHS
Health Research Authority

Dr Stephen Preece
Blatchford Building
University of Salford
Manchester
M6 6PU
s.preece@salford.ac.uk

Email: hra.approval@nhs.net

02 February 2018 [Re-issued 05 February 2018 to reflect the names of participating NHS sites]

Dear Dr Preece

Letter of HRA Approval

Study title:	**The influence of trunk inclination on lower limb moment and muscle activation.**
IRAS project ID:	**235079**
REC reference:	**18/NW/0030**
Sponsor	**University of Salford**

I am pleased to confirm that **HRA Approval** has been given for the above referenced study, on the basis described in the application form, protocol, supporting documentation and any clarifications noted in this letter.

Participation of NHS Organisations in England

The sponsor should now provide a copy of this letter to all participating NHS organisations in England.

Appendix B provides important information for sponsors and participating NHS organisations in England for arranging and confirming capacity and capability. **Please read *Appendix B* carefully**, in particular the following sections:

- *Participating NHS organisations in England* – this clarifies the types of participating organisations in the study and whether or not all organisations will be undertaking the same activities
- *Confirmation of capacity and capability* - this confirms whether or not each type of participating NHS organisation in England is expected to give formal confirmation of capacity and capability.

Appendix III

Search strategies

Search strategy for the trunk inclination:

The following databases were searched in November 2016 and again in February 2019: Web of science (All databases), Cochrane PubMed (Medline), CINAHL (EBSCO) and Science direct. Key words used were: 'trunk inclination', 'trunk lean', 'trunk flexion', 'posture', 'attitude' 'upper limb', 'thorax', 'pelvic inclination', 'biomechanics', 'Kinematics', 'Kinetics', 'moment', 'torque', 'angle', 'EMG', 'electromyography', 'muscle activation', 'muscle activity', 'co-contraction', co-activity', 'cocontraction', 'coactivity', 'Gait', 'walk', 'stand', 'healthy', 'able body', 'osteoarthritis', 'OA', 'orthosis', 'osteo', 'degeneration', 'knee',

Boolean logic (AND, OR) was used to combine them.

1. 'trunk inclination' OR 'trunk lean' OR 'trunk flexion' OR 'posture' OR 'attitude' OR 'upper limb' OR 'thorax' OR 'pelvic inclination'.
2. 'biomechanics' OR 'Kinematic' OR 'Kinetic' OR 'moment' OR 'torque' OR 'angle' OR 'EMG' OR 'electromyography' OR 'muscle activation' OR 'muscle activity' OR 'co-contraction' OR co-activity' OR 'cocontraction' OR 'coactivity'.
3. 'healthy' OR 'able body' OR 'osteoarthritis' OR 'OA' OR 'orthosis' OR 'osteo' OR 'degeneration'.
4. Knee
5. 'Gait' OR 'walk' OR 'stand'.

Papers were excluded: standing, initial walking

Search strategy for the hip flexor muscle length:

Key words used were: Hip flexion tightness, Hip flexor contracture, Thomas test /Modified Thomas test, Iliacus Test/ Iliopsoas Test, hip flexor muscle tightness, Posture, trunk inclination, trunk forward, Trunk lean, pelvic inclination, upper limb, thorax, Kinematics, angles, moment, torque, kinetics, Gait, walk, stand, healthy, biomechanics,

Boolean logic (AND, OR) was used to combine them.

1. Hip flexion tightness, Hip flexor contracture, Thomas test /Modified Thomas test, Iliacus Test/ Iliopsoas Test, hip flexor muscle tightness, hip flexor muscle length
2. Posture, trunk inclination, trunk forward, Trunk lean, pelvic inclination, upper limb, thorax
3. Kinematics, angles, moment, torque, kinetics, biomechanics,
4. Gait, walk, stand, healthy.

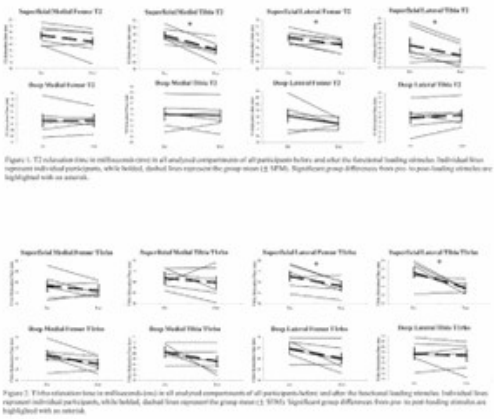
Appendix IV

OARSI 2018 abstract

stimulus, which consists of 25 min of challenged walking on an instrumented, dual-belt treadmill capable of moving with 6 degrees of freedom. During walking, participants are subjected to changes in speed, inclines and declines, lateral sways, and random pre-specified perturbations in the form of rapid belt slips, sagittal plane pitches, and frontal plane sways. Immediately following challenged walking, participants undergo a post-loading MRI with the same sequences as the baseline scan. T1rho and T2 relaxation maps are generated using software developed in-house by fitting image intensities of the T1rho and T2 weighted images pixel-by-pixel to the equation $S(TE) \propto \exp(-TE/T2)$ using a Levenberg-Marquardt mono-exponential fitting algorithm implemented in ITK. Superficial and deep load-bearing regions of medial and lateral articular cartilage of the femur and tibia are manually segmented and analyzed using 3D Slicer software. The reader is blinded to scan order. The individual and group mean T1rho and T2 relaxation times before and after challenged walking were plotted and compared using paired t-tests.

Results: Six participants at-risk for knee OA have completed the protocol to date. Following loading, T2 relaxation time of the superficial medial tibia, superficial lateral femur, and superficial lateral tibia were all significantly lower ($P = 0.03$) compared to baseline (Figure 1), and T1rho relaxation time of the superficial lateral femur and superficial lateral tibia were significantly lower ($P = 0.04$) compared to baseline (Figure 2).

Conclusions: T1rho and T2 relaxation time decreased in several superficial load-bearing regions after challenged walking, suggesting a decrease in water content in articular cartilage after the functional loading stimulus. These results support the use of the standardized challenging walking test to evoke acute changes in knee articular cartilage.



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DYNAMIC LIMIT OF STABILITY AND ANKLE JOINT FUNCTION FOLLOWING NEUROMUSCULAR TRAINING OF UNSTABLE ANKLE JOINTS

A.R. Ibrahim, A.A. Abdallah. *Faculty of Physical Therapy, Giza, Egypt*

Purpose: This study investigated and correlated between the dynamic limit of stability and functional level of the ankle joint in patients with unilateral chronic ankle instability after receiving dynamic neuromuscular training.

Methods: Forty patients of both sexes were examined. They were assigned into two equal sex-matched groups; experimental (Group A) and control (Group B). The mean age, weight and height were 19.15 ± 1.66 years, 65.3 ± 9.52 kg, and 170.4 ± 8.29 cm respectively for Group A and 19.6 ± 1.5 years, 68.5 ± 6.87 kg, and 170.1 ± 8.83 cm respectively for Group B. Both groups were tested before and after a 4-week period during which Group A received neuromuscular training. The Biodex Balance system was used for assessing the dynamic limit of stability and the ankle joint functional assessment tool (AJFAT) was used for assessing the functional level of the ankle joint.

Results: Mixed Design MANOVA revealed that the dynamic limit of stability test duration decreased and the AJFAT score increased significantly in Group A after training compared with before ($P = 0.000$). Similarly, the dynamic limit of stability test duration decreased and the AJFAT score increased significantly in Group A compared with Group B after training ($P = 0.000$). Pearson correlation showed significant strong negative correlation between the dynamic limit of stability test duration and AJFAT score in Group A ($r = -0.647$, $P = 0.000$).

Conclusions: Improvement in dynamic stability and functional joint stability level were perceived with neuromuscular training. This improvement reflects the ability of training to enhance ankle joint sensorimotor capabilities.

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HOW DO LOWER LIMB JOINT MOMENTS AND MUSCLE ACTIVATIONS CHANGE WHEN FORWARD TRUNK LEAN INCREASES?

W.A. Alghamdi †‡, S.J. Preece †, R. Jones §, H. Tucker §. † *Univ. of Salford, Manchester, United Kingdom;* ‡ *Al Baha Univ., Al-Baha, Saudi Arabia;* § *Univ. of Bath, Bath, United Kingdom*

Purpose: There has been a large amount of previous research which has demonstrated changes in lower limb joint moments and muscle activation patterns in people with knee osteoarthritis (OA). One of the changes most consistently observed is an increase in hamstring activity during the early stance phase of walking. However, although this pattern may lead to increased co-contraction and compressive knee joint loading, it is not clear whether it is a localised muscular response or the result of a change in upper body positioning. People with knee OA stand and walk with an increase in sagittal plane inclination of the trunk. Increased trunk inclination, or forward lean, will alter the position of the ground reaction force vector relative to lower limb joint centres and this may partially explain the previously observed changes in moments and hamstring muscle patterns. However, the precise relationship between forward trunk lean and moments/muscle activation has not been investigated. Therefore, this study sought to quantify the effect of small changes in trunk forward lean.

Methods: Kinematic, kinetic and EMG data were collected from 19 health participants in four different walking conditions: normal walking (NW), NW + 5° forward lean, NW + 10° forward lean, NW − 5° degree forward lean. In order to ensure the appropriate trunk inclination angle, a real time biofeedback approach was used to provide instruction to participants after each walking trial. A minimum of 7 successful trials (defined as being within +/−2 degrees of the target trunk inclination) were recorded for each condition. EMG data was collected from the medial/lateral hamstrings, medial/lateral quadriceps and medial/lateral gastrocnemius and normalised, for each muscle, using a maximal isometric contraction. Following data collection, joint moments and muscle activations were derived. Average moments and muscle activation were then values calculated over the period 15−25% of stance as this corresponds to a period of peak knee loading. A repeated measure ANOVA, with Bonferroni post hoc testing, was then used to determine if different forward lean conditions were associated with differences in moment and muscle patterns.

Results: As trunk lean increased, there was a significant increase in both the hip and ankle moment (p&l 0.01) but no change in the knee moment. Interestingly, post hoc testing showed significant increases of 100% in the hip moment and 500% in the ankle moment between the NW condition and the NW + 5° condition (p&lt 0.01). There were also marked changes in muscle activation patterns. Specifically, both the semitendinosus (Figure below) and biceps femoris muscles showed large increases in activity as trunk lean increased (p&l 0.01). Post hoc testing showed changes of 100% in semitendinosus activity and 62% in biceps femoris activity between the NW condition and the NW + 5° condition (p&l 0.01). However, although there was a significant increase in medial gastrocnemius activity (p&l 0.01), there were no changes in either of the quadriceps muscles or the lateral gastrocnemius muscle.

Conclusions: These data demonstrate that relatively small increases forward trunk lean are associated with large increase in hamstring activity but insignificant changes in quadriceps activity. Taken together, these findings indicate that walking with an increased forward lean will increase co-contraction during the period 15−25% of stance. Given that this period of stance is associated with peak knee loads, the data demonstrate that subtle changes in upper body position have the potential to increase knee loads. Although further study is needed in people with knee osteoarthritis, the results indicate that interventions

Appendix v

Participant information sheet

PARTICIPANT INFORMATION SHEET

Title of study: The influence of trunk inclination on lower limb moments and muscle activation.

Name of researcher: Wael Alghamdi

You are being invited to take part in a research study to help us understand a potential mechanism of pain in the knee osteoarthritis. Before you decide, it is important for you to understand why the research is being done and what it will involve. This document gives you important information about the purpose, risks, and benefits of participating in the study. Please take time to read the following information carefully. If you have any questions, then feel free to contact the researcher whose details are given at the end of the document. Take time to decide whether or not you wish to take part.

What is the purpose of the study?

Individuals with knee osteoarthritis suffer from pain during normal activities such as walking, standing or climbing stairs. We aim to gain more information about the cause of pain in knee osteoarthritis (OA). We are studying the biomechanics of walking, both in people with knee OA and also healthy volunteers. For our current project we are trying to understand how trunk inclination (forward lean of the upper body) affects the way in which the muscles in the legs work. We think increased trunk lean may be responsible for abnormal muscle patterns in people with knee OA and that this could overstress their knee joint, leading to more pain.

Why have I been invited to take part?

You have been invited as you are either a healthy individual or you are affected by knee OA.

Do I have to take part?

No, taking part is completely voluntary. If you are interested, contact the researcher (details at the end of this information sheet). If you are not interested, then just disregard this letter.

What will happen to me if I participate in this study?

If you agree to take part in the study, you will be required to visit the Podiatry laboratory at the University of Salford on a single occasion. At the start of your visit, the study will be explained in full and, provided you are still happy to proceed, you will complete a consent form. After this we will measure your height and weight and then ask you to change into shorts and a comfortable T-shirt. We will then ask for permission to access your knee x-ray data via a data access form. You will then complete a short questionnaire which will allow us to understand how much your knee osteoarthritis interferes with your daily life (note that if you are healthy subject we will omit this part). Unless you tell us not to, we will write to your GP to let them know you have taken part in this study (if you are a participant with knee OA). We will also ask your GP for your NHS number.

What will the testing involve?

1. Firstly, we will measure the flexibility of the muscles at the front of your hip. For this test, you will be lie on your back and we will ask you to hold your non-tested knee to your chest (Figure

1). The other (tested leg) will then be allowed to hang freely and we will use a device which can measure the angle of the upper leg to give us an indication of your hip flexibility.

2. The researcher will then place small electrodes over specific muscles on the front and back of your thigh and on your calf. Before each of these electrodes is positioned, the researcher may need to shave a very small area of skin and will then clean this area with an exfoliating cream and an alcohol wipe.
3. Once the electrodes are in place, the researcher will then position reflective markers on your legs, feet and upper body using hypo-allergenic tape. With the electrodes and markers in place the researcher is able to capture biomechanical data during movement.
4. To begin with you will be asked to stand still whilst a standing trial is recorded. We will ask you do to this a number of times and, on some tests, ask you to lean slightly forward or backwards. For each test, we will use computer software to show you how much to lean.
5. Then you will be required to walk normally 5-10 times up and down our laboratory at your normal walking speed. Once this is complete, you will walk again, but this time, leaning either slightly forward or backwards. Again, we will give you feedback after each trial to let you know that you have done it correctly.
6. Following the walking trials, you will be asked to perform some maximal muscle. These tests will involve you pushing as hard as you can against the examiners hand to maximally activity specific groups of muscles.

Figure 1 Hip flexor muscle tightness test.

If you get tired during any point, then we will give you sufficient time to rest. We anticipate that the total duration of this visit would be no longer than 1.5-2 hours.

Expenses and payments?

You will receive £20 for taking part in this experiment by way of a thank you for your time. We will also cover any travel costs you might have incurred. If you can't travel independently, then can arrange for a taxi to collect you.

What are the possible disadvantages and risks of taking part?

This is a very simple, straightforward study with negligible risks. The laboratory measurements of walking are often carried out in routine clinical practice and will be performed by a fully trained researcher with state-of-the-art equipment.

What are the possible benefits of taking part?

There are no immediate benefits to you of participating in the study. However, the results will help us understand if leaning forward will lead to increased muscle activity during walking. This could ultimately help us to develop effective treatments for people who suffer with knee arthritis.

What if there is a problem?

The university has insurance to cover against any harm to you which may occur whilst you are taking part in these tests. However, if you decide to take legal action, you may have to pay for this. If you wish to complain, or have any concerns about any aspect of the way you have been approached or treated during the course of this study, you can contact the project supervisor Dr Steve Preece on 0161 295 2273 or email s.preece@salford.ac.uk and if you are not happy you may then contact Professor Sue McAndrew, Ethics Chair, Mary Seacole Building, University of Salford, M5 4WT on 0161 295 2778 or email: S.McAndrew@salford.ac.uk.

What will happen if I don't carry on with the study?

You can withdraw from this study at any time without loss of any non-study related benefits to which you would have been entitled before participating in the study. There is no danger to you if you leave the study early. If you want to withdraw you may do so at any time by notifying the study representative listed in the "Contact Information" section below. If you do decide to withdraw then all the data which has been collected up to that point will remain part of the study unless you specifically request that it is destroyed. Note that data can't be withdrawn after data analysis has been completed.

Who is organizing and funding the research?

This study is organised and funded by the University of Salford.

Will my taking part in the study be kept confidential?

All information which is collected about you during the course of the research will be kept strictly confidential. Any information about you which leaves the University of Salford will have your name and address and any other identifying features removed so that you cannot be recognized from it. We are happy to send each participant in the study a summary of the results. Please indicate on the consent form if you would like to receive this summary and also confirm that you are happy for us to retain your contact information for 2-3 years to allow us to send this information to you.

Further information and contact details:

If you require more information about the study, want to participate, or if you are already participating and want to withdraw, please contact

Name:	Wael Alghamdi
Email:	w.alghamdi@edu.salford.ac.uk
Phone:	07460675029
Address:	School of Health Sciences

Appendix VI

WOMAC questionnaire

WOMAC Osteoarthritis Index LK3.1 (IK)

Section A

PAIN

Think about the pain you felt during the last 48 hours caused by the arthritis in your knee to be injected.

(Please mark your answers with an " ✗ ".)

QUESTION: **How much pain have you had . . .**	Study Coordinator Use Only
1. when walking on a flat surface? none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	PAIN1 ____
2. when going up or down stairs? none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	PAIN2 ____
3. at night while in bed? (that is - pain that disturbs your sleep) none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	PAIN3 ____
4. while sitting or lying down? none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	PAIN4 ____
5. while standing? none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	PAIN5 ____

V3 - English for USA
(at baseline)

WOMAC Osteoarthritis Index LK3.1 (IK)

Section B

STIFFNESS

Think about the stiffness (not pain) you felt during the last 48 hours caused by the arthritis in your knee to be injected.

Stiffness is a sensation of **decreased** ease in moving your joint.

(Please mark your answers with an " ✗ ".)

	Study Coordinator Use Only
6. How **severe** has your stiffness been **after you first woke up** in the morning? none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	STIFF6 _____
7. How **severe** has your stiffness been after sitting or lying down or while resting **later in the day**? none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	STIFF7 _____

V3 - English for USA
(at baseline)

WOMAC Osteoarthritis Index LK3.1 (IK)

Section C

DIFFICULTY PERFORMING DAILY ACTIVITIES

Think about the difficulty you had in doing the following daily physical activities during the last 48 hours caused by the arthritis in your knee to be injected. By this we mean **your ability to move around and take care of yourself**.

(Please mark your answers with an " ✗ ".)

QUESTION: **How much difficulty have you had . . .**	none	mild	moderate	severe	extreme	Study Coordinator Use Only
8. when going down the stairs?	❑	❑	❑	❑	❑	PFTN8 ____
9. when going up the stairs?	❑	❑	❑	❑	❑	PFTN9 ____
10. when getting up from a sitting position?	❑	❑	❑	❑	❑	PFTN10 ____
11. while standing?	❑	❑	❑	❑	❑	PFTN11 ____
12. when bending to the floor?	❑	❑	❑	❑	❑	PFTN12 ____
13. when walking on a flat surface?	❑	❑	❑	❑	❑	PFTN13 ____

V3 - English for USA
(at baseline)

WOMAC Osteoarthritis Index LK3.1 (IK)

DIFFICULTY PERFORMING DAILY ACTIVITIES

Think about the difficulty you had in doing the following daily physical activities during the last 48 hours caused by the arthritis in your knee to be injected. By this we mean **your ability to move around and take care of yourself**.

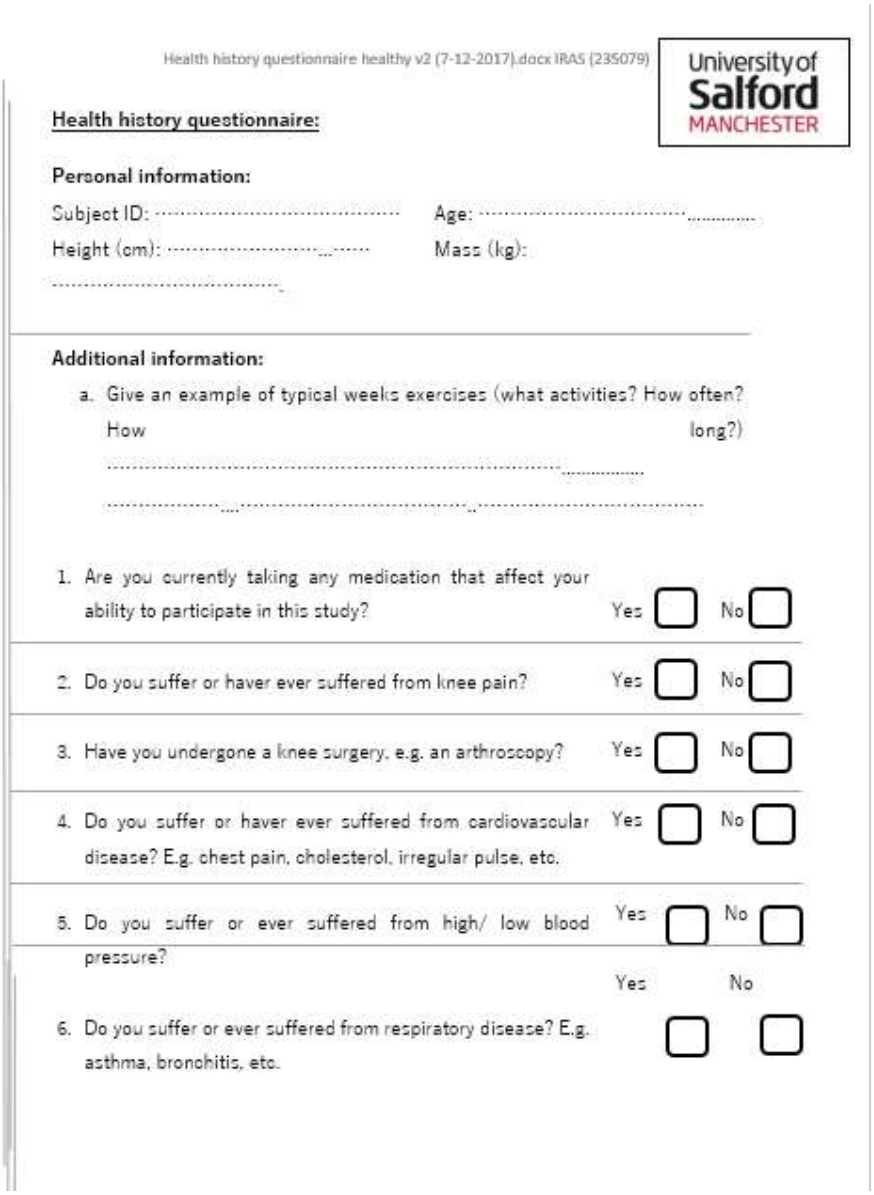
(Please mark your answers with an " ✗ ".)

QUESTION: **How much difficulty have you had . . .**	Study Coordinator Use Only
14. getting in or out of a car, or getting on or off a bus? none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	PFTN14 ____
15. while going shopping? none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	PFTN15 ____
16. when putting on your socks or panty hose or stockings? none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	PFTN16 ____
17. when getting out of bed? none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	PFTN17 ____
18. when taking off your socks or panty hose or stockings? none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	PFTN18 ____
19. while lying in bed? none ❑ mild ❑ moderate ❑ severe ❑ extreme ❑	PFTN19 ____

V3 - English for USA
(at baseline)

Appendix VII

Healthy history questionnaire



Health history questionnaire healthy v2 (7-12-2017).docx IRAS (235079)

University of Salford MANCHESTER

Health history questionnaire:

Personal information:

Subject ID: ..... Age: .....

Height (cm): ..... Mass (kg):

.....

Additional information:

a. Give an example of typical weeks exercises (what activities? How often? How long?)

.....

.....

1. Are you currently taking any medication that affect your ability to participate in this study? Yes ☐ No ☐
2. Do you suffer or haver ever suffered from knee pain? Yes ☐ No ☐
3. Have you undergone a knee surgery, e.g. an arthroscopy? Yes ☐ No ☐
4. Do you suffer or haver ever suffered from cardiovascular disease? E.g. chest pain, cholesterol, irregular pulse, etc. Yes ☐ No ☐
5. Do you suffer or ever suffered from high/ low blood pressure? Yes ☐ No ☐
6. Do you suffer or ever suffered from respiratory disease? E.g. asthma, bronchitis, etc. Yes ☐ No ☐

7. Do you suffer or ever suffered from diabetes?	☐	☐
8. Do you suffer or ever suffered from epilepsy/ seizures?	☐	☐
9. Have you had a cold or feverish illness within the last two weeks?	☐	☐
10. Do you ever lose balance because of dizziness, or do you ever lose consciousness?	☐	☐
11. Are you currently receiving treatment or medical advice from a GP or physiotherapist?	☐	☐
12. Are there other reasons, not mentioned above, why you should not exercise? E.g. an accident, pregnancy, surgeries, or anything else?	☐	☐

Appendix VIII

ORS 2019

The effect of inter-subject variations in trunk inclination on lower limb moment and muscle activation during walking

W. A. Alghamdi [1,2], S. J. Preece[1], R. Jones[1], H. Tucker [3],

[1] Univ. of Salford, Manchester, United Kingdom, [2] Al Baha Univ., Al-Baha, Saudi Arabia, [3] Univ. of Bath, Bath, United Kingdom

Disclosures: The authors have no financial conflicts to disclose.

INTRODUCTION: Previous research has shown that people with knee osteoarthritis (OA) exhibit differences in sagittal hip moments, sagittal knee moments and muscle activation patterns during normal walking. Data is also emerging which suggests that that people with knee OA walk with an increased sagittal plane inclination of the trunk. This increased trunk inclination, or forward lean, will alter the position of the ground reaction force vector relative to lower limb joint centres and may be the mechanism that underlies the alterations in sagittal plane moments and muscle activity, observed in previous studies of knee OA. However, it is also possible that these biomechanical alterations are a direct response to the disease. Therefore, in order to better understand the effect of increased trunk inclination, this study sought to compare sagittal plane moments and muscle activation patterns between two groups of young healthy subjects who habitually walk with different trunk inclination angles.

METHODS: Kinematic, kinetic and EMG data were collected from 34 young (age range 21-35) healthy participants during normal walking at their self-selected speed. Participants provided informed consent following approval from the Institution Ethics Board. EMG data was collected from the medial/lateral hamstrings, medial/lateral quadriceps and medial/lateral gastrocnemius and normalised, for each muscle, using a maximal isometric contraction. Lower limb kinematic data were collected from the foot, shank, thigh and pelvic segments and trunk segment was defined from the greater trochanters and acromions and tracked using markers on the jugular notch, T2 and T8. Following derivation of the individual EMG profiles and kinetic trajectories, sagittal trunk inclination angle was used to define two groups using a modified median split. The first group, forward leaners (FW), contained n=15 participants and the second group, backward leaners (BW), contained 14 participants, with the remaining five subjects excluded as they were within the standard error of measurement from the median trunk angle. Peak moments and EMG amplitudes, averaged across muscle-specific windows, were then compared between the two groups using an independent t-test.

RESULTS: There was a difference in trunk angle of approximately 3° across the gait cycle between the FW and BW groups (Figure below) . FW were observed to have an increased hip extensor moment peak and higher medial and lateral gastrocnemius muscle activity across the window 55-85% of stance phase. FW also demonstrated 70% more biceps femoris muscle activity during the period -30-0% of stance phase.

DISCUSSION: These data show that healthy people, who habitually walk with an increased forward lean, demonstrate differences in specific biomechanical patterns which are similar to the previously observed differences between people with knee OA and healthy age-matched controls. The most striking finding was that the FW groups demonstrated significantly more hamstring activity, which will act to increase hamstrings-quadriceps co-contraction. Importantly, the observed differences of 3° between the two groups is similar to the differences previously observed between people with knee OA and healthy controls and therefore it seems possible that some of the previously observed changes lower limb muscle patterns may be due to altered upper body position.

SIGNIFICANCE/CLINICAL RELEVANCE: Inter-subject differences in sagittal trunk inclination appears to be associated with alterations in hamstring activity and hip moments. Future clinical management of people with knee OA may need to focus on improving upper body position during normal walking.

Appendix IX

Test-retest reliability study of gait kinetics and EMG

1.1 Introduction

Clinical gait analysis is like any clinical test which could be subjected to measurement error. Reliability is defined as a differences in measurement that taken by a single examiner under the same condition and separated by certain of time. Good reliability is needed to be able to test the hypothesis. Outcomes from the clinical analysis may be affected by many factors which could not be eliminated during collecting the data. It has been noted that positioning of the markers on bony prominences lead to increase the measurement error (A. Cappozzo, Catani, Leardini, Benedetti, & DellaCroce, 1996). In addition, Baker (2006) demonstrated that due to the adipose tissue, the palpation of bony prominences were difficult (Baker, 2006). Furthermore, markers position is an essential factor to determine the position of joint centre and it could lead to make error in calculation joints kinematics and kinetic if the markers positioned in wrong place (Baker, 2006; Stagni, Leardini, Cappozzo, Benedetti, & Cappello, 2000). Furthermore, it has been revealed that the EMG to be sensitive to many factors such the physiological cross talk and electrode site. Aforementioned factors may affect negatively on the quality of measuring the gait analysis and EMG. Therefore, it is essential to assess the test re-test reliability of gait analysis and EMG data.

1.2 Aim

This study is aimed to assess the test re-test of gait kinetics and muscle activities in adult healthy subjects between days. This is to ensure that able to accurately understand the effects of trunk lean on joint moments and muscle activation. In addition, this study to ensure consistency of results in time separated by one week. Prior to the study, procedures (as described in details in method chapter) were explained in detail to the participants, however participants were asked to come to the lab two separate time. Once they were happy, they signed an informed consent form.

1.3 Procedure

Five young healthy subjects (3 male. 2 female) visited the Podiatry lab two times with one week a part. Participant were recruited from Salford university. Participant inclusion and exclusion criteria in section 3.2.3 and 3.2.4 above were adopted in this study. Full lab protocol was applied as explain in details in 3.1. However, participants in this study were not required to perform the trunk lean conditions, only normal walking data were collected. The second visit (after the first visit by an interval of 5-8 days) was performed following the identical protocol. The intra-class correlation coefficients (ICC) were run to determine the test-retest reliability of gait kinetic and muscle activities between days for all subjects. The nearer the result is to 1, the greater the test-retest reliability (Growney, Meglan, Johnson, Cahalan, & An, 1997). When ICC used alone, it will not provide a full picture of the reliability (Munro, Herrington, & Carolan, 2012). Therefore, standard error of measurement (SEM) was calculated by the following equation:

SEM= pooled SD $\sqrt{}$ (1- ICC). As the pooled SD = $\sqrt{}$ (SD 1st visit)^2 + (SD 2nd visit)^2 / 2, with low values representing a good reliability (Cohen, 1988; Denegar & Ball, 1993).*

1.4 Result

The mean age, weight and height (mean ± stander deviation) of subjects was 26.5 ± 3.9 years, weight was 63 ± 7.9 kg and height was 1.70 ± 0.5 cm. Our result shows that the walking speed did not change significantly $p > 0.05$. ICC and SEM results for trunk angle, joint moment and muscle activation normalised by maximum isometric contraction (MVIC) are presented below.

1.4.1 Trunk angle and sagittal lower limb moment

Table 1-1 shows that the reliability result of the trunk angle the sagittal hip, knee and ankle moments during normal walking. The result showed that the trunk angle during normal walking was good reliable. In addition, the result showed that the sagittal hip, knee and ankle moments during normal walking was excellent and good reliable.

Table 1-1 The ICC and standard error of measurement (SEM) results of the trunk angle (over the full gait cycle) and lower limb joint moment (averaged between 15-25% of stance phase) during normal walking. (Trunk angle = º and Joint Moment (Nm/Kg).

	Mean (SD) 1st visit	Mean (SD) 2nd visit	ICC	SEM
Trunk angle	4.9 (1.8)	3.1 (1.1)	**.68**	0.848
Hip moment	.3 (.2)	.2 (.2)	**.91**	0.012
Knee moment	.6 (.2)	.7 (.3)	**.97**	0.043
Ankle moment	.0 (.1)	0 (.1)	**.96**	0.02

1.4.2 Muscle activation

Table 1-2 shows the ICC and SEM results of lower limb muscle activations (normalised by MVIC) averaged between 10-20% of stance phase (considering the EMD) during normal walking. As can be seen from Table 1-2, most of the ICC result showed that the reliability was excellent to good.

Table 1-2 The ICC and standard error of measurement (SEM) results of the muscle activation (normalized by MVIC) during normal walking.

	Mean (SD) 1st visit	Mean (SD) 2nd visit	ICC	SEM
Medial Gastrocnemius (MG)	.09 (.06)	.09 (.03)	**.70**	0.025
Lateral Gastrocnemius (LG)	.07 (.04)	.07 (.02)	**.90**	0.009
Vastus medialis (VMO)	.17 (.11)	.17 (.10)	**.92**	0.029
Vastus lateralis (VLO)	.17 (.11)	.20 (.11)	**.96**	0.020
semitendinosus (ST)	.08 (.03)	.07 (.03)	**.97**	0.005
Biceps femoris (BF)	.08 (.04)	.07 (.02)	**.86**	0.011

Appendix IIX

OARSI 2019

COULD INCREASED TRUNK FLEXION UNDERLIE ALTERATIONS IN KNEE MUSCLE ACTIVITY IN PEOPLE WITH KNEE OA?

Purpose:

There is now emerging evidence that people with knee osteoarthritis (OA) walk with an increased flexion of the trunk. This increased flexion may shift the position of the centre of mass relative to the hip, knee and ankle joint, which could in turn lead to changes in muscle activation patterns during walking. Interestingly, previous research has shown that people with knee OA walk with increased activity of the hamstring and quadriceps muscles during early stance. Modelling studies suggest that such altered muscle patterns may increase the loading on the joint, with imaging studies suggesting that co-contraction may accelerate cartilage loss. It is therefore important to understand if alterations in muscle coordination, associated with knee OA, result from an increased flexion of the trunk. This study had two aims. The first was to compare trunk inclination between people with knee OA and healthy control subjects. The second aim was to understand whether instructing healthy subjects to walk with a small increase in trunk flexion resulted in a change in muscle patterns towards those characteristic of people with knee OA.

Methods:

Kinematic, kinetic and EMG data were collected from 20 people with radiographically diagnosed osteoarthritis of the knee and 20 matched healthy participants. Each participant walked at their self-selected speed. In addition, the healthy individuals were instructed to walk with a 5° increase in trunk flexion using a real time biofeedback approach. For each condition, EMG data was collected from the medial/lateral hamstrings, medial/lateral quadriceps and medial/lateral gastrocnemius and normalised, for each muscle, using a maximal isometric contraction. Following data collection, trunk flexion angles and muscle activations were derived and then average values calculated over the period 15-25% of stance as this corresponds to a period of peak knee loading. Unpaired t-tests were then used to investigate differences in trunk flexion between the two groups and a paired t-test used to understand whether instructing the healthy participants to increase trunk flexion lead to a change in muscle activation over early stance.

Results:

Individuals with knee OA were observed to walk with 2.8° more trunk flexion ($p<0.05$). Specifically, the group with OA walked with a mean (SD) of 4.2 (1.9)° and the healthy individuals walked with a mean (SD) of 2.4 (2.8)°. Although there were minimal differences in the activation of the gastrocnemius muscles between the two groups, the biceps femoris and vastus medialis activations were significantly larger in the individuals with OA ($p<0.05$). Furthermore, there was a non-significant trend for both vastus lateralis and semitendinosus to be larger in the individuals with OA ($p=0.08$). Interestingly, when the healthy group were instructed to walk with 5° more trunk flexion, there was a 50% increase in biceps femoris ($p<0.001$, Figure 1a) and a 175% increase in semitendinosus ($P<0.001$, Figure 1b) activity over the period of interest. Furthermore, the peak in semitendinosus activity shifted to later in stance, matching the profile observed in the individuals with knee OA (Figure 1b). Although there were minimal changes in quadriceps activity, there was a pronounced increase in the activity of the two gastrocnemius muscles ($p<0.01$) when trunk flexion was increased.

Conclusion:

These data support the idea that people with knee OA walk with increased trunk flexion. The data also support the idea that increases in trunk flexion may be part of a mechanism which underlies the alterations in hamstring activity observed in individuals with knee OA. However, it is likely that the increase in quadriceps activity, associated with knee OA, is a result of a different mechanism. Nevertheless, future clinical interventions for knee OA could consider targeting trunk flexion during walking. If successful, this may reduce hamstring activity which may lead to a corresponding reduction in the compressive forces at the knee.