An Evaluation of Knee Joint Laxity, Mechanics and Muscle Activation Following Sustained Deep Flexion Kneeling

by

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Author's Declaration

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners. I understand that my thesis may be made available electronically to the public.

Daniel Mines

Abstract

Knee osteoarthritis (OA) is a complex disease with several proposed mechanisms for both the initiation and progression of the disease. Within the next 30 years, 1 in 4 Canadians are expected to have OA and 30% of the workforce will have difficulty performing occupational activities due to OA. One at-risk group is workers whose occupations require frequent and intermittent kneeling: habitual kneelers. To better our understanding of how knee OA is initiated in this population, biomechanical studies are needed to support or refute current hypothesized pathways that link occupational kneeling to knee OA. It is well documented that frontal plane knee laxity changes throughout the progression of knee OA but it is not known whether laxity changes are a cause or a result of the disease. This thesis work explores a laxity mechanism for knee OA initiation in habitual kneelers. Study 1 aimed to reliably capture frontal plane knee joint laxity using an improved device. Ten healthy, young participants volunteered (5 males, 5 females). ICC scores ranged from 0.95 to 0.99 suggesting excellent reliability of the device. An MDD of 1.22˚ was calculated and used to inform laxity decisions in Study 2. Study 2 was novel as it was the first to determine what changes occur in passive frontal plane knee joint laxity - in addition to gait mechanics and muscle activation - following a kneeling exposure. Fifteen healthy, young participants volunteered (8 males, 7 females). Contrary to what was expected, frontal plane knee joint laxity did not change following the kneeling exposure. However, during gait, knee flexion angle at heel contact and peak knee flexion angle during early stance phase were both affected by the kneeling exposure. These findings link kneeling exposure to immediate changes in measures indicative of knee joint instability and altered loading that have the potential to damage knee joint cartilage. Thus, the findings support the epidemiological evidence of a higher risk of knee OA development in habitual workers, though likely through some other mechanism than increased frontal plane knee joint laxity.

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List of Abbreviations

OA – Osteoarthritis

CCI – Co-contraction index

MVC – Maximum voluntary contraction

VL – Vastus lateralis

VM – Vastus medialis

LH – Lateral hamstring

MH – Medial hamstring

RF – Rectus femoris

FPL – Frontal plane laxity

KAA – Knee adduction angle

KFA – Knee flexion angle

KAM – Knee Adduction Moment

KFM – Knee flexion moment

NA – Net activation

ICC - Intraclass correlation coefficient

SEM – Standard error of measurement

MDD – Minimal detectable difference

LOA – Limits of agreement

EMG – Electromyography

ANOVA- Analysis of variance

Chapter 1
Introduction: Thesis Overview

Osteoarthritis (OA) is the most common form of arthritis and it affects 1 in 8 (13%) Canadians aged 15 or older (Bombardier, Hawker & Mayer, 2011). Within the next 30 years, 1 in 4 Canadians are expected to have OA and 30% of the workforce will have difficulty performing occupational activities due to OA (Bombardier, Hawker & Mayer, 2011). Osteoarthritis is costly to society. Over the next 30 years, direct costs, indirect costs and the total economic burden of OA will drastically increase. In 2010, the total economic burden of OA in Canada was estimated to be $27.5 billion, with direct costs of $10.2 billion and indirect costs of $17.3 billion. If no changes are made in prevention and identifying early risk factors, by the year 2040, the total economic burden of OA will be an estimated $1455.5 billion (Bombardier, Hawker & Mayer, 2011). At the individual level, OA is the most common cause of disability in Canada, with an individual suffering from pain, decreased quality of life, and decreased in functional abilities (MacDonald, Sanmartin, Langlois & Marshall, 2014).

Canadians are most frequently diagnosed with knee joint (29%) or hip and knee joint (29%) osteoarthritis, suggesting that nearly 50% of all OA diagnoses involve the knee joint (MacDonald, Sanmartin, Langlois & Marshall, 2014). Knee OA is a complex disease that is thought to have several biological, functional, and structural factors that influence its initiation and progression (Andriacchi, 2012). Both systemic (age, gender, genetics, and nutrition) and local mechanical factors (joint overuse, injury, obesity, muscle

weakness/coordination, laxity, alignment, posture) have been identified as risk factors for knee OA (Figure 1-1). Strong associations with both advancing age and obesity means that as Canadians age, there is a need for a better understanding of knee OA initiation mechanisms and strategies for individuals to manage their symptoms and progression of the disease (Toivanen et al., 2009; Felson, 2002). Additionally, for many people with symptomatic osteoarthritis, it has been found that it takes several years to get a diagnosis (MacDonald, Sanmartin, Langlois & Marshall, 2014). This finding is critical because the initiation phase of knee OA is the best opportunity we have to identify and modify early knee OA risk factors in these individuals. Although progression of knee OA has been thoroughly researched, it is evident that little research has focused on early, modifiable risk factors that may be present during the initiation phases of knee OA.

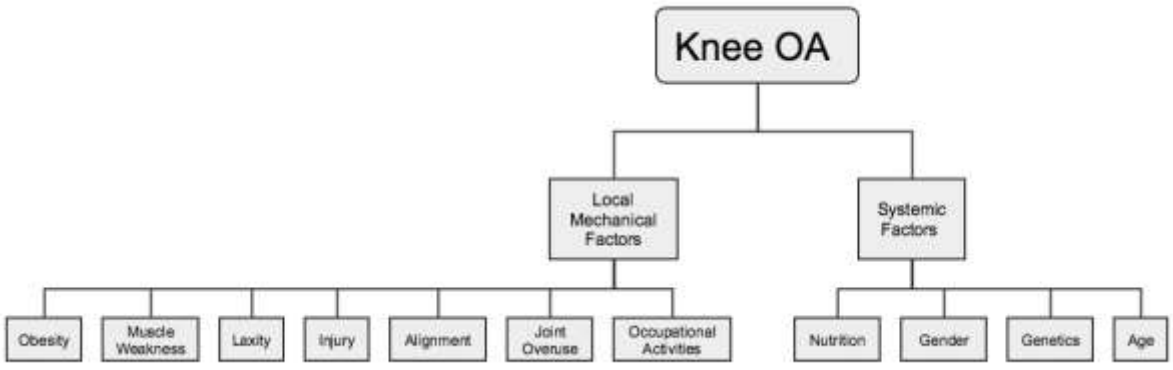


Figure 1-1: Risk factors of knee OA. Local mechanical risk factors are on the left, systemic factors on the right. Modified from Arden & Nevitt, 2006.

Recently, epidemiological studies have indicated that deep knee flexion activities such as kneeling and squatting at work are associated with increased rates of knee OA (Coggon et al, 2000; D'Souza et al., 2008; Muraki et al., 2009). These findings warrant the biomechanical study of these occupations and the daily occupational activities performed by workers. Kneeling (unsupported, supported, and sitting on heels) and squatting postures at work can involve a combination of sustained and intermittent periods of knee flexion greater than 90°. Examples of occupational kneeling tasks include installing tiles as a floor layer or laying bricks as a masonry worker (Kajaks, 2008; Jensen, Rytter & Bonde, 2010). One theory of knee OA development is that cartilage becomes conditioned to the loads it is exposed to, and then following abnormal stresses and strains on the tissues (which occur in kneeling), injury ensues due to an inability to respond appropriately (Andriachhi et al., 2004). Despite this epidemiological evidence that these workers are at increased risk for knee OA development, little research is being done to explore how occupational exposures may influence the development of knee OA in these workers. A pilot study by Kajaks and Costigan (2015) was the first study that explored the effects of prolonged kneeling on mechanics and neuromuscular measures, including knee flexion angles, external moments and muscle activation. While this study found differences in measures after prolonged kneeling was performed, their results were limited due to the inability to describe the mechanism through which these changes occurred. The authors theorized that knee joint laxity might be a key variable needed to explain the pathway to knee OA development for habitual kneelers, by linking kneeling to mechanical and neuromuscular changes. However,

laxity was not measured. Additionally, the sample only consisted of healthy, young males, but females also spend significant time in kneeling postures, during both activities of daily living and occupational activities.

Changes in frontal plane mechanics have been observed (Sharma et al., 1999; van der Esch, Steultjens, Wieringa, Dinant & Dekker, 2005) throughout the progression of knee OA (not necessarily in habitual kneelers), including increases in passive frontal plane laxity, and in frontal plane angles, moments, and muscle activation patterns during gait. It has been hypothesized that modifications in gait kinematics may make a habitual kneeling group more vulnerable to knee OA (Gaudreault, Hagemeister, Poitras & de Guise, 2013). Gaudreault et al. (2013) found that a habitual kneeling group, without knee OA, had altered knee adduction and flexion angles when compared to a healthy control group, suggesting that changes may occur due to cumulative kneeling exposures before disease progression. A summary of the hypothesized pathway to knee OA development through a laxity mechanism has been developed. Each element will be explored later in the literature review (Chapter 2) with the highlighted elements in Figure 1-2 being the focus of this thesis work. The elements that are not highlighted in the figure, measuring ligament creep, compression, and stability in the knee joint directly, and in turn, the consequences in terms of knee joint tissue integrity, are beyond the scope of this project.

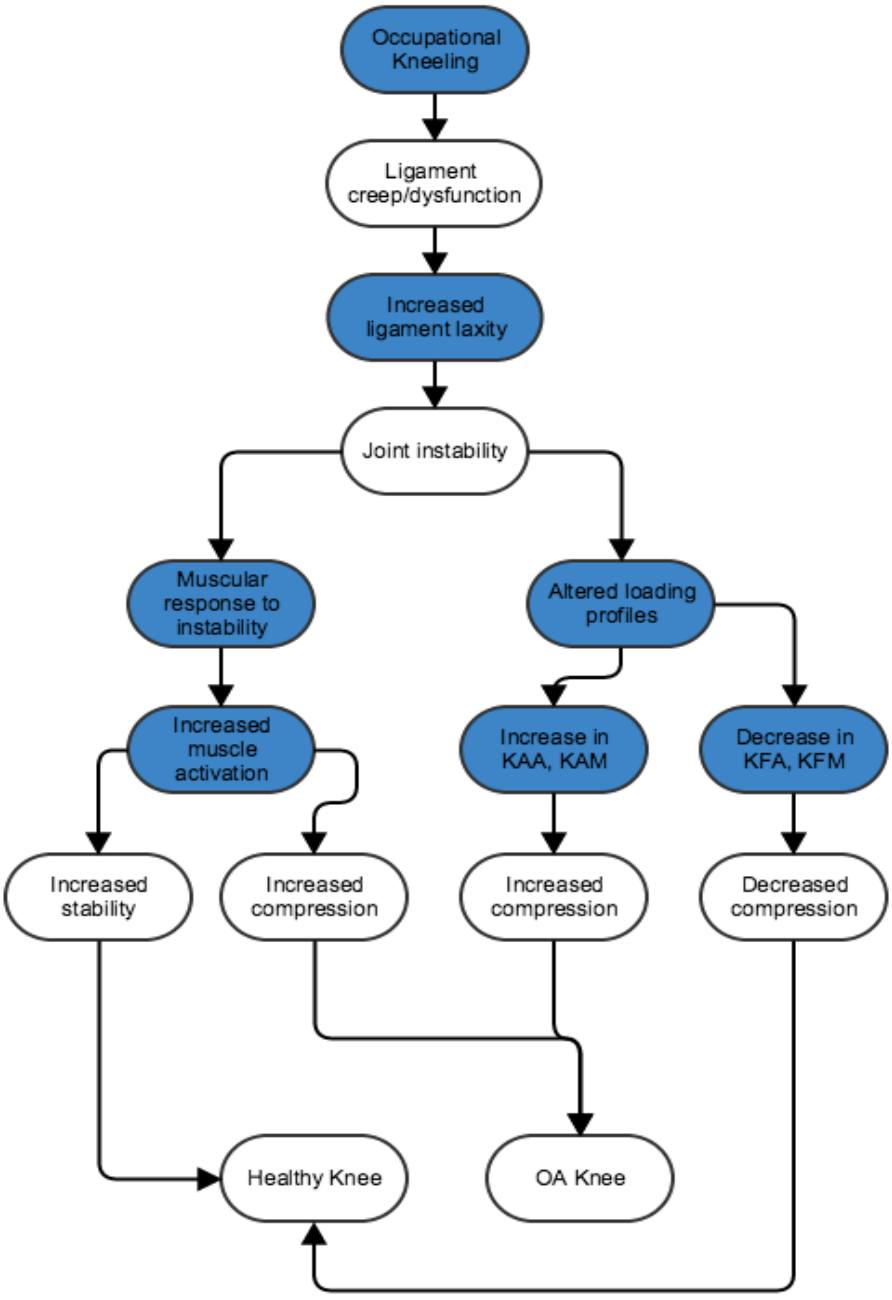


Figure 1-2: The proposed pathway to knee OA development through a laxity mechanism in habitual kneelers (Modified from Kajaks & Costigan, 2015). KAA = Knee adduction angle, KAM = Knee adduction moment, KFA = Knee flexion angle, KFM = Knee flexion moment.

The purpose of this investigation was to investigate whether an acute kneeling exposure could elicit any identifiable changes to passive frontal plane knee joint laxity, knee joint mechanics, or muscle activation. This was achieved by conducting two studies. Study 1 (Chapter 3) involved designing, creating, and testing an improved frontal plane knee joint laxity device. This device was then used in Study 2 (Chapter 4) to determine if frontal plane knee joint laxity increased after an acute 30-minute deep knee flexion exposure. Figure 1-3 shows the complete thesis study design. This thesis was designed for two separate manuscripts, and thus, Chapter 2 will read as a general literature review while Chapter 3 and Chapter 4 will read as separate, stand-alone papers.

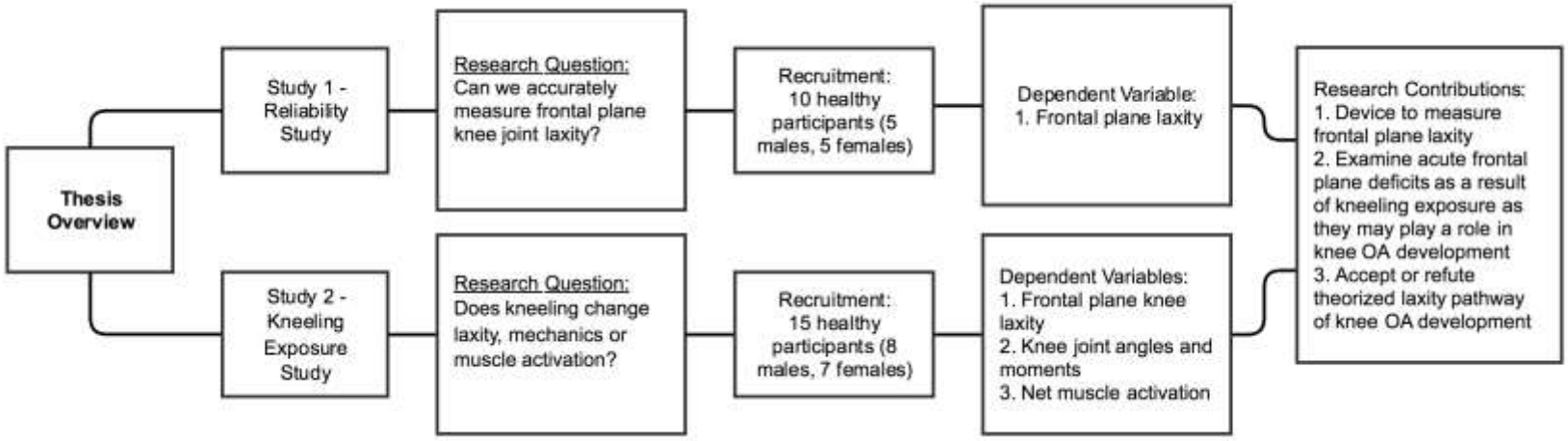


Figure 1-3: Thesis study design showing motivations, recruitment, dependent variables and contributions of each study

Chapter 2
Literature Review

This section will review studies supporting the theoretical framework behind increased knee joint laxity as a mechanism for knee OA development in habitual kneelers (Figure 1-2). First, section 2.1 shows the epidemiological data that demonstrates that habitual kneelers are an at-risk population for knee OA. Second, section 2.2.1 describes the literature to support that ligament dysfunction likely occurs immediately following an acute kneeling exposure, and could result in joint instability. Third, sections 2.2.2 and work together to describe how an individual's response to increased laxity could be to attempt to increase stability leading to abnormal loading, which may result in damage to articular cartilage. Section 2.2.4 describes how muscle activation is affected in this pathway, while sections 2.3 and 2.4 describe how muscle activation and laxity are important measures throughout the course of knee OA development and progression.

2.1 Occupational Risk Factors for Knee Osteoarthritis

Epidemiological studies have provided three main points of evidence which, when taken together, provide strong support for the relationship between occupation and knee osteoarthritis and provide motivation for the biomechanical study of high flexion occupational activities.

1. Specific occupations have been associated with knee osteoarthritis. While the relationships shown were strong statistically, the conclusions drawn were based on

specific job titles, not specific job tasks or postures, and therefore cannot be accurately interpreted from a biomechanical perspective (Felson, 1988).

2. More recent epidemiological studies have identified specific occupational activities, such as kneeling and squatting, that have been associated with knee osteoarthritis. This effort to isolate activities that are performed during a typical workday and to categorize time spent doing these activities is a crucial step that provides the impetus for analysis of these activities from a biomechanical perspective in an attempt to determine why they may be detrimental to the knee joint (Cooper et al., 1994; Coggan et al., 2000).
3. Although knee pain is not a direct indicator of knee osteoarthritis, it is thought to be an important risk factor for knee OA development (Robbins et al., 2011). In addition to the associations with the risk of knee OA specifically (see points 1 and 2), strong associations have been found between knee pain and occupation (O'Reilly et al., 2000).

One of the limitations that earlier epidemiological studies faced was having the workers recall how much time was required in postures such as standing, squatting and kneeling. In an attempt to eliminate this recall bias, Kivimaki, Rhiimaki & Hanninen (1992) used video analysis while participants were at work to categorize time spent in standing, kneeling, and squatting postures. This study compared an at risk group whose occupations (carpet and floor laying) required kneeling more often, to a group with a non-kneeling occupation (painting). They found that although the groups did not differ in time spent in

squatting postures, the carpet and floor layers spent 42% of their time in kneeling postures and their occupation was more at risk for knee morbidity and osteophytosis. These observations support the idea that increased kneeling time at work is related to knee osteoarthritis but this study does not provide a threshold for exactly how much daily kneeling is required, nor does it identify mechanisms of knee OA initiation or progression that are related to kneeling.

Previous studies have indicated occupational exposures based on low, moderate, or high levels while others have quantified exposures by asking workers how much time per day they spend in kneeling postures. Cooper et al. (1994) compared a group of individuals with painful, radiographic knee OA to age and sex matched controls and found a strong association (OR 3.4, 95%CI 1.3-9.1) between knee osteoarthritis and more than 30 minutes of daily kneeling at work. Coggon et al. (2000) confirmed that a daily duration of occupational kneeling over 1 hour nearly doubles the risk (OR 1.7, 95%CI 1.1-2.7) for knee osteoarthritis. The results of these two studies, along with similar findings in other studies, indicate that an at-risk kneeling group should be defined as workers who kneel at least 30-60 minutes daily (D'Souza et al., 2008; Muraki et al., 2009) but there is also evidence that workers must kneel at least 2 hours daily to see similar risk (Manninen et al., 2002). It should be noted that occupational squatting has also been linked to an increased risk of osteoarthritis as people that perform 1-3 hours of squatting at work have been found to be at double the risk for knee OA (Coggon et al., 2000; Zhang et al., 2004).

The results of these epidemiological studies strongly suggest that deep knee flexion activities at work are risk factors for knee osteoarthritis. It is likely that morphological, biomechanical and neuromuscular changes can occur due to prolonged or repetitive use of these postures. The aim of Study 2 (see Figure 1-3) was to determine if biomechanical and neuromuscular variables in gait differ following a strenuous kneeling activity. Possible mechanisms of how and why these changes can occur will be explored in subsequent sections of this review.

2.2 Etiopathogenesis of Knee OA

Over the past 25 years, the definition of knee OA has evolved as a more accurate understanding of the etiology and pathomechanics of the disease have been recognized (Brandt et al., 2008). Previous definitions of the disease focused on joint damage in general but more current definitions recognize knee OA as a failure of an organ (the synovial knee joint) comprised of many tissues and thus its causes are numerous. Although knee OA is a complex disease involving many systems, the purpose of this section will be to explain mechanically, at the tissue level, how the disease process may initiate to abnormal mechanics at the knee joint.

There are two distinct phases to knee OA: the initiation phase and the progression phase. It is believed that during the initiation phase, mechanical insults -either injury or abnormal loading- cause negative adaptations in soft tissues of the knee joint. Though it may be difficult to identify one initiator of knee OA, it has been hypothesized that the development of knee OA starts with an initiating mechanical insult to the joint (Radin et al.,

1991; Andriacchi et al., 2004; Brandt et al., 2008). Whether this insult occurs in the ligaments, cartilage or a combination of both, has yet to be determined. The mechanism proposed in this thesis work is through ligament dysfunction (Figure 1-2) and the following sections will describe how prolonged deep knee flexion postures *may* initiate this mechanism in knee joint tissues.

2.2.1 Joint Laxity with Deep Knee Flexion

There is little research related to ligament creep during deep knee flexion angles greater than 90˚. But, in order to understand how a ligament-based mechanism can lead to knee OA from sustained kneeling, it is important to review how ligament creep may occur from frequent and intermittent deep flexion kneeling. Ligament creep is induced by constant load causing exponential lengthening and consequently, a temporary form of laxity in the loaded ligament (Solomonow, 2004). Jackson et al. (2001) found that, following 20 minutes of prolonged lumbar flexion, changes in feline supraspinatus due to ligament creep had not returned to baseline conditions after 7 hours of rest. In fact, after the 7-hour rest period, the ligament had only returned to 79% of its pre-stretch tension. Although this animal model is not a direct comparison to the behaviour of a human knee joint, it gives an indication that joint stability may be compromised due to ligament creep from as little as 20 minutes of static kneeling. Maintaining joint stability is the primary role of ligaments and with deficiency, the joint may sublux and cause damage to the cartilage (Solomonow, 2004). The role of ligaments in joint stability during static deep knee flexion postures may be increased due to the fact that with these postures, it is likely that muscles play little role in stability

since little joint motion is required (Jackson et al., 2001; Wojtys et al., 1996) and, in most cases, net muscle activation in these sustained, static postures is very low (Tennant, Maly, Callaghan & Acker, 2014).

Previous literature has indicated that ligament dysfunction plays an important role in altering joint mechanics (Andriacchi & Dyrby, 2005; Chaudhari et al., 2008). Childs et al. (2004) proposed that the reduced flexion moment and flexion angles in OA groups are likely associated with a knee joint stabilizing mechanism to protect the joint in response to frontal plane laxity, functional instability and knee pain. Associated with the knee angle change, quadriceps and hamstrings activity increased – furthering support for this knee stabilizing mechanism (Childs et al., 2004). OA groups have also been shown respond to laxity by increasing co-contraction of muscle groups that cross the knee joint to increase joint stability (Lewek et al., 2005).

In an in-vitro study on the geometrical changes of knee ligaments during passive knee flexion, Belvedere et al. (2012) found ligament sub-bundles lengthen in the anterior cruciate ligament (ACL), medial cruciate ligament (MCL), and lateral cruciate ligament (LCL) and tighten in the posterior cruciate ligament (PCL) during deep flexion. Li et al. (2004) performed a cadaveric study examining in situ forces in ACL and PCL ligaments of the knee joint during simulated deep flexion kneeling. The ACL was found to be under increased tension at the start of knee flexion (<30˚), followed by a decrease in tension, then another increase in tension when reaching higher flexion angles of 150˚. The PCL was found to have peak forces at 90˚ of flexion postures. Another study supported that the MCL and LCL are

also under tension in deep knee flexion, in particular, the anterior portions of these ligaments are strained in deep knee flexion (Park et al., 2005). In vivo length change patterns have more recently been modeled for the MCL and LCL (Hosseini et al., 2014). This study supported the idea that the anterior portions of collateral ligaments have increasing length (up to 20% original length) with increasing flexion. If this strain is held constant for a period of time, it may induce ligament creep. These studies demonstrate that although kneeling is a sagittal exposure, and likely causes changes in the sagittal plane, tissues that support frontal plane stability (mainly the collateral ligaments) are also stretched during this posture and it is appropriate to expect that kinematic and kinetic changes will occur in the frontal plane.

Ligament deficiency is proposed to be a cause of laxity that is experienced during deep knee flexion postures, and habitual kneelers likely increase muscle activation to compensate for functional instability. While it may be suggested that increased co-contraction (and thus increased joint stability) may protect these workers from developing knee OA in the first place, it is also known that increased co-contraction results in increased joint compression forces (Childs et al., 2004), which has been associated with knee OA initiation and progression (see section 2.2.4). Additionally, although someone may increase co-contraction after kneeling to increase stability, they might never quite achieve the same stability as they had before performing a kneeling activity. This thought would support the theory that a change in location of loading may initiate the disease since an unstable joint (due to laxity and abnormal loading) could have a change in loading contact locations (see section 2.2.2). In the proposed study, frontal plane knee joint laxity will be measured since

frontal plane laxity and instability has been shown to be an integral part of the knee OA disease process (Lewek et al., 2004). The role of frontal plane knee laxity, specifically, in knee OA development is discussed in section 2.4.

2.2.2 Tibiofemoral Joint Contact Location, Area, and Stress during Deep Knee Flexion

It is believed that during the initiation phase, mechanical insults due to injury or abnormal loading cause negative adaptation in the articular cartilage fibers and matrix. Cartilage becomes exposed to abnormal joint loading when ligaments are injured or stretched which causes changes in knee motion that shift the typical load bearing contact location of the joint to a zone not conditioned to frequent load bearing (Andriacchi et al., 2004; Chaudhari et al., 2008). These new load bearing regions are not mechanically or structurally capable of withstanding the compressive or tensile forces they become exposed to and thus, they may fail under these new loading conditions (Chaudhari et al., 2008). These initial events may cause a cascade of biological, mechanical, and functional changes within the joint and initate the development phase of knee OA.

Currently it is unknown what maximum force articular cartilage can withstand before mechanical damage initiates knee OA and whether or not this maximal force threshold would be the same for different groups of people. For example, it may be possible that this threshold is different for two individuals of the same age, one who performs occupational kneeling and one who does not, or it could be different for two individuals of similar occupations but of different ages (Buckwalter, 2012; Seedhom 2006). Additionally, as the joint increases in

flexion angle, there are contact changes with regards to location, area and contact force magnitude both for the medial and lateral contact areas that need to be considered, as these are possible mechanisms for early degradation of the articular cartilage (Chaudhari et al., 2008).

Thambyah et al. (2005) investigated the contact stresses that are present during different loading conditions of walking and squatting in five cadaveric knees. They found that mean stresses increased by over 80% to 26.6 MPa for deep flexion loading conditions relative to stance phases of gait which averaged peak contact stresses of 14.1 MPa. These stresses, in part due to the increased force on the knee joint during high flexion activities, are concerning considering previous research has indicated that damage to the articular cartilage structures can be seen from cyclic and prolonged loading to the knee joint at impacts lower than the loads mentioned in the above study (Farquhar et al., 1996; Dekel et al., 1978). Walker et al. (2006) indicated that actual contact areas would depend not only on the changes in loading but the shape of menisci and deformation of the cartilage surfaces. It is quite possible that a combination of all three mechanisms - change in area, location and magnitude of load - are initiators of the disease. In this thesis, differences in contact area and loading location were represented by differences in knee joint kinematics based on skin-mounted motion tracking. Differences in knee joint adduction moments indicated changes in loading magnitude.

2.2.3 Why the changes in contact loading are detrimental to cartilage

Articular cartilage serves to minimize high joint contact stresses during loading and reduce friction at the joint during motion. Although damage thresholds for cartilage are not well established, negative adaption of cartilage to increased stress and changes in load direction and contact area (see section 2.2.2) is explained below, in terms of cartilage composition and structural organization.

In the 1970's, research began to categorize material and structural properties of articular cartilage. Water composes nearly 65 to 85% of the total weight for normal cartilage and is dispersed non-uniformly across the tissue (Maroudas, 1979). Higher cartilage hydration has been associated with higher permeability in the tissue (Maroudas, 1975). Maroudas (1975) found that once the cartilage hydration, expressed as a percentage of initial weight, dropped to 50%, there was visibly little to no permeability of the cartilage tissue. This is particularly concerning because high peak stresses (which occur during deep knee flexion activities) and high stress rates cause loss of water content and superficial layer cell death, all of which can be detrimental to the integrity of the cartilage and its function to disperse pressure across the tibial plateaus (Thambyah et al., 2005; Milentijevic & Torzilli, 2005). If cartilage loses hydration, it loses the ability to deform thus decreasing the contact area due to decreased joint conformity and increasing the stress on the area (Walker et al., 1972).

Kempson et al. (1970) were one of the first groups to correlate the stiffness of cartilage with the specific constituents of the cartilage. While water controls the permeability and viscoelastic properties of the cartilage, proteoglycans (PGs) and chondrocytes are two

structural components that control the load-carrying capabilities of the tissue. Using an indentation method, creep modulus of cartilage at 2 seconds was collected after each load application to eliminate the effect of thickness in the creep response. They found that as the glycosaminoglycan content increases, so does the creep modulus (compressive stiffness). The same positive relationship was not seen for increasing collagen content of the cartilage. These results indicated that PGs are responsible for biomechanical properties of cartilage under compressive loading. In a later study performed by the same research group, they showed that tensile properties of cartilage from the femoral head were associated with the collagen content and specifically, they found that parallel arrangement of fibers to the surface only in the superficial layer were associated with tensile and shear stiffness. Interestingly, this association was not seen in a perpendicular arrangement, nor was it seen in deep layers of cartilage (Kempson et al., 1970). The high-tensile-stiffness cartilage areas are along the periphery and as the knee approaches maximal flexion angles, the compressive load (contact area) shifts to these areas. Thus, the arrangements of the cartilage fibrils in the periphery are structured to resist tensile loads and not compressive loads, which could be detrimental to workers who repetitively perform deep knee flexion postures.

2.2.4 Muscle activation in Knee OA

Research on the muscle activation involved in the onset and progression of knee OA is important to identify whether patterns in lower extremity muscle activation differ between healthy populations and knee OA populations. To ensure normal knee joint stability and function, equilibrium between external forces and internal forces must be obtained (Bennell

et al., 2013). External knee joint loading is caused mainly by ground reaction forces and inertial characteristics of lower limb segments and by definition, is counteracted by internal structures such as muscles, ligaments, subchondral bone and cartilage (Bennell et al., 2013). It has been hypothesized that a loss of knee joint stability stimulates an increase in muscle activation but whether altered muscle coordination is a cause of knee OA or a consequence of knee OA is not well understood (Childs et al., 2004; Andriacchi, 2013). Understanding muscle activation patterns and the role of stability can provide further insight into how the knee is loaded during gait and kneeling, and aid in developing non-invasive measures which can be used in conjunction with radiographic scores to improve clinical classification of, and interventions for, those with varying severities of knee OA (Hubley-Kozey et al., 2009). In this thesis work, Study 2 (a non-OA cohort study), aimed to identify differences in muscle activation between pre and post-kneeling that could be used in the future as predictors of risk and as a foundation for prevention.

2.3 Muscle Activation Patterns during Gait Analysis in Knee OA

Much of the research done muscle activation associated with knee OA has been focused on patterns of activation (timing and amplitude) and co-activation of three lower extremity muscle groups: *quadriceps* (vastus medialis (VM), vastus lateralis (VM), rectus femoris (RF)), *hamstrings* (biceps femoris (LH), semitendinous (MH)) and *gastrocnemius* (lateral gastrocnemius (LG), medial gastrocnemius (MG)) (Childs et al., 2004; Hubley-Kozey et al., 2009; Zeni et al., 2010). These studies have indicated that there are alterations

in knee joint muscle activation patterns during gait and that some of these changes are consistent with increasing structural severity of knee OA.

The quadriceps have peak activation in the first 15% of the gait cycle, which aids in lengthening the limb to support the weight of the torso during the weight acceptance phase of gait (Winter & Yack, 1987). Appropriate eccentric loading of the quadriceps during this phase of the gait cycle serves as a protection mechanism to attenuate high impact loads that occur around heel contact (Bennell et al., 2013). In comparison to asymptomatic controls, studies have shown that individuals with moderate knee OA utilize higher quadriceps muscle activation throughout most of the gait cycle (Astephen et al., 2008; Hubley-Kozey et al., 2006). Hubley-Kozey et al. (2006) found that the amplitude of the VL and RF muscle activations were higher for an OA group compared to controls and that similar muscle activations occurred for VM. Additionally, Childs et al. (2004) found that quadriceps had longer durations of activity during stance. Hubley-Kozey et al. (2006) also found this trend in duration but specifically for VL and RF, which was thought to be a response to increase joint stability.

The roles of hamstrings during gait are to decelerate extension of the knee and prepare for initial loading and thus, the peak hamstrings activation occurs at the beginning and the end of the gait cycle (Yang & Winter, 1985). In comparison to asymptomatic control groups, moderate OA groups have higher amplitudes for LH during initial contact phase of gait (Hubley-Kozey et al., 2006). Initial joint changes due to mechanical insults may require the moderate OA group to unload the medial compartment during contact, and increasing LH

activity during this phase could potentially redistribute the contact force to the lateral side (Hubley-Kozey et al., 2006). Rutherford et al. (2013) found that prolonged activation of both hamstrings (especially LH) occurred during mid-stance in individuals with knee OA compared to the asymptomatic group, which is consistent with previous findings for similar participant groups (Hubley-Kozey et al., 2006). In the OA group, the role of prolonged activation during stance was thought to provide increased stiffness as the quadriceps also have prolonged activation during stance.

In knee OA groups, co-contraction indices (CCI) of lower extremity muscle pairs (both medial and lateral pairs) are increased throughout the gait cycle (Childs et al., 2004; Hubley-Kozey et al., 2009; Zeni et al., 2010). Knee joint instability or potentially, changes in mechanics at the hip or ankle, can elicit increased antagonistic muscle activity in individuals with knee OA (Zeni et al., 2010). Because there are many potential sources for altered co-contraction of muscles, it has been difficult to establish causal relationships between co-contraction and knee joint degradation. Hubley-Kozey (2009) investigated co-activation differences in lower limb muscles between asymptomatic (control), moderate knee OA and severe knee OA groups during gait. They found that differences in CCIs existed on lateral muscle pairs (VL/LH; VL/LG) for all three groups but differences in the medial muscle pairs (VM/MH; VM/MG) only occurred later in the disease process, between moderate and severe OA. This finding may indicate that intervention strategies to alter co-contraction should be different based on the knee OA severity level of the individual. An interesting paradox exists in studies that consider muscle co-activation strategies. While some believe that medial co-

contraction exists to increase stability of the joint due to structural changes in the medial compartment, this very strategy could potentially initiate mechanical changes because higher muscle co-contraction can lead to higher joint compressive forces (Lewek et al., 2005). Longitudinal studies are needed to assess how co-contraction indices change from healthy controls through to the development and progression of knee OA. While the current thesis work (Study 2) was cross-sectional in nature, it focused on variables during gait that are typically altered between asymptomatic controls and moderate knee OA groups. Because of the tendency for CCI to increase or decrease in knee OA groups, which in turn complicates interpretations of neuromuscular measures, net muscle activation was used as a surrogate measure of knee joint stability and total muscle activation during the loading phase of gait (Heiden et al., 2009).

2.4 Frontal Plane Laxity in Knee OA

Frontal plane knee joint laxity can be defined as the angular deviation of the tibio-femoral joint in the frontal plane following the application of a varus-valgus load (Sharma et al., 1999). In addition to this definition, there is a need to distinguish between passive and dynamic laxity. Passive frontal plane laxity implies that only the passive structures of the knee (ligaments, tendons, menisci) are contributing to the laxity measurement, while dynamic frontal plane laxity implies contributions from both passive and active structures of the knee joint. Passive laxity therefore should be confirmed by monitoring activation of muscles crossing the knee joint to ensure sufficient levels of muscle relaxation (less than 5% MVC) are met during laxity measurements.

Few studies have reported on passive frontal plane knee laxity despite it being considered an important risk factor by many in the knee OA community (Sharma et al., 1999; van der Esch, Steultjens, Wieringa, Dinant & Dekker, 2005; van der Esch, 2006, Shultz et al., 2007) (Table 2-1). Frontal plane laxity scores in both healthy and knee OA patients range from 2.0 – 19.5˚, with healthy participants having smaller frontal plane laxity deviations than knee OA groups (Sharma et al., 1999; van der Esch, Steultjens, Wieringa, Dinant & Dekker, 2005; Shultz et al., 2007). Frontal plane laxity in the knee joint has previously been reported to increase across knee OA grades (Sharma et al., 1999; van der Esch, Steultjens, Wieringa, Dinant & Dekker, 2005). In a knee OA cohort study, Sharma et al. (1999) showed frontal plane laxity increases across K/L grades, bone attrition grades, and minimal joint space width. In another study, van der Esch et al. (2005) found that both joint space narrowing and malalignment, but not osteophyte formation were related to frontal plane laxity. The highest reported frontal plane laxity difference between adjacent levels for one characteristic (narrowing, malalignment, etc) was 4.0˚, which occurred between levels 0 to 1 in the knee joint space narrowing characteristic of osteoarthritic knees. The fact that this occurred between levels 0 and 1 for joint space narrowing suggests that this difference is occurring at an early stage of knee OA.

Measuring frontal plane laxity requires high precision measurement with high reproducibility due to the small differences expected in varus-valgus deviation trials. Instrumented devices that measure knee laxity in the frontal plane are important because previous work has shown that laxity measurements by physical examiners have poor within-

observer agreement (ICC 0.55) (Cushnaghan et al., 1990). Sharma et al. (1999) was the first research group to develop a measurement system to capture frontal plane laxity. In a healthy control group, aged 20-40 years old, the mean frontal plane measurement was 2.9˚ (1.0). These FPL measurements are lower than reported by the more recent studies of van der Esch (2006) and Shultz et al. (2007) (Table 2-1). The differences in measurements between research groups are likely due to differing forces applied on the lower leg, demographics of samples, and device design.

Table 2-1: Summary of passive frontal plane laxity studies with healthy participants

Variable	Shultz et al. (2007)	Van der Esch (2006)	Sharma et al. (1999)
Number of Participants (N)	10	20	12
Sex	M/F	M/F	M/F
Population	University students	University students	Young controls
Laxity (˚)	9.6 (3.0)	5.9 (2.6)	2.9 (1.0)

There is a need to better understand how frontal plane laxity changes across both the development and progression of knee OA as patients with knee OA demonstrate both higher passive frontal plane laxity (Sharma et al., 1999; van der Esch, Steultjens, Wieringa, Dinant & Dekker, 2005) and higher external knee adduction moments (Lewek et al., 2004) than healthy controls. Study 1 of this thesis work (Chapter 3) was carried out to quantify the

reliability of the frontal plane laxity measurements that were used to address the first hypothesis in Study 2 (Chapter 4). This hypothesis focused on the first steps in the proposed pathway between kneeling exposure and knee OA initiation, which suggested that FPL would increase following a knee straining exposure (Figure 1-2).

Chapter 3 : Study 1 - Reproducibility of in-vivo frontal plane knee laxity measurements using an improved device and motion tracking in healthy participants

3.1 Introduction

Frontal plane knee laxity is measured as the angular deviation of the tibio-femoral joint with an applied varus-valgus load (Sharma et al., 1999). Measuring frontal plane laxity requires high precision and reproducibility due to the small differences expected in varus-valgus deviation trials. Instrumented frontal plane laxity measurement devices are required because laxity measurements determined by physical examination can have poor within-observer reliability (0.55, Cushnaghan et al., 1990) and the role of frontal plane laxity in knee OA initiation and progression is unknown (Chang, Lee, Zhao, Ren & Zhang, 2014).

Reproducibility refers to the ability to achieve similar scores on repeated measurements in an unchanging object or person (de Vet, Terwee, Knol & Bouter, 2006). When considering reproducibility, the measurements of reliability and agreement answer two different questions. Agreement describes how close the results of the measurements are within individual subjects by measuring absolute error in repeated measurements; this concerns measurement error. Reliability differs in that it is concerned with whether or not individuals within a group can be distinguished from each other despite measurement error; this concerns variability between study persons (de Vet, Terwee, Knol & Bouter, 2006; van der Esch, Steultjens, Wieringa, Dinant & Dekker, 2005). For joint laxity, agreement can be defined by the standard error of measurement (SEM), the minimal detectable difference

(MDD), and limits of agreement (LoA). Reliability is represented by intraclass correlation coefficients (ICCs) ranging from 0 to 1, where 1 represents perfect reliability (van der Esch, Steultjens, Wieringa, Dinant & Dekker, 2005). SEM, MDD and LoA are expressed in the metric unit of the measurement, degrees for this study. MDD values allow clinicians to assess if differences among measurements are meaningful. For example, if the difference between two measurements is greater than the MDD, that difference cannot be entirely attributed to measurement error (Roebroeck et al., 1993). LoA provide another way to ensure the differences in measurements between sessions are in an acceptable clinical error range (Portney & Watkins, 2000).

To our knowledge, few studies have measured reliability of frontal plane knee joint laxity measurement. Sharma et al. (1999), van der Esch et al. (2005) and Shultz et al. (2007) have all reported relatively good within-observer reliability; therefore, similar methodological and protocol considerations were used for this study. However, to improve the interpretability of the results, potential sources of error in previous methods were identified and then addressed in the design of the improved device for this study. Specifically, the new design addressed potential sources of error caused by frictional and gravitational forces, definition of knee joint center of rotation, and lack of muscle activation monitoring.

Specific purposes of the study were to:

I) Design a device that measures frontal plane knee joint laxity with good reproducibility. In particular, good within-observer agreement was needed to assess

potential laxity changes due to acute knee-straining exposures in future studies (including Study 2).

II) Improve on previous designs by addressing potential sources of error affecting the accuracy of laxity measurements.

3.2 Methods

This study was reviewed and received ethics clearance through the University of Waterloo Office of Research Ethics. Ten healthy participants (5 males and 5 females) provided written informed consent. Exclusion criteria included current pain in the lower limbs and previous lower limb injuries that required surgical treatment. These criteria were adopted due to their potential impact on knee joint laxity measurements (van der Esch, Steultjens, Wieringa, Dinant & Dekker, 2005). The age of the participants was also limited to 30 years, as age is known to affect frontal plane laxity scores (Sharma et al., 1999).

Electromyography (Wave Plus, Cometa, Citislano, Italy) of the dominant leg quadriceps (vastus lateralis, vastus medialis) (Appendix C: Electrode Placements) was sampled at 2048Hz with a built-in bandpass filter of 10-500Hz. EMG data were treated with bias removal, full-wave rectification and low-pass filtering at 6 Hz using a Butterworth filter (Winter, 1990). EMG data was amplitude normalized to maximum amplitude of the linear envelope of the quadriceps MVC exercise performed. Two MVC trials of 5-second duration were taken while the participant was seated in a knee extension machine with the knee flexed at an angle of 45 from full knee extension. EMG signals were real-time monitored for spikes and appropriate relaxation of muscle activation.

Kinematic data was collected at 64 Hz using an 18-camera Optotrak motion capture system (Northern Digital Inc., Waterloo, ON, Canada). Marker clusters, each equipped with 5 Optotrak smart markers in a non-collinear orientation, were placed on the participant bilaterally for the thighs, and unilaterally on the right shank and foot. Care was taken to ensure that marker clusters were placed in appropriate areas for visibility during knee laxity trials and minimum soft tissue deformation (De Rosario et al., 2012). The following landmarks were digitized (right leg only except for the thigh) to define segments: Thigh – greater trochanter, lateral epicondyle and medial epicondyles of femur; Shank – lateral and epicondyles of femur, medial and lateral malleoli; Foot – lateral and medial malleoli, calcaneus, 1st and 5th metatarsal heads. Registration and alignment was completed with a 16-marker cube over a 60 second calibration, force plate corners were digitized using the probe and were saved to be used for transformations between the force plate, segment, and global coordinate systems (Figure 4-2). All digitization was performed with the participant standing in the anatomical position. Knee angles were calculated (Visual3D, C-motion, Germantown, MD) following the ISB recommendations for the knee (Wu & Cavanagh, 1995) using a ZXY (flexion/extension – adduction/abduction – axial rotaton) Cardan sequence (Appendix D). Kinematic data were filtered using a dual pass 2nd order Butterworth low-pass filter at a cut-off frequency of 10 Hz (Kristianslund, Krosshaug & van den Bogert, 2012). Missing data points were interpolated using a third-order cubic spline in order to fit the missing frames of data up to a maximum of 10 frames (Howarth & Callaghan, 2010).

The frontal plane laxity measurement device consisted of four main parts: a chair with backrest; a free-moving arm (tibial sled) with Plexiglas base; zinc-plated ball bearings; and a Plexiglas surface/table (Figure 3-1). Multiple fixation sites and modalities aimed to minimize internal/external rotation of the lower leg and thigh (Figure 3-2). Frontal plane laxity was measured by applying a 2.28kg load, via a near-frictionless cable-pulley system, to the medial and lateral aspects of the tibial sled, until a steady moment of 10N.m was reached (Chang, Lee, Zhao, Ren & Zhang, 2014). The moment arm of the applied load was held constant for each participant at 0.45m by measuring the distance from the condylar clamps to the point of load application on the sled to ensure it did not change. The leg was re-positioned to neutral following each load application.

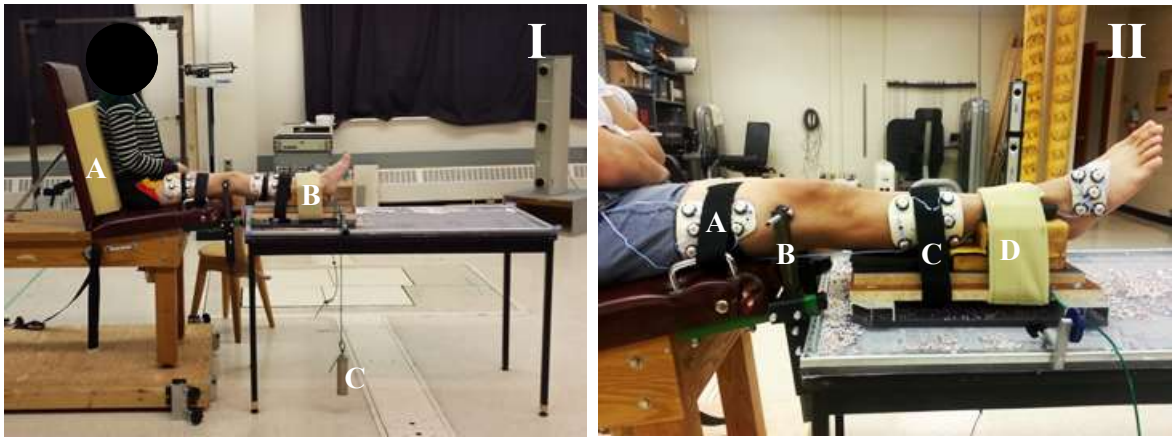


Figure 3-1: I) Frontal plane laxity jig set-up. A: Back rest; B: Free-moving arm; C: Dead weight pulley system **II) Fixation techniques for the assessment of knee joint laxity.** A: a Velcro bandage crossed the thigh distally above the knee joint; B: femoral condylar clamps steadied the femur; C: a Velcro bandage crossed the lower leg proximally below the knee joint; D: a 'v'-shaped clamp was used to secure the lower leg distally such that the Achilles tendon nestled deep into the v-shaped clamp. This design allowed for near-frictionless movement of the lower leg relative to the thigh.

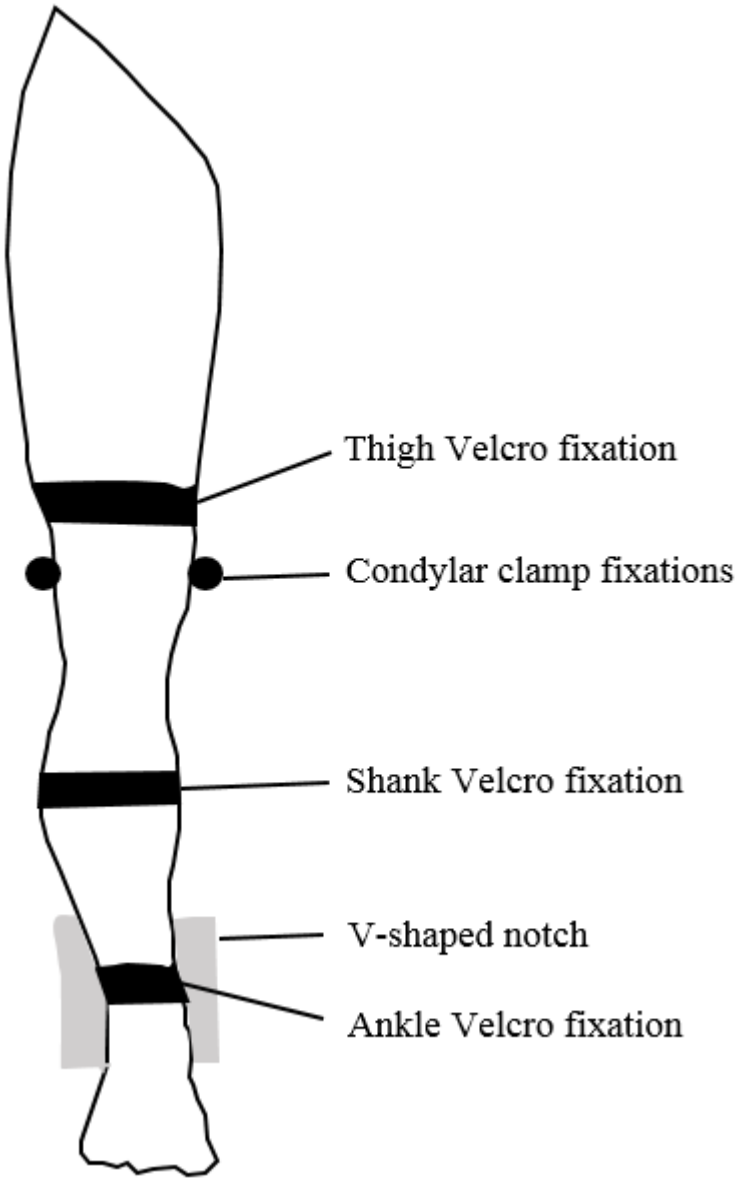


Figure 3-2: Birds-eye view of the fixation techniques including the v-shaped notch that the ankle nestled tightly in (chair, sled and table with ball bearings removed).

The experimental set-up was similar to that of Sharma et al. (1999) and van der Esch et al. (2005) except for four main changes that aimed to address potential sources of error in

the original designs. Firstly, the device was specially designed to be near-frictionless (Figure 3-1). The table-sled interface consisted of 4mm diameter zinc-coated ball bearings sandwiched between two layers of Plexiglas. Secondly, the device chair was tilted 20˚ below horizontal to maintain a knee flexion angle of 20˚ (Sharma et al., 1999; van der Esch, 2006). Since the tibia was horizontal, the effect of gravity on the moving segment (the tibia) was eliminated. Previous designs achieved the same flexion angle with the shank hanging from the device chair but a gravitational force, in addition to any applied load, would have contributed to the frontal knee angles achieved when load was applied. Thirdly, the knee was not forced to rotate about a fixed mechanical axis on the device, which allowed the knee to rotate about its natural, dynamic frontal plane knee joint centre. Lastly, previous work has suggested it is necessary to distinguish between passive laxity, with muscles relaxed, from dynamic laxity, with muscles active (Küpper, Loitz-Ramage, Corr, Hart & Ronsky, 2007). To our knowledge, no previous study has monitored activation of muscles crossing the knee to confirm sufficient relaxation (< 5% MVC) during laxity measurements. Therefore, MVC normalized muscle activity was monitored in this study (Carvalho and Callaghan, 2011).

The mean knee laxity was obtained from each of two measurement sessions within the same visit. There were three laxity measurements taken in each session (Figure 3-3).

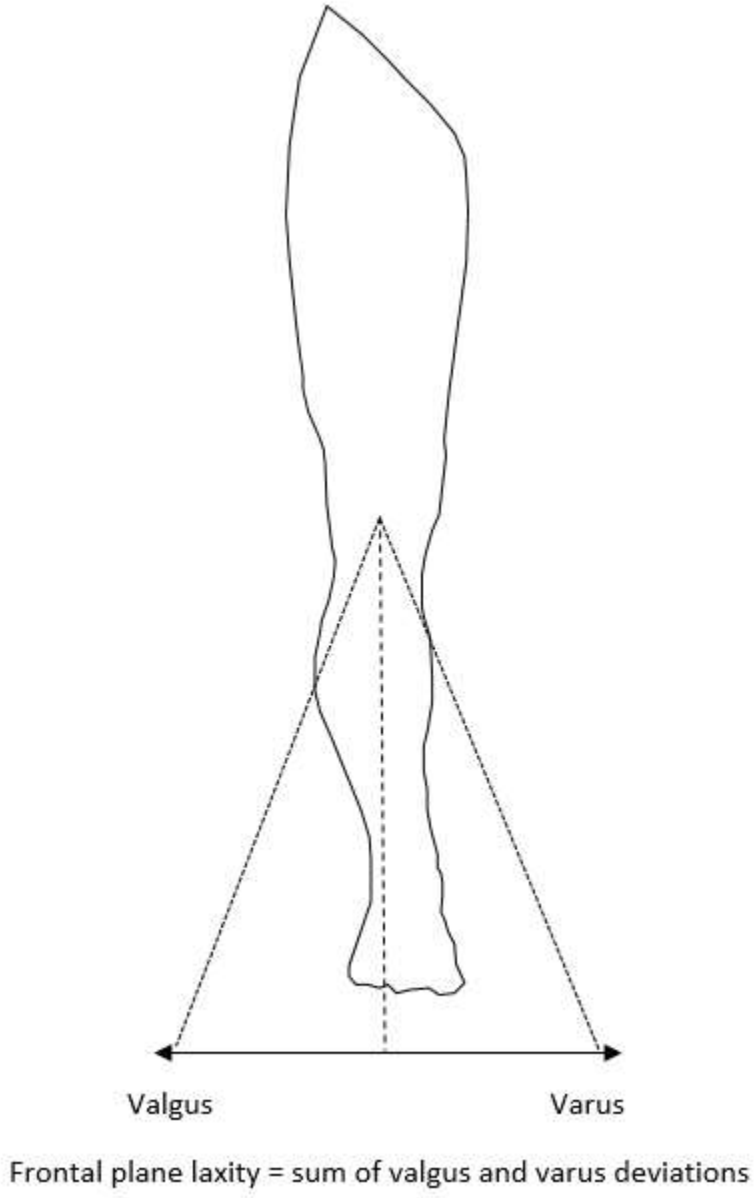


Figure 3-3: Birds-eye view demonstrating that load was applied in the valgus and varus directions and frontal plane laxity was calculated as the total of each deviation in a trial.

Following the first session, the experimenter marked the location of the condylar clamps, thigh and shank straps, and area where the shank made contact with the v-shaped clamp to assist in repositioning the participant into the device for the second set of measurements. In between measurements, the participant was seated in a chair directly beside the laxity device to eliminate any affect of movement on subsequent laxity trials. The participant then returned to the device, was repositioned to neutral via marked landmarks and pushing the top of the sled securely against the edge of the table. LoA were calculated for mean laxity between the two sessions. Intra-rater reliability was assessed using ICCs where a coefficient greater than 0.75 was considered excellent (Portney & Watkins, 2000). Within-session ICC scores (ICC_1, ICC_2) were expressed as the measured variance within one rater, across three measurements (model 3, 3). To obtain the between-session ICC_3, the three measurements in each session were used to express the measured variance within one rater across both sessions (model 3, 1) (Figure 3-4).

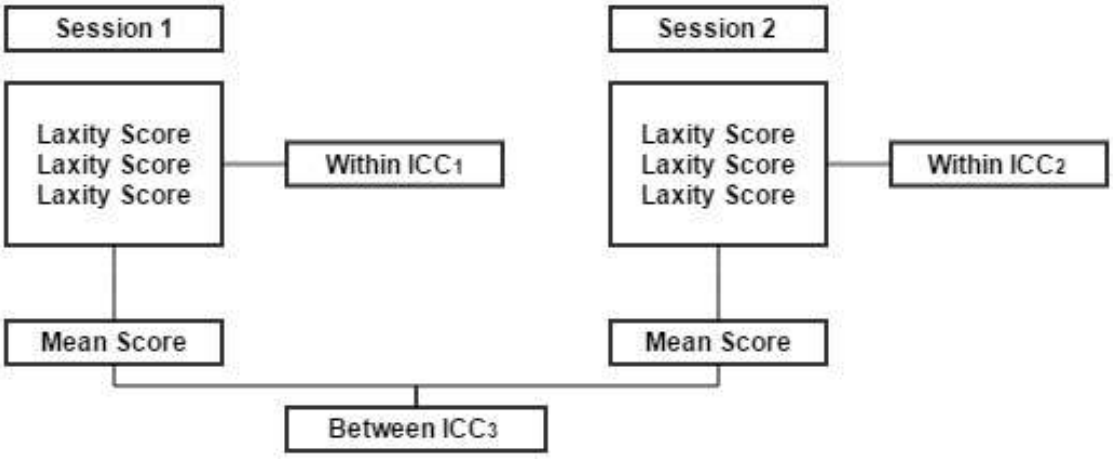


Figure 3-4: Experimental design including reliability and agreement parameters. One rater (a human movement scientist) performed all laxity measurements. The rater measured frontal plane laxity of the dominant leg of each participant in two sessions, each with three laxity measurements (total of six measurements per participant).

To obtain the SEM (Equation 3-1) and MDD (Equation 3-2) values, a univariate model of analysis of variance was performed. The model was used to estimate the absolute measurement error (error variance term) across sessions:

Equation 3-1

$$SEM = \sqrt{\sigma_e^2}$$

MDD was computed as the 95% confidence limit of the SEM using the following formula:

Equation 3-2

$$MDD = 1.96 \times \sqrt{2} \times SEM$$

The MDD was then used to represent the minimal change that could be interpreted as clinically relevant (Beckerman et al., 2001). LoA were calculated for mean laxity between the two sessions. All ICCs, the SEM, and the MDD were calculated using the Statistical Package for the Social Sciences (SPSS) version 21.0 (SPSS, Chicago, IL, USA).

3.3 Results

Study sample characteristics and reproducibility parameters were compared to previous devices (Table 3-1). For the one rater who performed all measurements, the mean knee laxities from the first and second sessions were 7.65˚ (2.4) and 7.68˚ (2.6), respectively. During all laxity trials EMG activity levels were confirmed to be <5% MVC. The rater's within-sessions ICCs were 0.95 (ICC_1 95% CI 0.87, 0.99) and 0.99 (ICC_2 95% CI 0.96, 0.99). The rater's between-session ICC_3 was 0.97 (95% CI 0.89, 0.99). The error variance term generated from the ANOVA results was 0.194 (Figure 3-5).

Measure: MEASURE_1

Source	Session	Type III Sum of Squares	df	Mean Square	F	Sig.
Session	Linear	.005	1	.005	.024	.879
Error(Session)	Linear	1.743	9	.194		

Figure 3-5: Error variance term from the statistical analysis used to calculate SEM and MDD.

Generalized across sessions for the same rater, the SEM was 0.44˚ and the MDD was 1.22˚ (Table 3-1). Agreement was initially assessed by plotting the means of each session against each other (Figure 3-6). To further compare to our results to Shultz et al. (2007), a limits of

agreement (LoA) analysis was performed on the mean laxity scores from session 1 and session 2 (Bland & Altman, 2007). The LoA provide an upper and lower limit within which 95% of differences between the laxity scores from sessions 1 and 2 can be expected to fall. The lower and upper limits of agreement were -1.27˚ and 1.21˚, respectively (Figure 3-7).

Table 3-1: Comparison of participant characteristics and statistical parameters for frontal plane laxity measurements in the literature.

Variable	Current Study	Shultz et al. (2007)	Van der Esch (2006)	Sharma et al. (1999)
Number of Participants (N)	10	10	20	12
Sex	5M/5F	5M/5F	10M/10F	M/F (distribution N/A)
Population	University students	University students	University students	Knee OA
Measurement method	Motion tracking	Motion tracking	Electrical Goniometer	N/A
Sessions	Same-day	Between-day	Between-day	Between-day
Laxity (˚)	7.67 (2.4)	9.6 (3.0)	5.92 (2.6)	2.9 (1.0)
Intra-rater reliability ICC (within-session)	0.95 to 0.99	N/A	N/A	0.85 to 0.96
Intra-rater reliability ICC (between-session) **95% Confidence Intervals**	0.97 Rater A: (0.89, 0.99)	0.96 N/A	0.84 to 0.93 Rater A: (0.61, 0.94) Rater B: (0.81, 0.97)	0.84 to 0.90 N/A
LoA (˚)	-0.03 ± 1.24	0.6 ± 2.7	N/A	
Intra-rater agreement				
SEM (˚)	0.44	0.67	1.35	N/A
MDD (˚)	1.22	1.86[b]	4.30	N/A

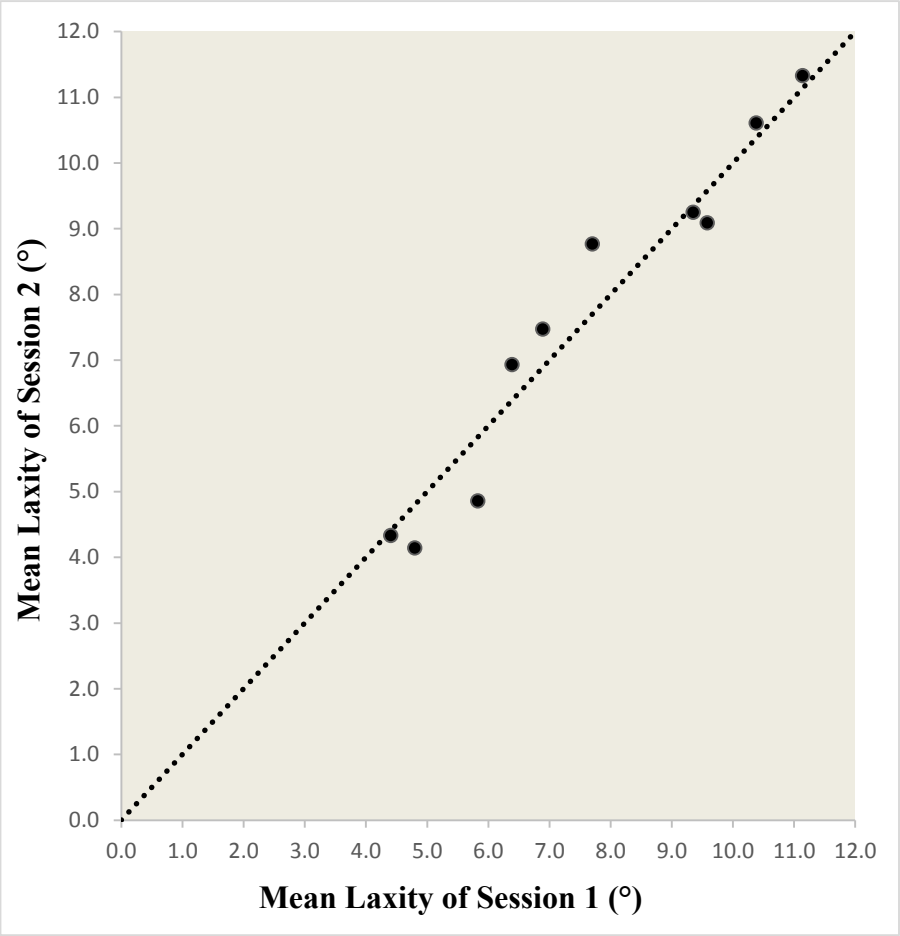


Figure 3-6: Comparison plot of mean laxity for each session. The dotted line represents 100% agreement of mean laxity between sessions for the rater. Each point represents one participant.

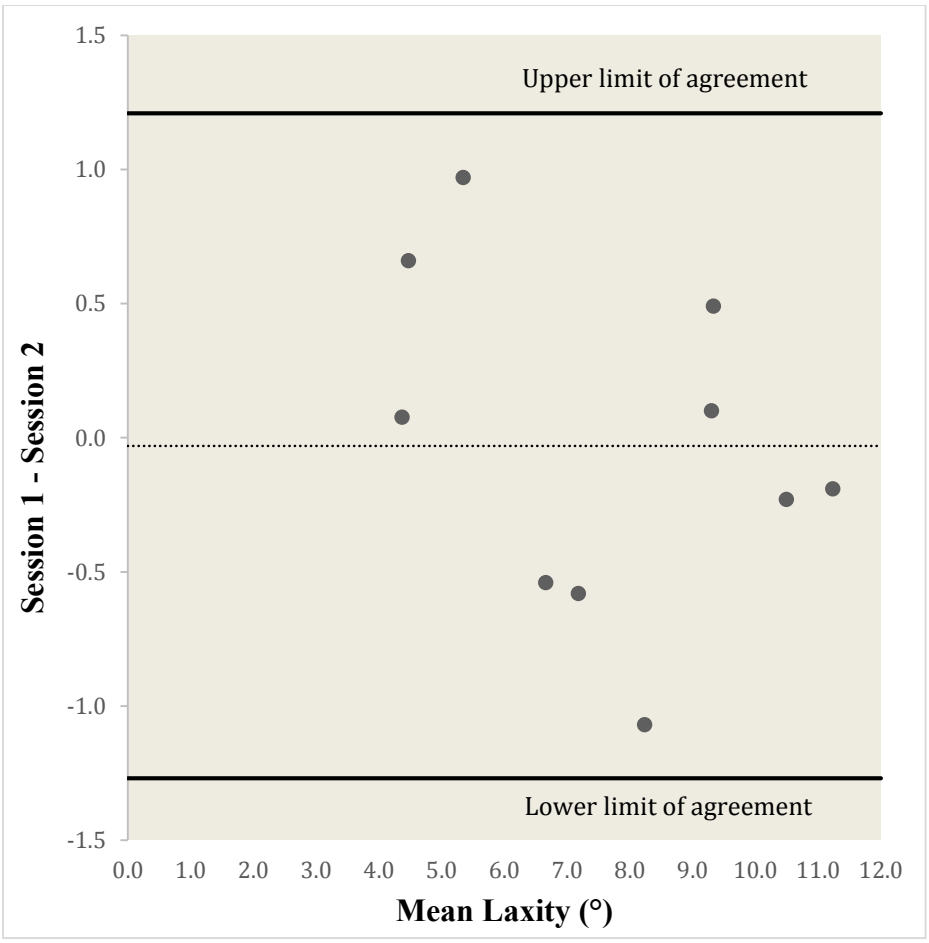


Figure 3-7: Difference between session 1 and session 2, plotted against the mean laxity for each participant. Dashed line shows the mean difference (-0.03˚). The solid black lines represent the 95% upper and lower limits of agreement (-1.27˚, 1.21˚).

3.4 Discussion

ICCs from the current study compared well to those of the previous reproducibility results found in literature (Table 3-1). Notably, the within-session ICC scores for the current study were higher than those reported by Sharma et al. (1999). The between-session reliability score of 0.97 in our study was comparable to that from Shultz et al. (2007) (0.96), and higher than those from van der Esch (2006) (0.84 to 0.93) and Sharma et al. (1999) (0.84 to 0.90). Therefore, the information in Table 3-1 serves as summary of study protocols and outcomes, but direct comparison between these studies is moderated by these differences in protocol.

Agreement parameters (SEM and MDD) were used to assess whether repeated measurements within an individual can be performed with minimal measurement error. The SEM and MDD values of 0.44˚ and 1.22˚ are lower than reported by Shultz et al. (2007) of 0.67˚ and 1.86˚ and considerably lower than van der Esch (2006) of 1.35˚ and 4.40˚. These findings suggest the method in this study could be more sensitive to changes due to an acute exposure (i.e. smaller differences could be detected with this device without attributing them to measurement error). The LoA of (-1.27˚, 1.21˚) are lower than the calculated LoA by Shultz et al. (2007) (-2.1˚, 3.3˚), suggesting they are in an acceptable clinical error range. Despite the fact that the purpose of each device was to measure frontal plane knee joint laxity, this was achieved through different designs, equipment, and loads. In addition to these differences, each group used different study samples. These recruitment differences would affect mean laxities and ICCs. In addition to all these factors, times between sessions were different between our study (same-visit) and the other studies (different-day). Same-visit study designs are useful to examine how knee-straining exposures may acutely change knee laxity, changes that might be missed with different-day designs. Other devices may also have excellent same-day reliability, but it has yet to be reported.

The reproducibility results confirm that the design considerations facilitated accurate and precise measurements. The dead weights, ball bearings and Plexiglas allowed for near-frictionless movement of the lower leg relative to the thigh. Tilting the chair allowed for the shank to remain horizontal while still achieving 20˚of knee flexion. This design removed the effect of gravity in the plane of measurement, which could have influenced laxity measurements in the other studies as they had the shank angled and hanging from an arm extending from the device chair. Finally, EMG was used to confirm that muscular activity was <5% of an MVC, a measure that has been used previously to classify a movement as passive (Küpper, Loitz-Ramage, Corr, Hart & Ronsky, 2007).

Although the design of this laxity device addressed potential sources of error in previous designs, there were some weaknesses in both the study protocol and the design of the device. Well-known limitations of using skin surface markers exist with digitizing landmarks and soft-tissue movement artifact, though given the small range of motion achieved; this may not have been a major concern. Using the same examiner to identify landmarks and the same protocol to reposition participants when they returned to the jig controlled these limitations. Performing the two sets of measurements within the same visit meant that error due to re-positioning of markers was eliminated. The use of one trained rater to perform all laxity measurements has previously been recommended, as intra-rater reliability is higher than inter-rater reliability (van der Esch, 2006). Finally, as a constant load is applied, the long axis of the tibia deviates from the original position. The maximum deviation that occurred in one direction, from any participant, was 7.0˚. Given the constant load of 22.36N and the moment arm of 0.45m, this small angular deviation of 7.0˚ resulted in a moment change from 10Nm initially to 9.98Nm in the final position.

The results of this study suggest that both within- and between-session ICCs were excellent (Portney & Watkins, 2000). Any difference between multiple, same-visit sessions of measuring frontal plane knee joint laxity measurements of 1.22˚ or greater can be identified as a change that cannot be solely attributed to measurement error. This MDD is smaller than previously reported, but it should be noted that the current study made changes to the design of the laxity device including a near-frictionless design and passive laxity verification and included highly accurate motion tracking with the goal of improving the MDD score. In the future, the reliability of the device should be tested in a multiple-visit study design to confirm adequate between-visit reliability. This consideration is important because in addition to being able to identify acute laxity changes (Study 2, Chapter 4), in the future, this device could be used to track laxity changes longitudinally in a clinical population across the course of knee OA. Although many knee OA models consider laxity as a factor that changes across the severity of the disease, it is currently unknown as to how it specifically plays a role in both initiation and progression of the disease. A better understanding of how laxity changes over time will aid in the identification of its role over the course of knee OA, which could assist in accepting or refuting current theories of knee OA initiation (Figure 1-3) and progression and guiding future prevention mechanisms.

Chapter 4: Study 2 - The Effect of Sustained Kneeling on Knee Joint Laxity, Mechanics and Muscle Activation

4.1 Introduction

The purpose of this study was to compare laxity, mechanics, and muscle activation before and after a kneeling protocol in gait. Chapter 2 explored structural, mechanical and physiological responses in the knee to gait and deep knee flexion activities, and discussed how these responses may put a habitual kneeler more at risk for developing knee osteoarthritis. Only one previous study by Gaudreault et al. (2013) has examined the effect of occupation (kneelers vs. non-kneelers) on adduction, flexion and internal rotation angles but their interpretations were limited to kinematics (they did not collect force plate data or measurements of muscle activity) during treadmill walking. Another study performed by Kajaks and Costigan (2015) studied a simulated occupational exposure to 30 minutes of static, full flexion kneeling and how it impacted mechanics and neuromuscular changes but that study did not measure knee joint laxity, which was central to the authors' proposed mechanism of knee OA in habitual kneelers. The current study was the first to look at the impact of 30 minutes of kneeling on knee joint laxity, mechanics and neuromuscular measures combined. It was hypothesized that sustained kneeling would compromise the integrity of the knee joint structures, increasing frontal plane laxity and changing ambulatory loading profiles. Differences between pre- and post-kneeling variables during gait analysis were used to explain how this occupational exposure might initiate knee OA in high knee flexion occupations.

The specific hypotheses that were tested were all with respect to the right tibiofemoral joint of the knee and are as follows:

1. *Exposure to 30 minutes of kneeling will cause an **increase** in frontal plane knee joint **laxity**.*

2. *Exposure to 30 minutes of kneeling will cause **increases** in known surrogate knee joint **stability** parameters in response to instability.*

    a. **Flexion angles** at heel contact and **peak knee flexion angles** during early stance will be **higher** post-kneeling than pre-kneeling (Gaudreault, Hagemeister, Poitras & de Guise, 2013), resulting in a lower flexion range of motion during gait. A higher knee flexion angle at foot contact has been demonstrated as severity of knee osteoarthritis increases. Overall decreased knee flexion throughout stance has been viewed as a strategy to protect the joint from pain particularly when combined with increased muscle activity (Childs et al., 2004). A reduced sagittal plane knee flexion/extension range has also been found in moderate knee OA patients (Childs et al., 2004; Zeni et al., 2010).

    b. **Peak flexion moment** will be **lower** in the early stance phase of gait post-kneeling than pre-kneeling. This variable is thought to be a consequence of both reduced sagittal plane flexion/extension range and increased muscle activation in response to instability.

    c. **Mean net muscle activation** across stance phase will be **higher** post-kneeling than pre-kneeling. Higher muscle activation has been found in moderate OA patients compared to healthy controls (Childs et al., 2004; Hubley-Kozey et al., 2009; Heiden et al., 2009); Zeni et al., 2010). Net muscle activation was calculated as an indicator of knee joint stability (Heiden et al., 2009).

3. *Exposure to 30 minutes of kneeling will cause a change in loading environment – location/contact area, or magnitude during the early stance phase of gait between pre- and post-kneeling measures.*

    a. **Adduction angles** at foot contact and peak adduction angle during early stance will be **higher** post-kneeling than pre-kneeling (Gaudreault, Hagemeister, Poitras & de Guise, 2013). Higher adduction angles are related to higher adduction moments, which may be indicative of greater medial joint loading (Gaudreault, Hagemeister, Poitras & de Guise, 2013).

    b. **Peak adduction moment** during early stance will be **higher** post kneeling than pre kneeling. A higher external knee adduction moment is associated with increased medial loading and knee OA development and progression (Lewek et al., 2004; Andriacchi et al., 2004; Astephen et al., 2008).

4.2 Methods

4.2.1 Study Design Overview

Each participant performed two sets of laxity measurements and two sets of gait trials; one of each before and after a sustained kneeling protocol (Figure 4-1). Each laxity and gait set consisted of 3 measurements. After the first set of gait trials and laxity measurements, participants underwent the sustained kneeling protocol. The protocol consisted of 3 cycles of ten minutes of sustained, full flexion kneeling. A 5 minute seated rest period was provided after each of the kneeling cycles to simulate a 2:1 work-rest ratio.

Figure 4-1: Experimental design of kneeling protocol

4.2.2 Participants

Eight males and seven females were recruited from the University population. Only right leg dominant participants were selected due to the current design of the laxity jig. The exclusion criteria included current pain in the lower limbs, previous lower limb injuries that required surgical treatment, and currently taking analgesics or anti-depressive medication. The age of the participants was limited to 30 years, as age is known to affect frontal plane laxity scores (Sharma et al., 1999). All of the above were designated as exclusion criteria due to the potential impact on knee joint laxity measurements (van der Esch, Steultjens, Wieringa, Dinant & Dekker, 2005). The study was reviewed and received clearance through a University of Waterloo Research Ethics Committee and all participants provided written informed consent. Participants wore shorts and t-shirts and were shoeless for all trials. Anthropometric data including height, weight, and age were recorded

4.2.3 Electromyography

Participants were then equipped with the surface electrodes for data collection. EMG locations were verified by systematically asking participants to contract their hamstrings and quadriceps to ensure proper location and functioning of the electrodes. Electrodes were placed on the right leg for the quadriceps (rectus femoris, vastus lateralis, vastus medialis) and hamstrings (biceps femoris, semitendinosus). All electrode sites were placed according to SENIAM guidelines (Appendix C: Electrode Placements) (Hermans et al., 1999), with an inter-electrode distance of 2 cm (De Luca, 1997). Maximum voluntary isometric contractions (MVCs) were performed to provide a reference for comparing EMG amplitudes between

muscle sites for normalization purposes. Two MVC exercises were performed to test hamstrings and quadriceps muscles respectively: a) Prone knee flexion at 115° knee flexion while lying on a massage table against fixed resistance; b) seated knee extension with the knee joint at an approximate angle of 45º of knee flexion from full extension against weighted resistance (Burden et al., 2003). Two trials of each MVC exercise were performed for 5 seconds with a 60-second rest period between each exercise. Electromyography was measured using a wireless amplifier system (Cometa, Italy) and sampled at 2048Hz with a built-in bandpass filter of 10-1000Hz.

4.2.4 Motion Tracking and Kinematics

Marker clusters, each equipped with 5 Optotrak smart markers in a non-collinear orientation, were placed on the participant bilaterally for the thighs, and unilaterally on the right shank and foot. Care was taken to ensure that marker clusters were placed in appropriate areas for visibility during deep knee flexion and minimum soft tissue deformation (De Rosario et al., 2012). The following landmarks were digitized (right leg only except for the thigh) to define segments: Thigh – greater trochanter, lateral epicondyle and medial epicondyles of femur; Shank – lateral and epicondyles of femur, medial and lateral malleoli; Foot – lateral and medial malleoli, calcaneus, 1st and 5th metatarsal heads. All digitization was performed with the participant standing in the anatomical position. Kinematic data was collected using an 18-camera Optotrak motion capture system (Northern Digital Inc., Waterloo, ON, Canada) at a sampling rate of 64 Hz. Knee angles were calculated (Visual3D, C-motion, Germantown, MD) following the ISB recommendations for

the knee (Wu & Cavanagh, 1995) using a ZXY (flexion/extension – adduction/abduction – axial rotaton) Cardan sequence (Appendix D). This rotation sequence was chosen to reflect ISB recommendations with the understanding that the first rotation is static so it likely did not affect the second rotation. If laxity was to be examined at multiple flexion angles, adduction/abduction should be the first rotation in the sequence.

4.2.5 Kinetics

Kinetic data was collected with four AMTI force platforms (Advanced Mechanical Technology Inc., Watertown, MA, USA) at a sampling rate of 2048 Hz. Force plate amplifiers were turned on a minimum of 4 hours before data collections and were zeroed upon the arrival of each participant. After registration and alignment was completed with a 16-marker cube over a 60 second calibration, force plate corners were digitized using the probe and were saved to be used for transformations between the force plate, segment, and global coordinate systems (Figure 4-2). All raw signals were collected using First Principles software (version 1.2.3).

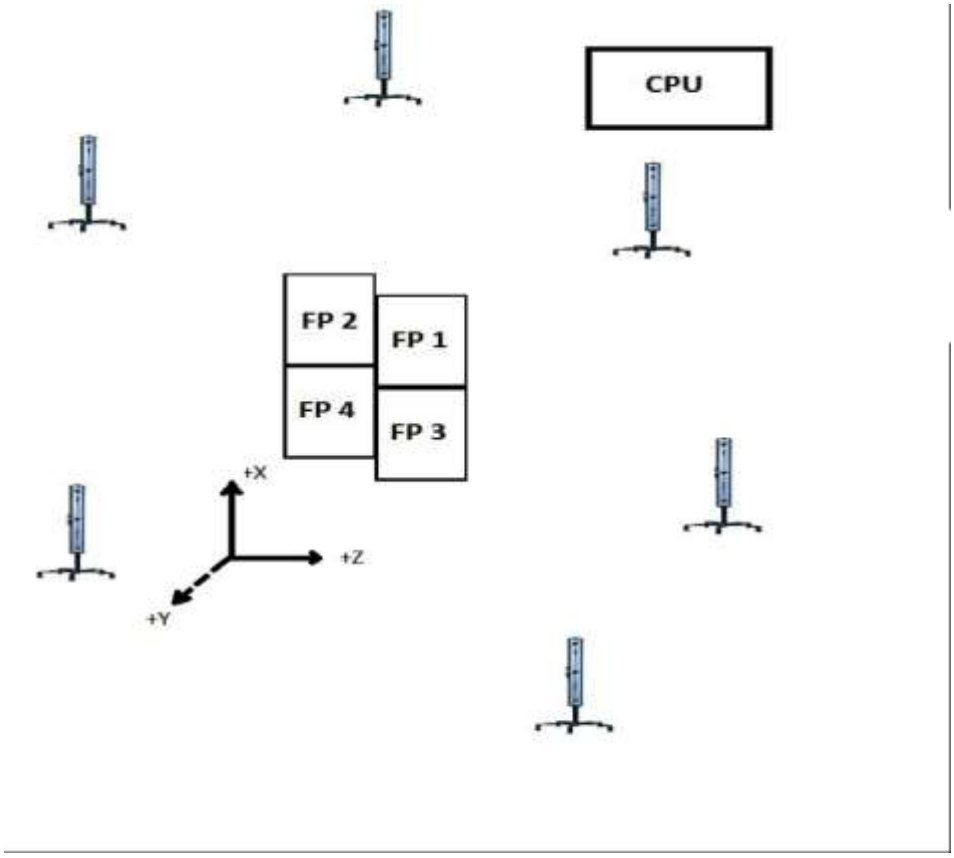


Figure 4-2: The laboratory set-up.

4.2.6 Standardized Gait Trials

Participants performed a minimum of 3 successful pre-kneeling and post-kneeling gait trials at their natural self-selected pace. Though studies have shown that gait velocity affects moments at the knee, this is less of a concern for a repeated measures design, and the decision to use a self-selected pace was made to capture the true events that occur after a sustained static kneeling exposure. Previous work has shown that within-subject waveform variance does not change within conditions, despite not controlling for gait velocity (Kajaks

and Costigan, 2015). A 9-meter walkway was used and a successful trial was one in which the participant's right foot landed entirely on any of the 4 force plates. The stride in which the participant's heel made contact with the force plate was analyzed for each gait trial. Each trial was recorded for 10 seconds and was monitored for marker visibility and EMG signal quality. Trials were excluded if there were more than 15 frames of consecutive missing data or spikes in EMG data representative of noise in the environment.

4.2.7 Standardized Laxity Measurements

Participants were seated in the laxity device and all fixation methods described in Chapter 3, Section 3.2 were implemented to ensure no axial rotation occurred at the shank, thigh or hip. A moment of 10N.m was applied in both directions in the frontal plane, and the total angular deviation in the frontal plane was recorded as frontal plane laxity for each trial. Each set (pre- and post-kneeling) consisted of 3 trials of total frontal plane laxity. The mean of the 3 trials in each set was used for analysis.

4.2.8 Kneeling Protocol

After the completion of the initial gait and laxity trials, the kneeling protocol was performed. Participants adopted a full flexion kneeling posture such that their buttocks rested just above or on their heels. They were not given directions on whether they should kneel with the foot in a dorsi-flexed (Figure 4-3) or plantar-flexed (Figure 4-4) position but rather were told to adopt a position where they felt both balanced and comfortable with their buttocks on the back of their heels. It should be noted that all participants chose to adopt the plantar-flexed posture. Foam padding was provided for the participants to place under their

ankles as they assumed the kneeling posture to cushion the top of the foot and prevent discomfort due to prolonged stretching of structures on the anterior side of the ankle. This was provided for every participant, and every participant used it to decrease discomfort during kneeling (Figure 4-4). Additional mats were placed on top of the force plates to minimize the pain from knee-ground contact forces that could have potentially impacted laxity or gait measures. This kneeling protocol was thought to be more strenuous on the passive tissues of the knee joint than hand supported kneeling, for example. If laxity changes were to exist, they should occur due to strain on ligaments that occurs in higher knee flexion angles.

Figure 4-3: Dorsi-flexed kneeling

Figure 4-4: Plantar-flexed kneeling

The kneeling protocol was adopted from Kajaks and Costigan (2015) in which participants in their study performed 30 minutes of static kneeling in 3 ten-minute bouts separated by five-minute bouts of rest. A work-to-rest ratio of 10 minutes of activity to 5 minutes of rest has been shown to induce ligament creep in feline supraspinous ligament in static flexion (Courville et al., 2005). Although the effects of this kneeling protocol were studied previously, the proposed mechanism by which altered stability and joint loading occurs is via joint laxity – a measurement that was not collected in the previous study (Kajaks and Costigan, 2015). Additionally, females were included since they also perform kneeling work and should not be excluded. This plantar-flexion kneeling posture was chosen

to capture ligament creep that may occur during the most extreme flexion angles, as opposed to adopting a posture that may more closely resemble an occupational kneeling exposure. Discomfort data was monitored every 5 minutes throughout the kneeling protocol as participants were asked to rate their levels of discomfort on a visual analog data sheet (Appendix A: Visual Analog Scale). During rest periods, participants were seated on a chair and were asked to sit with little movement of the lower limbs. The first two rest periods were 5 minutes in duration but the final rest period was only long enough to give 1 minute of static rest, and to verify all markers were securely fastened to the lower limbs and that EMG equipment was in working order. There were no cases where instrumentation verification took any longer than the 1 minute of static rest. During the final rest period, participants were asked to stand after 1 minute of rest so that visibility of markers and EMG signal quality could be checked. As soon as equipment was in proper order, post-kneeling laxity measurements were recorded and followed immediately by post-kneeling gait trials.

4.3 Data Analysis

Custom Matlab programs (Mathworks, Inc., Natick, MA) and Visual 3D pipelines (C-Motion Inc., Germantown, MD) were used to analyze raw kinematic, kinetic and EMG signals and output dependent variables. Kinematic and ground reaction force data were filtered using a dual pass 2nd order Butterworth low-pass filter at a cut-off frequency of 10 Hz (Kristianslund, Krosshaug & van den Bogert, 2012). Missing data points were interpolated using a third-order cubic spline in order to fit the missing frames of data up to a maximum of 10 frames (Howarth & Callaghan, 2010). Inter-segmental joint angle and

moment variables were calculated in Visual 3D software with a custom built pipeline. The automatic gait events function in Visual 3D software was used to identify gait events (heel contact and toe off), with the force threshold set to 20N. External knee moments were resolved in the tibia coordinate system (Mundermann, Dyrby, Hurwitz, Sharma, and Andriacchi, 2004) and magnitude normalized to % body weight multiplied by height to eliminate confounding effects of sex. Moments were time normalized to 100% of the stance phase of gait (Moisio, Sumner, Shott, & Hurwitz, 2003), with heel contact and toe-off representing 0% and 100% of stance phase respectively. Positive external moments in the sagittal plane represent flexion moments and positive moments in the frontal plane represent adduction moments. ISB recommendations were used to define the local knee joint coordinate systems as outlined in Wu et al. (2002) (Appendix D: Local Coordinate Systems for Lower Extremities).

All EMG data was treated with bias removal, full-wave rectification and low-pass filtering at 6 Hz using a Butterworth filter (Winter, 1990). EMG data was amplitude normalized to maximum amplitude of the linear envelope of the two MVC exercises performed for each muscle. Net muscle activation was determined for each leg by calculating the sum of all of the MVC normalized EMG signals, and used as an additional surrogate measure of total knee joint stability and generalized co-contraction. Net muscle activation was calculated for each gait trial and the mean value during the early stance phase of gait was calculated for successful trials.

A summary of the dependent measures that were extracted from each trial of the data set is summarized (Table 4-1). "Early stance phase" was defined as the first 50% of stance phase of gait.

Table 4-1: Dependent variables of interest for pre- and post- kneeling for both laxity and gait parameters

Parameter type	**Stage of Gait**	**Dependent Variables**
Hypothesis 1 (Increased Laxity)		Frontal plane laxity
Hypothesis 2 (Response to Instability)	Heel Contact	Flexion angle
	Early Stance Phase	Peak flexion angle Peak flexion moment
	Stance Phase	Mean net muscle activation
Hypothesis 3 (Change in loading environment – location/contact area, or magnitude)	Heel Contact	Adduction angle
	Early Stance Phase	Peak adduction angle Peak adduction moment

4.4 Statistical Analysis

Means and standard deviations for each kinematic, kinetic and neuromuscular outcome measure (see Table 4-1) were calculated for each trial, then averaged to get a pre-kneeling and post-kneeling mean for each participant. Laxity was represented by the mean of three frontal plane laxity measurements that were recorded during each pre- and post-kneeling measurement set. Statistical analyses were performed using SPSS software (Version 12.1). One-tailed paired sample t-tests on participant means were used to test for differences between pre- and post-kneeling outcome variables. Alpha was set to 0.05 prior to conducting the experiment. A Bonferroni correction for multiple comparisons was used, where the alpha level of 0.05 was divided by the number of comparisons to determine the corrected alpha significance level. This resulted in a corrected alpha level of 0.00625 (0.05/8 comparisons).

4.5 Results

The study sample consisted of 8 males (*Age*: 22.2 (2.2) years; *Height*: 1.74 (0.08) m; *Weight*: 77.8 (11.9) kg) and 7 females (*Age*: 22.8 (3.3) years; *Height*: 1.65 (0.05) m; *Weight*: 62.1 (6.9) kg). Two-way mixed ANOVAs were performed on a within factor of sex and between factor of time (pre vs. post. kneeling). There were no sex main effects or interactions, so one-tailed paired sample t-tests were performed for the main effect of time. Discomfort data was not a main outcome measure, but increases greater than 2cm from baseline were present in some participants (Appendix B: Kneeling Discomfort Data).

4.5.1 Laxity Response

There was no significant difference in frontal plane laxity scores between pre-kneeling (M=8.46°, SD= 3.9°) and post-kneeling (M=8.09°, SD=3.5°) conditions (p=.0685). Only 3 of 15 participants (P4, P5, P7) (Figure 4-5) displayed laxity changes greater than the MDD of 1.22° (Table 3-1). Additionally, all 3 participants were females and all 3 exhibited a decrease in laxity post-kneeling – a change in the opposite direction of that hypothesized (Figure 4-5). This finding rejects hypothesis 1, which stated that mean frontal plane knee laxity would increase in response to the kneeling exposure.

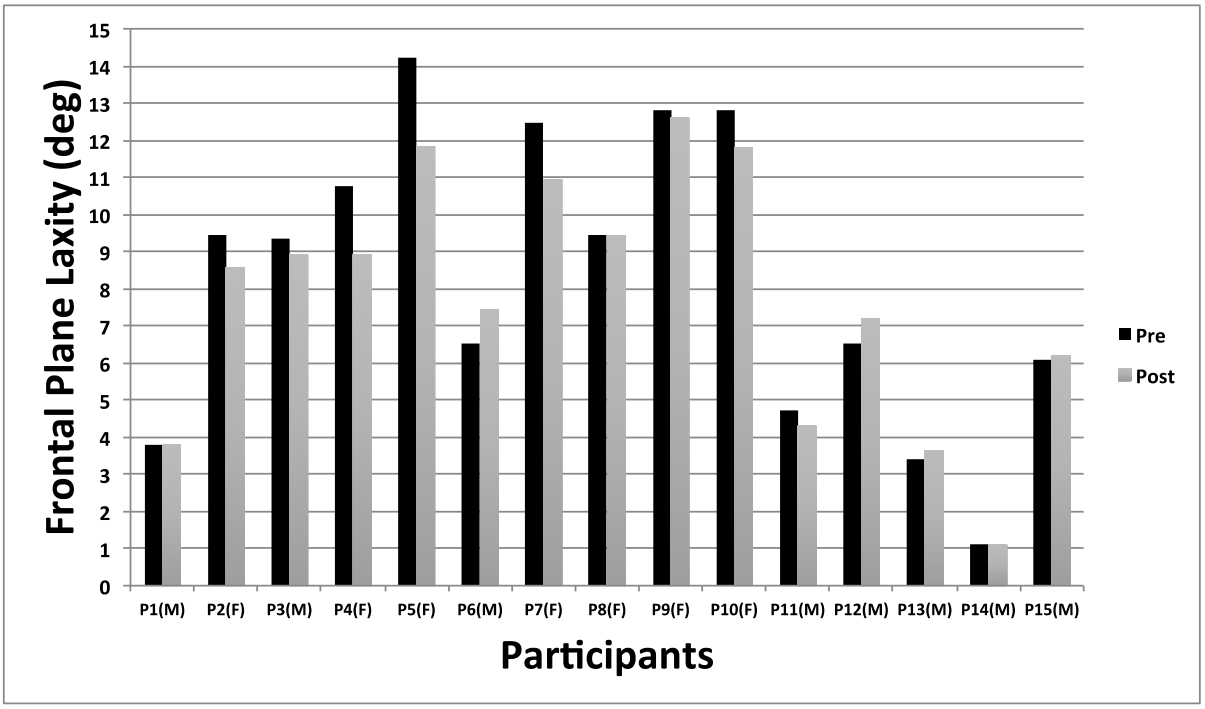


Figure 4-5: Frontal plane laxity of each participant pre- and post-kneeling.

4.5.2 Response to instability

A paired samples t-test was conducted to compare knee flexion angles at heel contact for pre- and post-kneeling conditions across participants (Figure 4-6). There was a significant difference in knee flexion angle at heel contact for pre-kneeling (Mean=10.8°, SD=5.4°) and post-kneeling (Mean=8.9°, SD=5.0°; p=0.0015) conditions (Figure 4-7).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreKFAHC	8.9040	15	4.99539	1.28980
	PostKFAHC	10.7718	15	5.37616	1.38812

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreKFAHC & PostKFAHC	15	.929	.000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	PreKFAHC - PostKFAHC	-1.86781	1.99213	.51437	-2.97101	-.76460	-3.631	14	.003

Figure 4-6: Paired t-tests results for differences in knee flexion angle across participants at heel contact pre- and post-kneeling. The p-value was divided by 2 to reflect a one-tailed test which was dictated by the structure of hypothesis #2a.

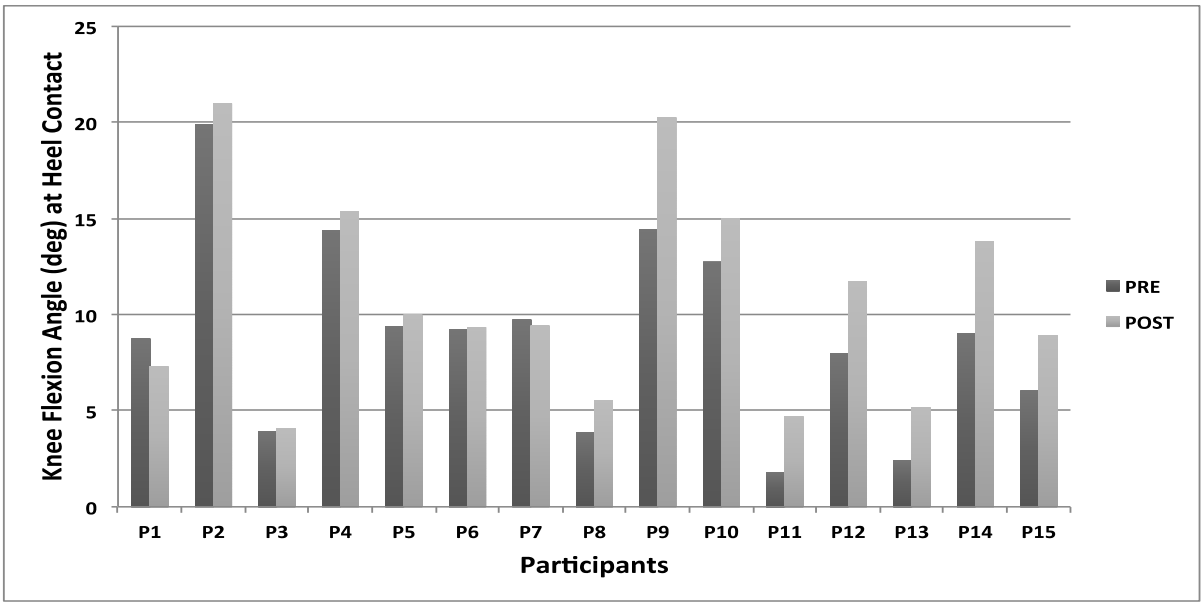


Figure 4-7: Mean knee flexion angles at heel contact for pre- and post-kneeling for each participant.

A paired samples t-test was conducted to compare peak knee flexion angles during early stance for pre- and post-kneeling conditions across participants (Figure 4-8). There was a significant difference in peak knee flexion angle during early stance between pre-kneeling (Mean=19.9°, SD=6.5) and post-kneeling (Mean=21.8°, SD=6.4; p=0.0025) conditions (Figure 4-9). This finding (Figure 4-8), along with results in (Figure 4-6), leads to acceptance of Hypothesis 2a, which stated flexion angle at heel contact would increase and that the peak flexion angle throughout early stance would increase in response to the kneeling exposure. In addition to the discrete measures (knee flexion angle at heel contact and peak knee flexion angle during early stance), which were compared statistically, the mean knee flexion curves for the entire stance phase are shown in Appendix E. A qualitative comparison of these curves

shows an increased knee flexion angle throughout early stance phase (0% to 50% stance phase) after the kneeling exposure.

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreKFAMax	19.9067	15	6.45847	1.66757
	PostKFAMax	21.7884	15	6.38181	1.64778

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreKFAMax & PostKFAMax	15	.941	.000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	PreKFAMax - PostKFAMax	-1.88171	2.19882	.56773	-3.09937	-.66404	-3.314	14	.005

Figure 4-8: Paired t-tests results for differences in peak knee flexion angle during early stance across participants for pre- and post-kneeling. The p-value was divided by 2 to reflect a one-tailed test which was dictated by the structure of hypothesis #2a.

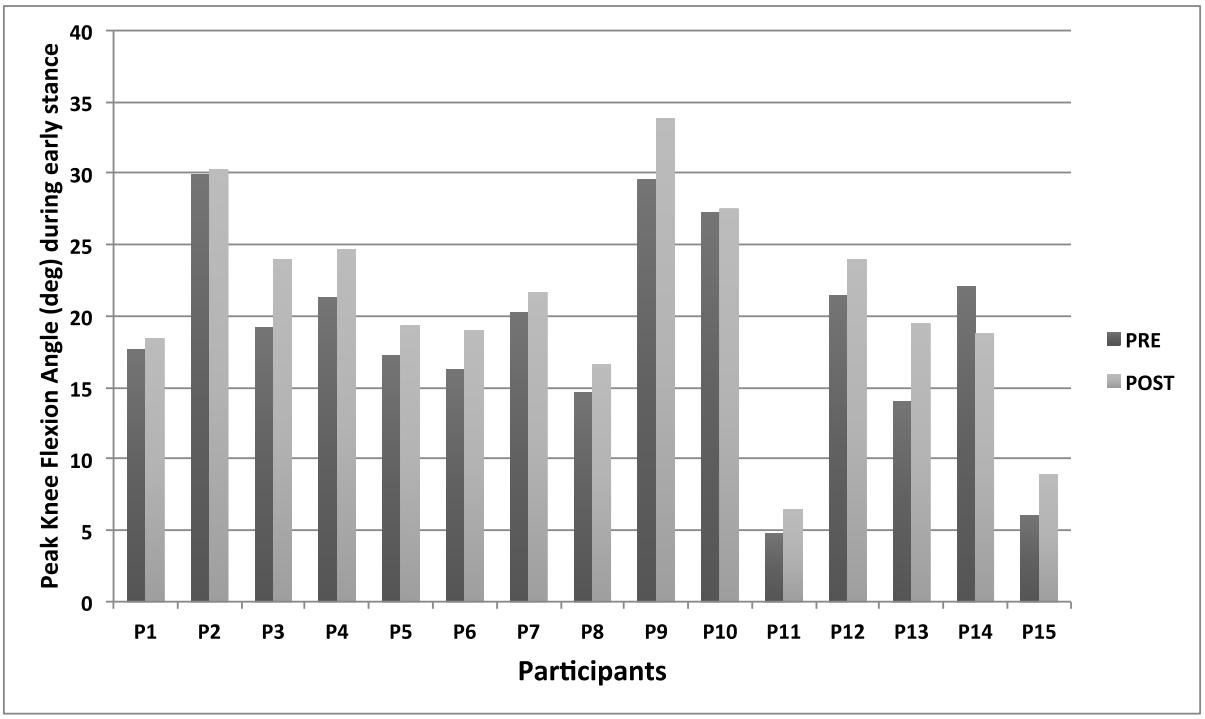


Figure 4-9: Peak knee flexion angles during early stance for pre- and post-kneeling for each participant.

A paired samples t-test was conducted to compare peak knee flexion moments during early stance for pre- and post-kneeling conditions across participants (**Figure 4-10**). There was no significant difference in peak knee flexion moment for pre-kneeling (Mean=2.9%BW*Height, SD=1.5%BW*Height) and post-kneeling (Mean=3.1%BW*Height, SD=1.3%BW*Height; p=0.1685) conditions (Figure 4-11). This finding leads to the rejection of Hypothesis 2b, which stated that peak knee flexion moment during early stance would decrease in response to the kneeling exposure.

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Pre1stPeakMax	2.8963	15	1.49834	.38687
	Post1stPeakMax	3.0602	15	1.32392	.34183

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Pre1stPeakMax & Post1stPeakMax	15	.905	.000

Paired Samples Test

		Paired Differences					t
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference		
					Lower	Upper	
Pair 1	Pre1stPeakMax - Post1stPeakMax	-.16386	.63787	.16470	-.51709	.18938	-.995

Paired Samples Test

		df	Sig. (2-tailed)
Pair 1	Pre1stPeakMax - Post1stPeakMax	14	.337

Figure 4-10: Paired t-tests results for differences in peak knee flexion moment during early

stance across participants for pre- and post-kneeling. The p-value was divided by 2 to reflect a one-tailed test which was dictated by the structure of hypothesis #2b.

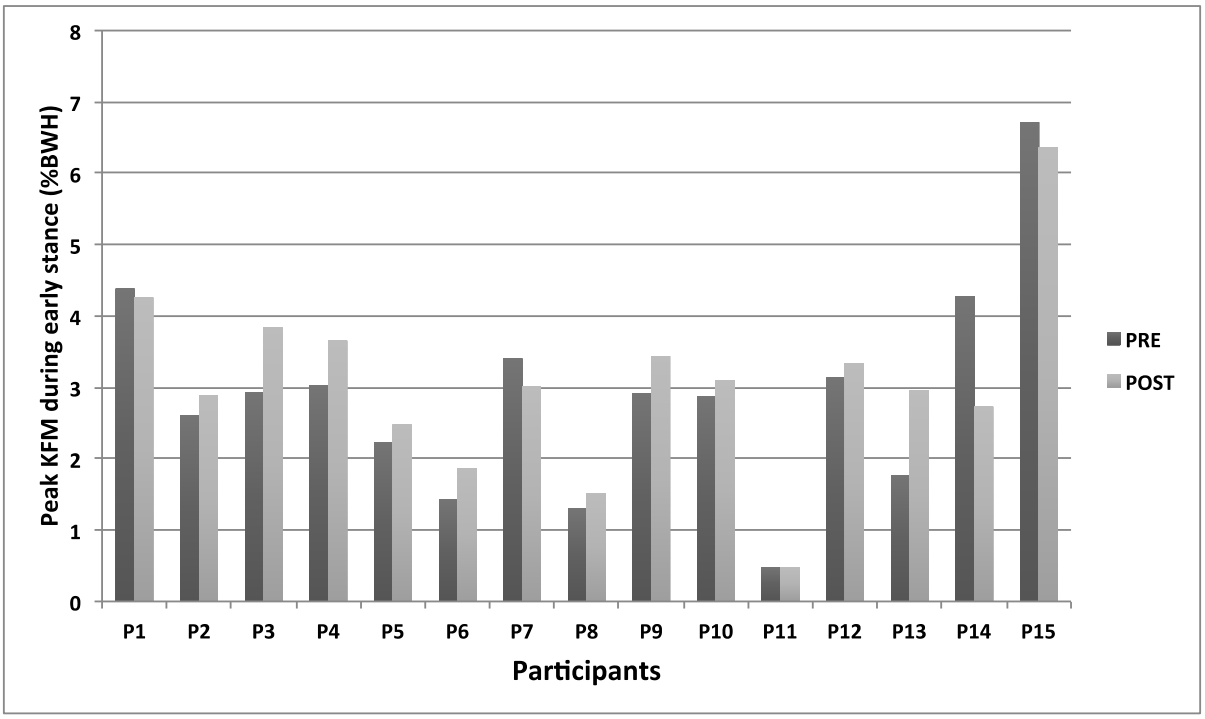


Figure 4-11: Peak knee flexion moments during early stance for pre- and post-kneeling for each participant.

A paired samples t-test was conducted to compare mean net muscle activation during stance for pre- and post-kneeling conditions across participants (Figure 4-12). There was no significant difference in mean net activation between pre-kneeling (Mean=31.0, SD=8.2) and post-kneeling (Mean=31.6, SD=9.2; p=0.251) conditions (Figure 4-13). This finding leads to the rejection of Hypothesis 2c, which stated that net muscle activation would increase in response to apparent knee joint instability caused by the kneeling exposure.

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreAvgNA	31.0096	15	8.18663	2.11378
	PostAvgNA	31.5638	15	9.19900	2.37517

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreAvgNA & PostAvgNA	15	.942	.000

Paired Samples Test

		Paired Differences					t
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference		
					Lower	Upper	
Pair 1	PreAvgNA - PostAvgNA	-.55419	3.11216	.80356	-2.27764	1.16927	-.690

Paired Samples Test

		df	Sig. (2-tailed)
Pair 1	PreAvgNA - PostAvgNA	14	.502

Figure 4-12: Paired t-tests results for differences in mean net muscle activation during stance across participants for pre- and post-kneeling. The p-value was divided by 2 to reflect a one-tailed test which was dictated by the structure of hypothesis #2c.

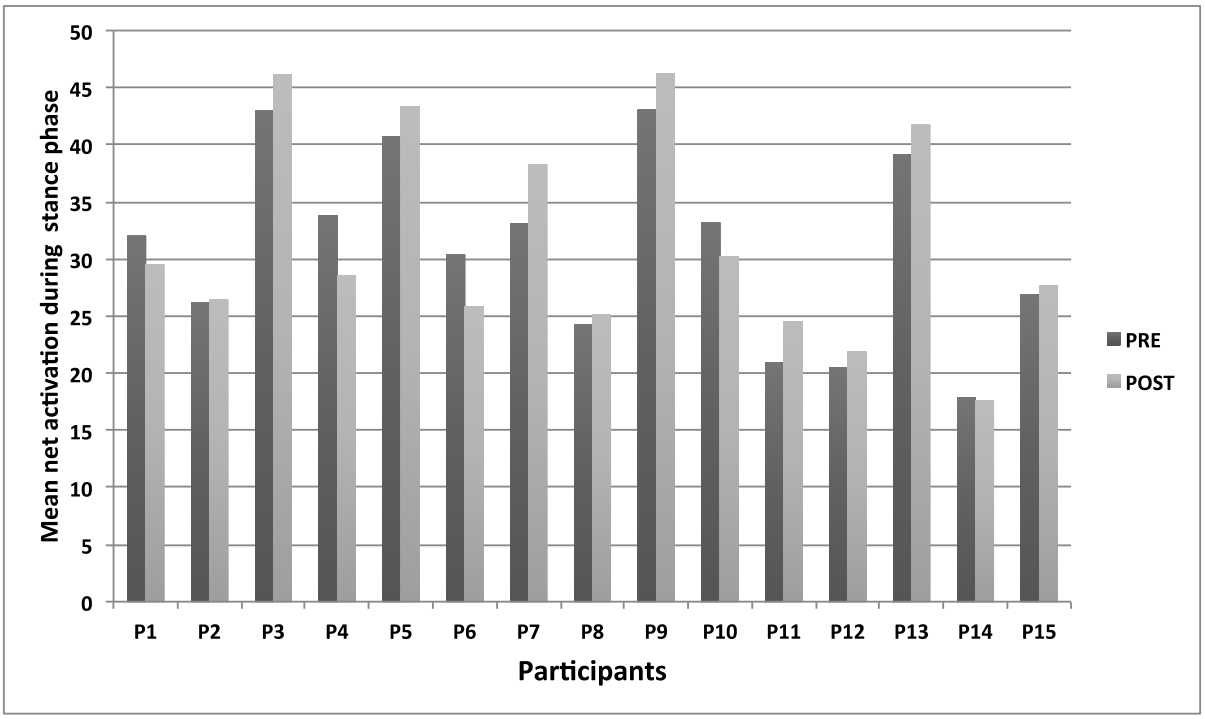


Figure 4-13: Mean net muscle activation during stance for pre- and post-kneeling for each participant.

4.5.3 Change in loading environment – location/contact area, or magnitude

A paired samples t-test was conducted to compare knee adduction angles at heel contact pre- and post-kneeling conditions across participants (Figure 4-14). There was no significant difference in knee adduction angle for pre-kneeling (Mean=1.3°, SD=2.8°) and post-kneeling (Mean=1.8°, SD=3.1°; p=.079) conditions (Figure 4-15).

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreKAA	1.3425	15	2.87333	.74189
	PostKAA	1.7857	15	3.07340	.79355

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreKAA & PostKAA	15	.927	.000

Paired Samples Test

		Paired Differences					t	df
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference			
					Lower	Upper		
Pair 1	PreKAA - PostKAA	-.44327	1.15208	.29747	-1.08127	.19473	-1.490	14

Paired Samples Test

		Sig. (2-tailed)
Pair 1	PreKAA - PostKAA	.158

Figure 4-14: Paired t-tests results for differences in mean adduction angles at heel contact across participants for pre- and post-kneeling. The p-value was divided by 2 to reflect a one-tailed test which was dictated by the structure of hypothesis #3a.

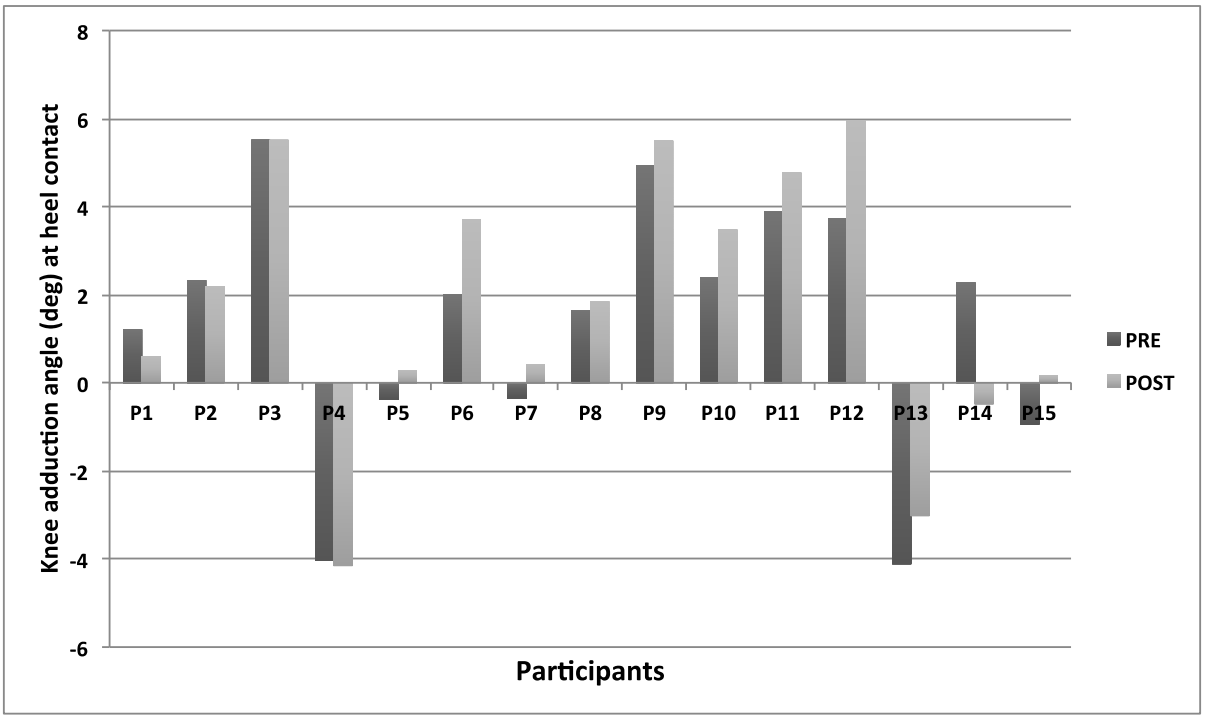


Figure 4-15: Mean knee adduction angles at heel contact for pre- and post-kneeling for each participant. Positive represents adduction, negative represents abduction at heel contact.

A paired sample t-test was conducted to compare peak knee adduction angle differences throughout early stance for pre- and post-kneeling conditions across participants (**Figure 4-16**). There was no significant difference in peak knee adduction angle for pre-kneeling (Mean=6.8°, SD=4.0°) and post-kneeling (Mean=6.7°, SD=4.5°; p=.448) conditions (**Figure 4-17**). This finding (**Figure 4-16**), combined with the previous finding that mean adduction angle at heel contact did not change (**Figure 4-14**), leads to rejection of Hypothesis 3a, which stated that knee adduction angle at heel contact and peak knee adduction angle during early stance would increase in response to the kneeling exposure. In addition to the

statistical analyses on discrete measures, **Appendix E** shows the mean adduction angle curves throughout stance phase.

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreKAAPeak	6.8107	15	3.96458	1.02365
	PostKAAPeak	6.7407	15	4.54913	1.17458

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreKAAPeak & PostKAAPeak	15	.895	.000

Paired Samples Test

		Paired Differences					t
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper	
Pair 1	PreKAAPeak - PostKAAPeak	.06997	2.03620	.52575	-1.05764	1.19759	.133

Paired Samples Test

		df	Sig. (2-tailed)
Pair 1	PreKAAPeak - PostKAAPeak	14	.896

Figure 4-16: Paired t-tests results for differences in peak adduction angles during early stance across participants for pre- and post-kneeling. The p-value was divided by 2 to reflect a one-tailed test which was dictated by the structure of hypothesis #3a.

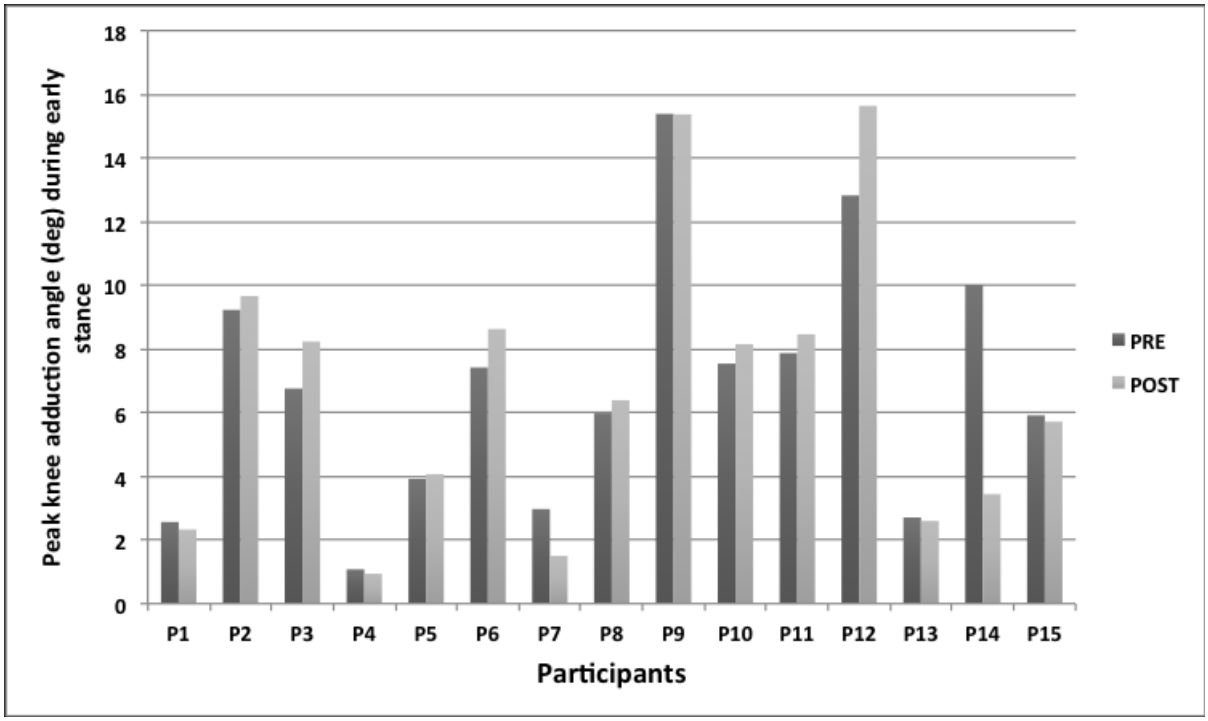
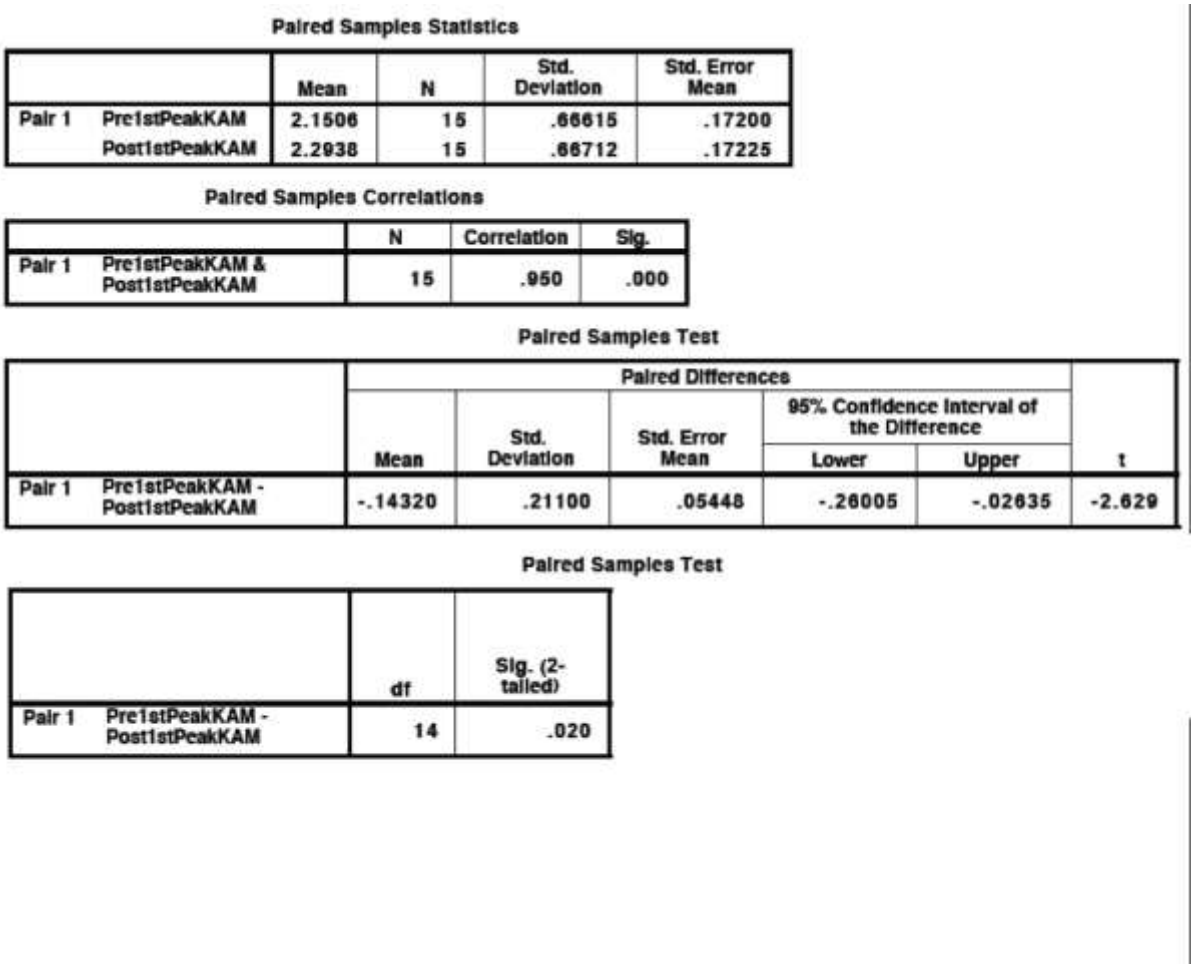


Figure 4-17: Mean peak knee adduction angles during early stance for each participant.

A paired sample t-test was conducted to compare peak knee adduction moment differences during early stance for pre- and post-kneeling conditions across participants (Figure 4-18). There was no significant difference in peak knee adduction moment for pre-kneeling (Mean=2.2, SD=0.67) and post-kneeling (Mean=2.3 SD=0.68; p=0.01) conditions (Figure 4-19). This finding suggests Hypothesis 3b is rejected, which stated that the knee adduction moment during early stance would be higher after the kneeling exposure. External knee adduction moment was the only kinetic variable that did show a trend towards significance following the kneeling exposure. Maximum knee adduction moments were

consistently higher post-kneeling; in fact, 8 of 15 participants in the sample had increases in knee adduction moment of greater than 5% of baseline (pre-kneeling stance phase).



Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Pre1stPeakKAM	2.1506	15	.66615	.17200
	Post1stPeakKAM	2.2938	15	.66712	.17225

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Pre1stPeakKAM & Post1stPeakKAM	15	.950	.000

Paired Samples Test

		Paired Differences					t
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper	
Pair 1	Pre1stPeakKAM - Post1stPeakKAM	-.14320	.21100	.05448	-.26005	-.02635	-2.629

Paired Samples Test

		df	Sig. (2-tailed)
Pair 1	Pre1stPeakKAM - Post1stPeakKAM	14	.020

Figure 4-18: Paired t-tests results for differences in peak adduction moments during early stance across participants for pre- and post-kneeling. The p-value was divided by 2 to reflect a one-tailed test which was dictated by the structure of hypothesis #3b.

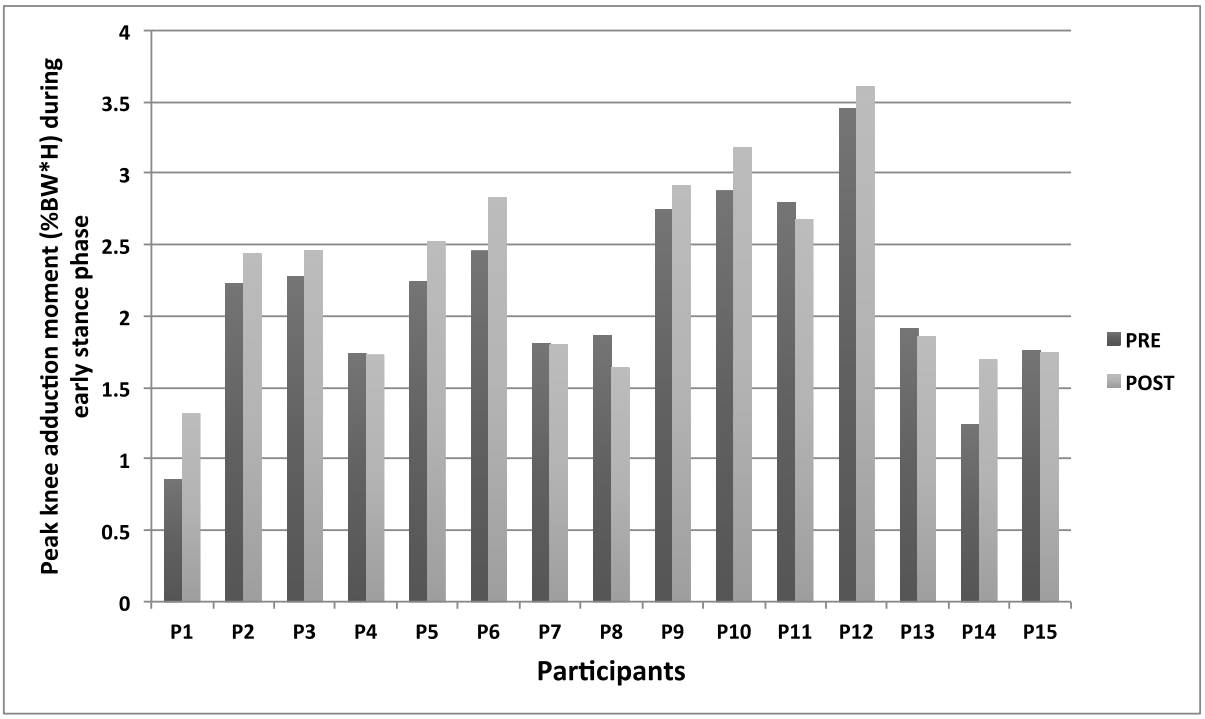


Figure 4-19: Peak knee external adduction moments during early stance for pre- and post-kneeling for each participant.

4.5.4 Summary of hypotheses and statistical results

A summary of the statistics for hypothesis testing is provided (Table 4-2: Hypothesis testing results

Table 4-2: Hypothesis testing results

Hypotheses	Results	Status
Laxity		
1. Exposure to 30 minutes of kneeling will cause an increase in frontal plane knee joint laxity.	There was no significant difference in frontal plane laxity scores pre-kneeling vs. post-kneeling (p=0.0685).	*Rejected*
Responses to instability		
2a. Flexion angles at heel contact and peak knee flexion angles during early stance will be higher post-kneeling than pre-kneeling	There was a significant difference in KFA (p=0.0015) at heel contact and a significant difference in peak KFA (p=0.0025) during early stance	*Accepted*
2b. Peak flexion moment will be lower in early stance phase of gait post-kneeling than pre-kneeling	There was no significant difference in peak KFM (p=0.1685) during early stance	*Rejected*
2c. Mean net muscle activation will be higher throughout stance phase of gait post-kneeling than pre-kneeling	There was no significant difference in mean net muscle activation (p=0.251)	*Rejected*
Change in loading environment – location/contact area, or magnitude		
3a. Adduction angles at heel contact and peak adduction angle during early stance will be higher post-kneeling than pre-kneeling	There was no significant difference in KAA at heel contact (p=0.079) or peak KAA during early stance (p=0.448).	*Rejected*
3b. Peak adduction moment during early stance will be higher post kneeling than pre kneeling	There was no significant difference in KAM during early stance (p=0.01).	*Rejected*

4.6 Discussion

The goal of the current study was to examine the role of a sustained kneeling posture on knee joint laxity, mechanics and muscle activation. Each of the measures included in the analysis were chosen from knee OA literature to either support or refute elements of the proposed pathway to knee OA development in a habitual kneeler group (Figure 1-2). Hypothesis 1 was not supported, as frontal plane laxity changes were not seen after kneeling. Hypothesis 2, which addressed expected changes in response to instability, was partially supported as flexion angles at heel contact and during stance increased post-kneeling potentially as a response to knee instability; however, flexion moment and net muscle activation did not change. Hypothesis 3, which suggested that a change in loading would occur post-kneeling, was not supported as there were no increases in external knee adduction moments post-kneeling.

Frontal plane laxity did not increase for any of the participants, but it did decrease for 3 female participants included in the study. It should be noted that there were no other consistent changes in other outcome measures across these females including discomfort scores. When examined individually, varus and valgus deviations showed a similar trend to that seen for total frontal plane laxity. It was hypothesized that frontal plane laxity would increase acutely post-kneeling given the strain that occurs in passive structures of the knee joint during deep knee flexion postures and kneeling (Hosseini et al., 2014; Thambyah et al., 2005) and based on the results of *in vitro* testing on feline supraspinatus ligaments. This

theoretically makes sense due to viscoelastic tissues being under stress and strain during the kneeling posture and results from Sharma et al. (1999) that suggest that frontal plane laxity precedes OA.

There are a number of possible explanations for not seeing a change in knee laxity. Firstly, though the theoretical framework of a 2:1 work/rest ratio is scientifically supported, anecdotal evidence within the current research group suggests that workers adopt many different postures within the same bout of an occupational task as they become uncomfortable over time. Adopting other postures, such as a one-legged kneel, squat, or hands-supported kneeling, likely contribute to a more variable work/rest ratio in the workplace. Secondly, frontal plane knee laxity measurements require high reproducibility. Chapter 3, Section 3.2 describes the methods used for measuring frontal plane knee joint laxity in the current study. Although the laxity device was proven to be highly reliable, it is possible that this set of participants were more or less variable than the 10 participants used to test reliability in Chapter 3. This would either make it more or less difficult to capture any changes in frontal plane laxity. Thirdly, although ligaments in the knee joint may have become lax, the ability to detect changes in frontal plane knee laxity could have been hindered by increasing stiffness in other biological tissues that contribute to this particular method to measure frontal plane laxity in the knee joint. For example, during articular cartilage creep, there is a loss of water content which results in stiffness and the inability of the cartilage to deform (Walker et al., 1972). In addition, a decision was made at the start of the study to not control the participants' alignment during the kneeling posture. The

instructions to the participants were to have one knee on each of the adjacent force plates, though differing statures would have achieved this with slightly different postures and alignments. If participants were forced to control this alignment in the kneeling posture, the results could have differed since they likely chose the most comfortable alignment. Lastly, although frontal plane laxity increases initially in the course of knee OA, particularly between healthy individuals and mild OA groups, the timing at which this increase occurs is unknown and the results of this study suggest this may be more of a result of knee OA than a cause of knee OA.

Hypothesis 2, which suggested that there would be a change variables that could be interpreted as a response to in knee joint instability, was only supported both by knee flexion angle at heel contact and peak knee flexion during the early stance phase of gait. The mean pre-kneeling KFA at HC and peak in early stance were 8.9° (5.0) and 19.9° (6.5) respectively, while the post-kneeling measures of KFA at HC and peak in early stance were 10.8° (5.4) and 21.8° (6.4). This mean difference between pre- and post-kneeling measures, in each case of approximately 2 degrees, is small, but for some of the participants, the difference was greater than 5 degrees for both measures. The repeated measures design likely found a significant difference, however small, because of the paired design. A clinically significant value for these differences has not been established. It should also be noted that the motion tracking markers were not repositioned and because this was a repeated measures protocol, the change in KFA is more likely to reflect an actual change in kinematics, rather than uncertainty in the angle measurement (e.g. due to changes in marker placement that

could occur). During post-kneeling gait trials, participants landed with increased knee flexion initially but the knee flexion angle at the end of stance phase was the same between pre-and post-kneeling gait trials (Appendix E). This results in a reduced sagittal plane angle range, which has been hypothesized to be a mechanism by which moderate knee OA patients stabilize and stiffen the joint (Childs et al., 2004). However, stability in knee OA literature is also commonly associated with increased co-contraction and net muscle activation (Lewek et al., 2004; Astephen et al., 2008; Zeni et al., 2010). Mean net muscle activation during stance phase was not different between pre- and post-kneeling in this study. In the hypothesized pathway, after a kneeling exposure, muscle activation would increase during the loading phase of gait, as increased stability is necessary due to reduced stability of passive structures such as knee joint ligaments. As noted previously, net muscle activation was the only variable that did not achieve a post-hoc power analysis effect size >0.80. Therefore, the inability to achieve sufficient statistical power for this variable with a sample size of 15 suggests interpretation of stability using this neuromuscular measure should be done with caution. It could also be that some of the variables that were expected to change in response to instability (Hypothesis 2) did not, in fact, change significantly after the kneeling exposure, because the measure of instability in this thesis was frontal plane laxity, which, itself, did not change either for most participants. Thus, the rejection of hypotheses 2b and 2c may be a direct result of the rejection of hypothesis 1. The acceptance of Hypothesis 2a, despite a lack of change in joint laxity, may be because, although changes in knee flexion angle have been associated with an attempt to increase joint stability (Childs et al., 2004; Zeni et al., 2010),

they have also been associated with an attempt to mediate pain or discomfort (Childs et al., 2004). Some, although not all, participants with relatively high scores on the visual analog scale (Appendix B) also had some of the largest increases in knee flexion angle at heel contact (Appendix E).

Hypothesis 3 focused on the knee joint loading element of the proposed knee OA development pathway. It is well known that knee joint loading – in particular, the external knee adduction moment - changes during the progression phase of knee OA (Mundermann, Dyrby, Hurwitz, Sharma, and Andriacchi, 2004). The proposed knee OA pathway in this study suggests that knee joint loading changes occur acutely, after a single bout of a knee-straining exposure. Hypothesis 3 was not supported as external knee adduction moments during stance phase did not increase post-kneeling. This is inconsistent with previous work by Kajaks and Costigan (2015) who found that the external knee adduction moment increased following 30 minutes of deep flexion kneeling. Though both studies reported small mean differences (0.12%BWH vs. 0.07Nm/kg) after kneeling using the same work-rest ratio, differences in the design (females and more participants included in the current study) and methods (filter cut-offs kept the same for kinematics and kinetics in the current study) may have contributed to the differing statistical significant findings. Out of all the variables studied though, the trend for increasing external knee adduction moments post-kneeling was the most consistent across study participants (8 of 15). A change in external maximum knee adduction moment could occur due to a change in knee adduction angle, ground reaction forces or muscle activation. Higher adduction angles have been related to higher adduction

moments, which may be indicative of greater medial joint loading (Gaudreault, Hagemeister, Poitras & de Guise, 2013). This relationship was not observed in this study, nor did muscle activation change. It is possible that the knee adduction angle changes observed in osteoarthritic gait (Duffell et al., 2014) develop over time in response to repeat altered loading. The results of this study neither support (since both angles and moments did not increase) nor refute (since neither increased while the other decreased) this theory of knee OA development – at least from an acute exposure. A clinically significant value has yet to be established for acute exposures but it would likely be smaller than chronic OA values. Ground reaction forces were not part of the analysis of the current study, though Childs et al. (2004) reported decreased ground reaction forces in knee OA patients when compared to age- and sex-matched controls. Based on the results of this study, there would be value added in including this variable in future studies on the initiation of knee OA.

Although knee pain is not a direct indicator of knee osteoarthritis, it is thought to be an important risk factor for knee OA development (Robbins et al., 2011). Additionally, strong associations have been found between knee pain and occupation (O'Reilly et al., 2000). Knee discomfort was monitored every 5 minutes during the kneeling protocol using a VAS protocol (Appendix A). Knee discomfort monitoring during the kneeling protocol determined that high levels of discomfort (up to 6/10 on VAS) are present during sustained deep knee flexion, however, this discomfort is transient and returns to baseline levels following 5 minutes of seated rest after each 10 minute bout of kneeling (Appendix B). What is also apparent in the data collected on discomfort is that there is a cumulative effect of the

kneeling exposure on discomfort (Figure 4-20). After the end of each bout of 10 minutes of kneeling, many of the participants reported high levels of discomfort, often higher than the previous levels indicated at the end of the prior 10 minutes of kneeling. Though this data set is specific to the kneeling posture chosen in the current study, these acute high levels of knee discomfort warrant further investigation on biological variables such as blood occlusion, micro tearing of passive structures, and nerve damage, all of which are beyond the scope of the current project but are likely contributors to knee pain and should be a focus for future knee OA development studies given the role pain may play in knee OA development (Robbins et al., 2011).

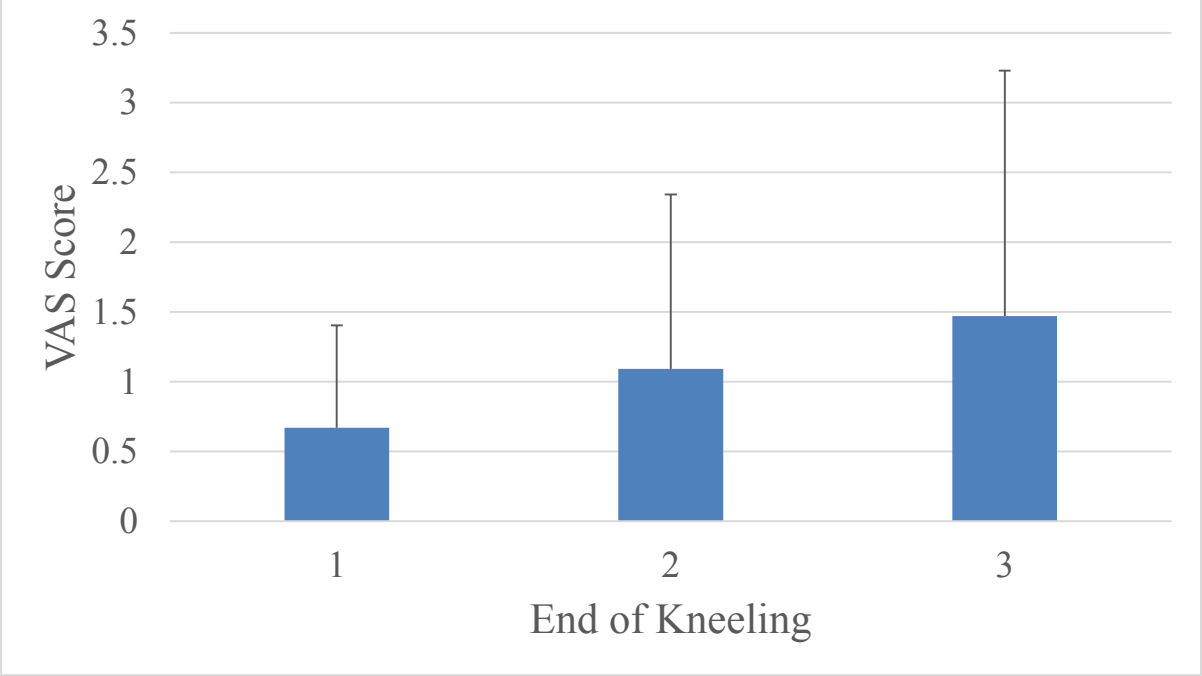


Figure 4-20: VAS mean scores across participants at the end of each bout of kneeling.

The current study and the results of Kajaks & Costigan (2015) confirm that there are elements of the proposed knee OA development pathway that support the biomechanical

relationship between prolonged static deep knee flexion and knee OA. Both studies observed changes in mechanics during gait that can be considered negative adaptations that can damage cartilage. The current study findings refute the proposed mechanism where an acute laxity change plays a role in knee OA initiation in habitual kneelers. Furthermore, while our study examined immediate changes in frontal plane laxity and gait parameters, Gaudreault et al. (2013) showed that there are chronic kinematic gait parameter changes for those who participate in at least 30 minutes of daily knee-straining work for over 5 years. This finding is especially interesting given the fact that all study participants included in their study did not have any evidence of symptomatic or radiological knee OA. Although the same adduction angle changes were not found in this study, there was an increase in flexion angle post-kneeling at the same stance phase events (heel contact and peak of early stance) that was reported for knee-straining workers, however the angles and differences between groups were higher than those observed in this study and in another previous study (Barrios et al., 2009).

Although increased frontal plane laxity did not occur, and so the first step outlined in the pathway (Figure 1-2) was not supported, one can acknowledge that there are many other pathways to knee OA development, which is why the variability across participants in adaptations (or lack thereof) to the kneeling exposure is not surprising (Figure 4-7, Figure 4-9, Figure 4-11, Figure 4-13, Figure 4-15, Figure 4-17, Figure 4-19). It is worth noting that although much focus has been spent on frontal plane laxity as a common outcome examined in knee OA literature, other studies have also suggested that laxity in both the sagittal and transverse

planes are worth investigating to assess laxity changes across knee OA progression (Shultz et al., 2007). This study provides evidence of the following changes after an acute bout of kneeling: increased knee flexion at heel contact (Figure 4-7) and increased peak knee flexion in early stance (resulting in decreased range of knee flexion during stance phase) (Figure 4-9). These findings may suggest that although collateral ligaments have previously been found to be stretched during deep knee flexion, this acute, sagittal plane exposure shows mainly changes in the sagittal plane. Although chronic kneeling exposures were beyond the scope of this study, perhaps longer durations and repeated exposures could potentially result in the collateral ligament changes that have been reported in animal model studies (Park et al., 2005; Hosseini et al., 2014).

Chapter 5: Overall Contributions and Future Directions

This thesis examined frontal plane knee joint laxity and gait mechanics before and after a bout of full flexion kneeling to determine if there is support for the following theoretical mechanism of knee OA initiation in habitual kneelers: First, kneeling results in a change in frontal plane knee joint laxity, which results in instability in the joint. Next, gait mechanics and muscular activation change in an attempt to provide more joint stability. These changes negatively affect the joint loading environment (in a manner that is detrimental to cartilage integrity). This research endeavour resulted in the following contributions:

1. **The design, implementation and reliability testing of an improved frontal plane knee laxity measurement device.** The device was shown to be highly reliable (within visit ICC = 0.97). Tracking frontal plane laxity is of particular importance due to the increases that have been reported throughout the progression of knee OA (Sharma et al., 1999; van der Esch, Steultjens, Wieringa, Dinant & Dekker, 2005). Future work should aim to determine how sample size, BMI, pain, different raters, and multi-day visit schedules affect both the reliability and the laxity scores achieved. An interesting application for future work would be to look at changes to passive stiffness. In the current design, more weights would need to be applied to create a passive stiffness curve that could be compared at differing angles during varus and valgus applications. Additionally, motion capture was used because it was the most accurate measurement tool available but other measurement modalities could be explored for future clinical applications. Future applications of this device could include longitudinal studies of laxity during OA progression and the effect of longer kneeling exposures in habitual kneeling.

2. **A 30-minute static, full flexion, knee straining exposure did not affect knee frontal plane laxity**. This was the first study to investigate the acute effects of knee straining postures on frontal plane knee joint laxity. Despite the ability to reliably detect small changes in frontal plane knee joint laxity, the current study did not see any changes following the kneeling exposure. This was a surprising finding given the duration of kneeling in deep flexion, the research on viscoelastic creep, and how much discomfort participants reported while kneeling. As noted in Chapter 4, Section 4.5.1, there are a number of possible reasons as to why no changes in frontal plane laxity were observed in the current study sample. Another possible explanation for seeing no differences in this study could be the mandatory rest break between kneeling bouts in this study which likely does not reflect how an occupational kneeler would perform work. Barring these reasons, it is quite possible that frontal plane knee joint laxity is merely a consequence of knee OA, rather than an element in the mechanism of initiation. To explore this possibility, future studies should aim to: include knee-straining workers (habitual kneelers), examine the effect of prolonged kneeling on squatting mechanics since squat performance might be more sensitive than gait to the effects of kneeling, and vary the frequency of activities to assess other exposure-response relationships that workers may be exposed to including lifting of heavy loads. Researchers should also aim to better understand viscoelastic properties of the passive structures of the knee joint and surrounding areas to further inform the choice of appropriate outcome variables.
3. **A 30-minute static knee straining exposure did affect gait parameters that can be used as surrogates to knee joint loading and stability.** While the results of the current study do not support laxity as a mechanism linking prolonged kneeling to changes in gait

parameters, knee flexion angles at heel contact, and peak knee flexion angle during early stance are outcome variables that change following this particular knee-straining exposure, for this study sample. Despite the complexity of changes in magnitude and direction for many of the outcome variables chosen, these variables can be used to support the notion that, after kneeling, there are changes that have been interpreted as a response to instability (flexion angles) and changes in knee joint loading (peak external knee adduction moments). Still, these variables are strictly surrogates of the forces inside the knee. A better understanding at the cellular and biological level is needed to understand what negative cartilage adaptations are occurring, if any, due to occupational kneeling as a stand-alone risk factor.

Though it was not included in the study, pilot results in our research group suggest that a significant level of blood occlusion occurs during deep knee flexion. Additionally, subjective anecdotes suggest there is a feeling of numbness in the lower leg that occurs after about 5-6 minutes of kneeling. Future cellular or biological studies should further assess blood occlusion and nerve damage in high flexion and the potential impact on knee joint health and integrity.

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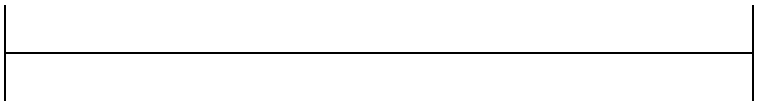
Appendix A: Visual Analog Scale

VISUAL ANALOG SCALE (VAS)

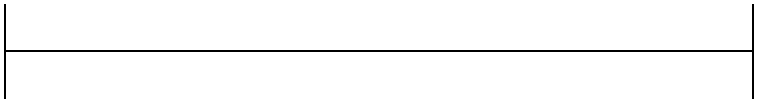
"On the scale, please indicate with a solid line, the level of discomfort you experienced during the exposure for each of the areas of the body indicated below"

ANKLE



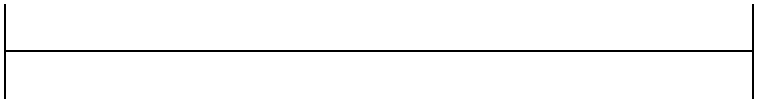
NO DISCOMFORT EXTREME DISCOMFORT

LOWER BACK



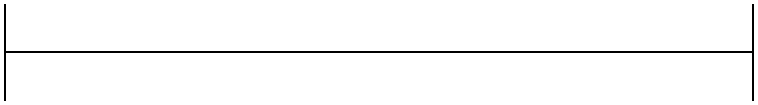
NO DISCOMFORT EXTREME DISCOMFORT

FRONT OF SHANK/KNEE



NO DISCOMFORT EXTREME DISCOMFORT

BACK OF SHANK/KNEE



NO DISCOMFORT EXTREME DISCOMFORT

Appendix B: Kneeling Discomfort Data

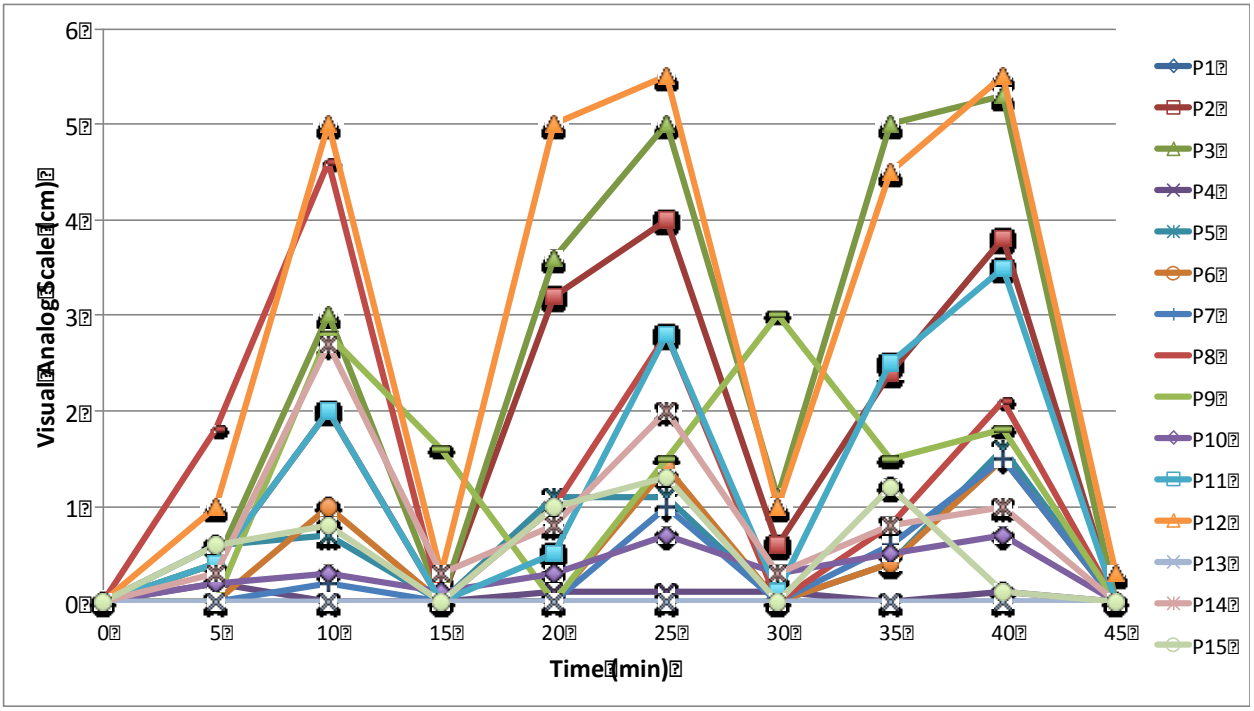


Figure B-1: Foot discomfort data for all participants throughout the kneeling exposure

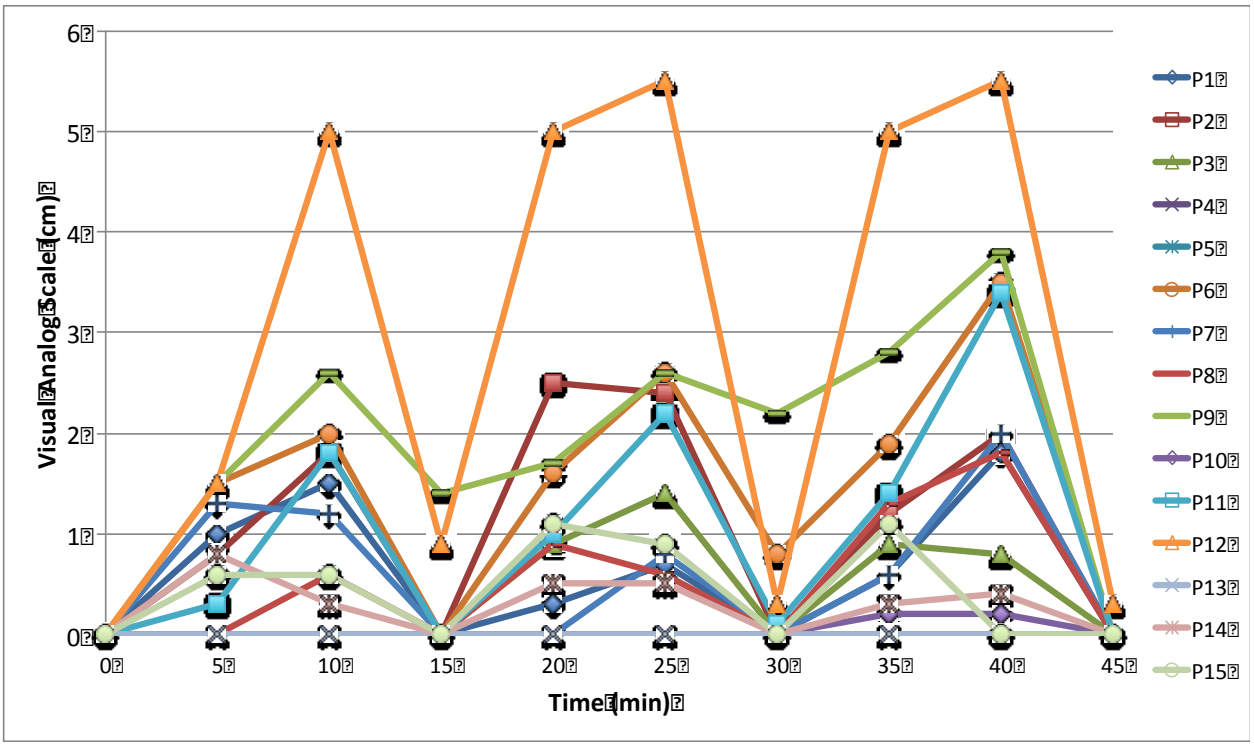


Figure B-2: Posterior knee/shank discomfort data for all participants during the kneeling exposure

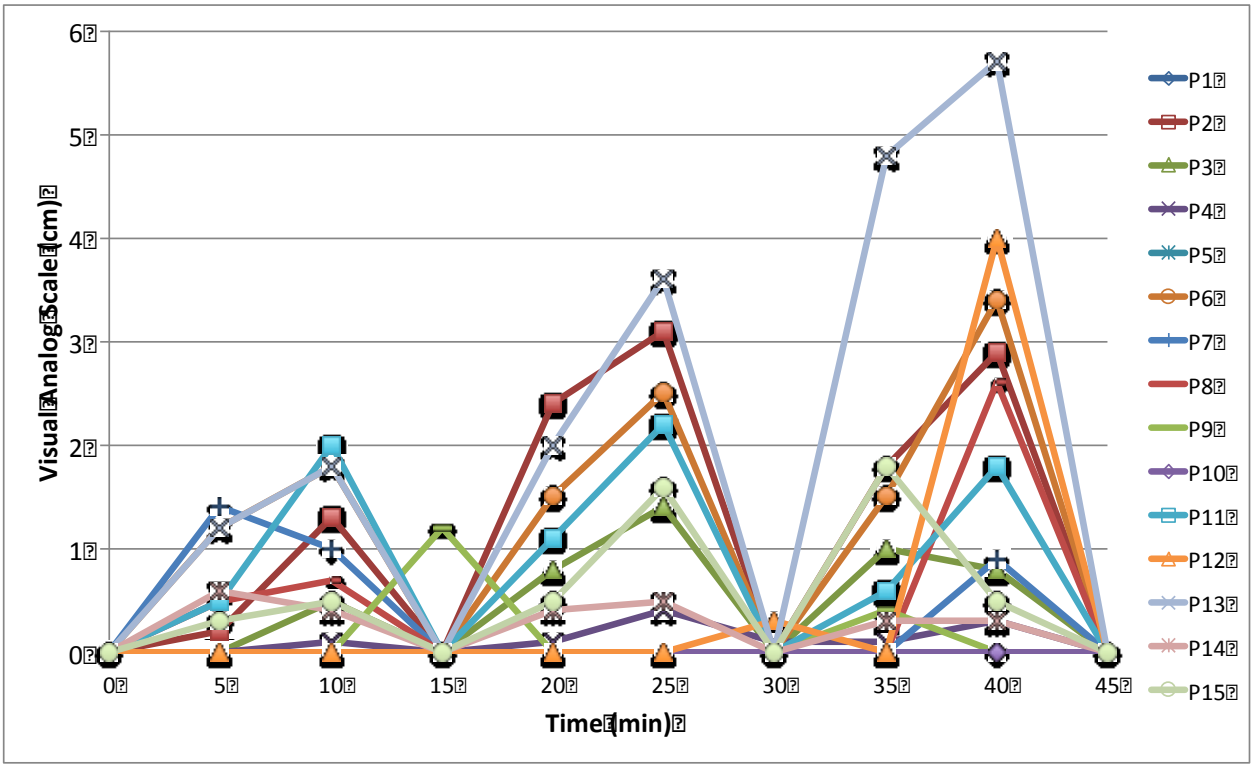
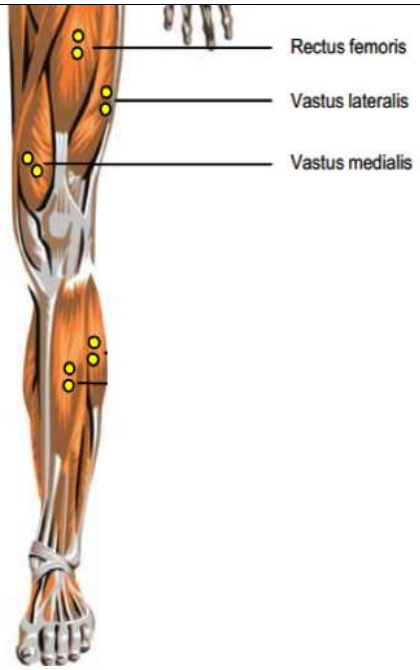


Figure B-3: Anterior knee/shank discomfort data during the kneeling exposure

Appendix C: Electrode Placements

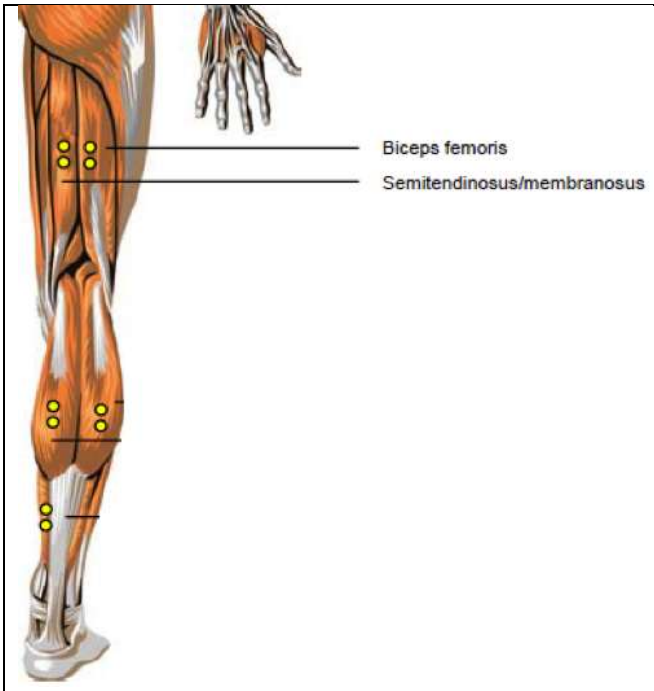
EMG Electrode Placements



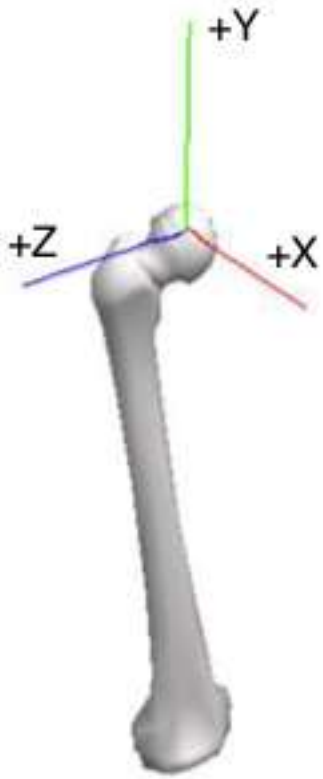
a. Vastus Medialis
Sensors are to be placed at 80% of the line from the anterior superior iliac spine (ASIS) and ending at the joint space in front of the anterior border of the medial ligament.

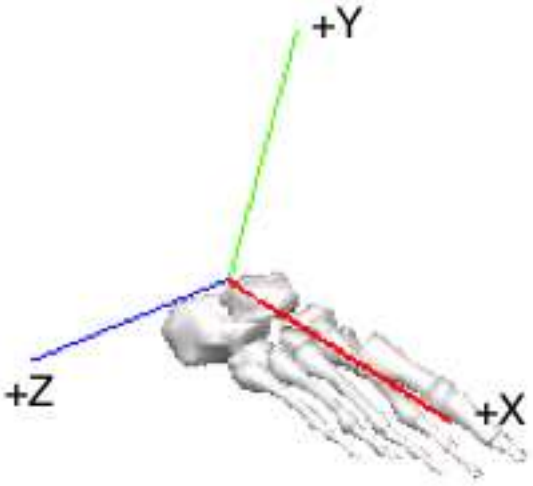
b. Rectus Femoris
Sensors are to be placed at 1/2 the distance along the line from the ASIS to the superior part of the patella.

c. Vastus Lateralis
Sensors are to be placed 2/3 of the distance along the line starting at the ASIS and ending at the lateral side of the patella.

	a. Biceps Femoris Sensors are to be placed at 1/2 of the distance along the line starting at the ischial tuberosity and ending at the lateral epicondyle of the tibia. b. Semitendinosus Sensors are to be placed at 1/2 of the distance along the line starting at the ischial tuberosity and ending at the medial epicondyle of the tibia.

Appendix D: Local Coordinate Systems

Femur  	Origin: The origin is defined as a quarter of the distance between the femoral greater trochanters, located medially from the right greater trochanter, along the negative Z-axis YZ plane: The YZ-plane is the plane defined by the greater trochanter and the lateral and medial femoral epicondyles. Y-axis: The Y-axis is defined as the vector from the knee joint center (mid-way point between the two condyles of the femur) to the origin. X-axis: The X-axis is the defined as the vector perpendicular to the YZ-plane, anteriorly. Z-axis: The Z-axis is defined as the vector perpendicular to both the Y-axis and the X-axis, calculated as the cross-product of X-axis by the Y-axis, and points laterally from the origin.
Shank	Origin: The origin is the midpoint between the lateral and medial tibial condyles YZ plane: The YZ-plane is defined by the lateral and medial tibial condyles and the lateral and medial malleoli.

	Y-axis: The Y-axis is defined as the vector from the knee joint center (origin) and the midpoint of the malleoli markers. X-axis: The X-axis is defined as the vector perpendicular to both the YZ-plane, anteriorly. Z-axis: The Z-axis is defined as the vector perpendicular to both the Y-axis and the X-axis, calculated as the cross-product of X-axis by the Y-axis, and point laterally from the origin.
Foot 	Origin: The origin is defined as the midpoint between lateral and medial malleoli markers. YZ-plane: The YZ-plane is the plane defined by the lateral and medial malleoli and the 1st and 5th metatarsals. Y-axis: The Y-axis is defined as the vector from the midpoint of the 1st and 5th metatarsal markers and the midpoint of the malleoli markers. X-axis:

	The X-axis is defined as the vector perpendicular to the YZ-plane, anteriorly. Z-axis: The z-axis is defined as the vector perpendicular to both the Y-axis and the X-axis, calculated as the cross-product of X and Y, and points laterally from the origin.

Appendix E: Mean curves across participants for Study 2 dependent variables

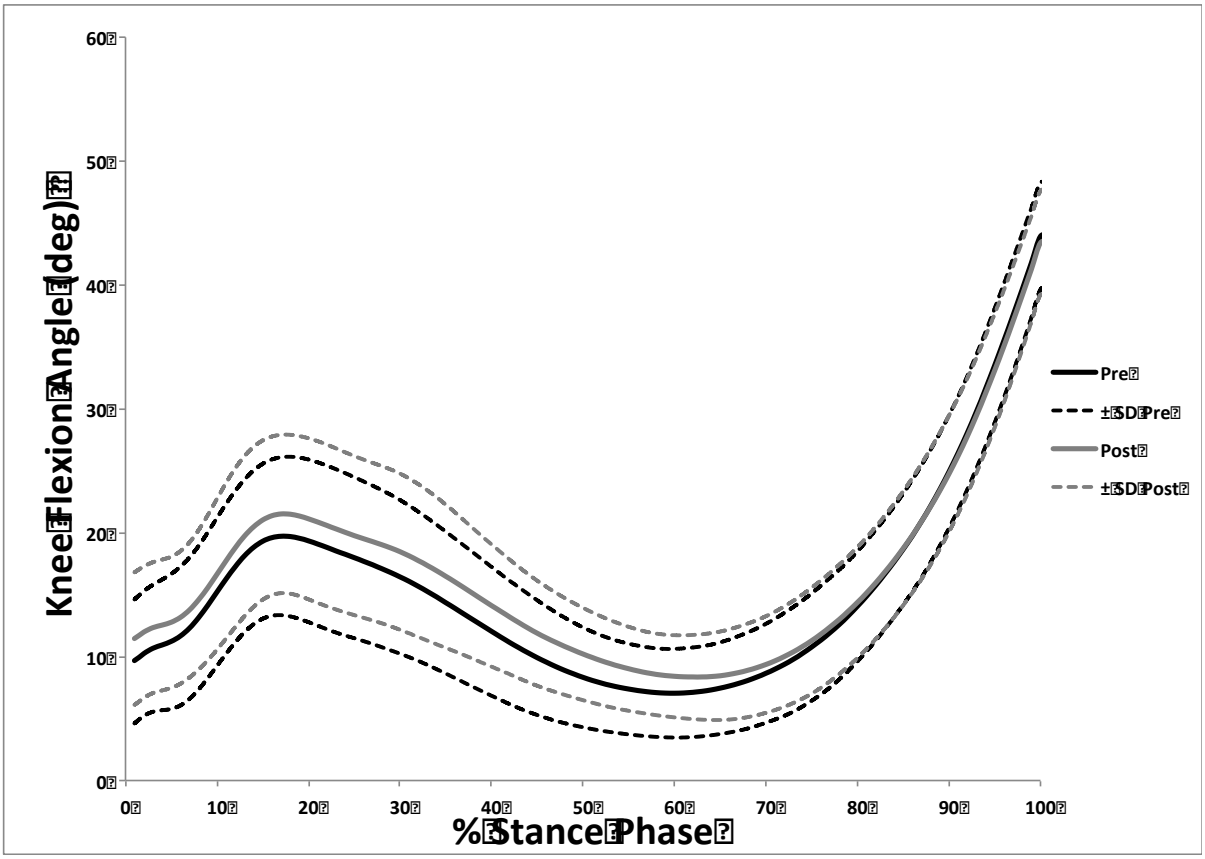


Figure E-1: Mean curves for knee flexion angle during early stance for pre- and post-kneeling. Heel contact represents 0% of stance phase, toe-off represents 100% of stance phase. Positive values represent flexion moments.

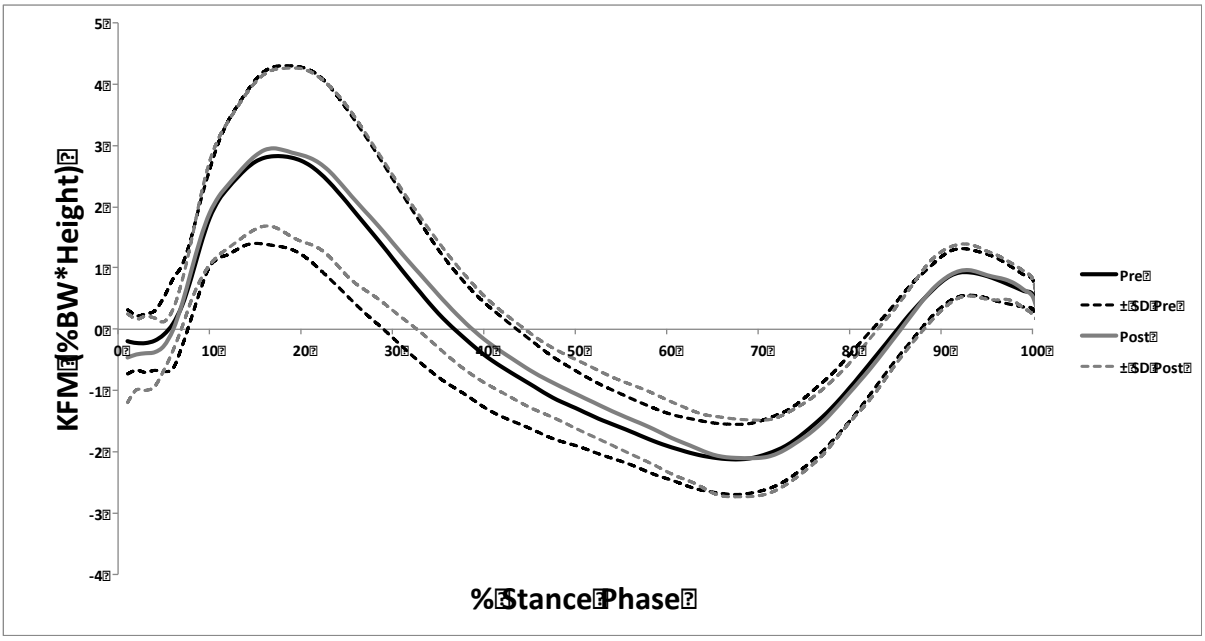


Figure E-2: Mean curves for knee flexion moments during early stance for pre- and post-kneeling. Heel contact represents 0% of stance phase, toe-off represents 100% of stance phase. Positive values represent flexion moments.

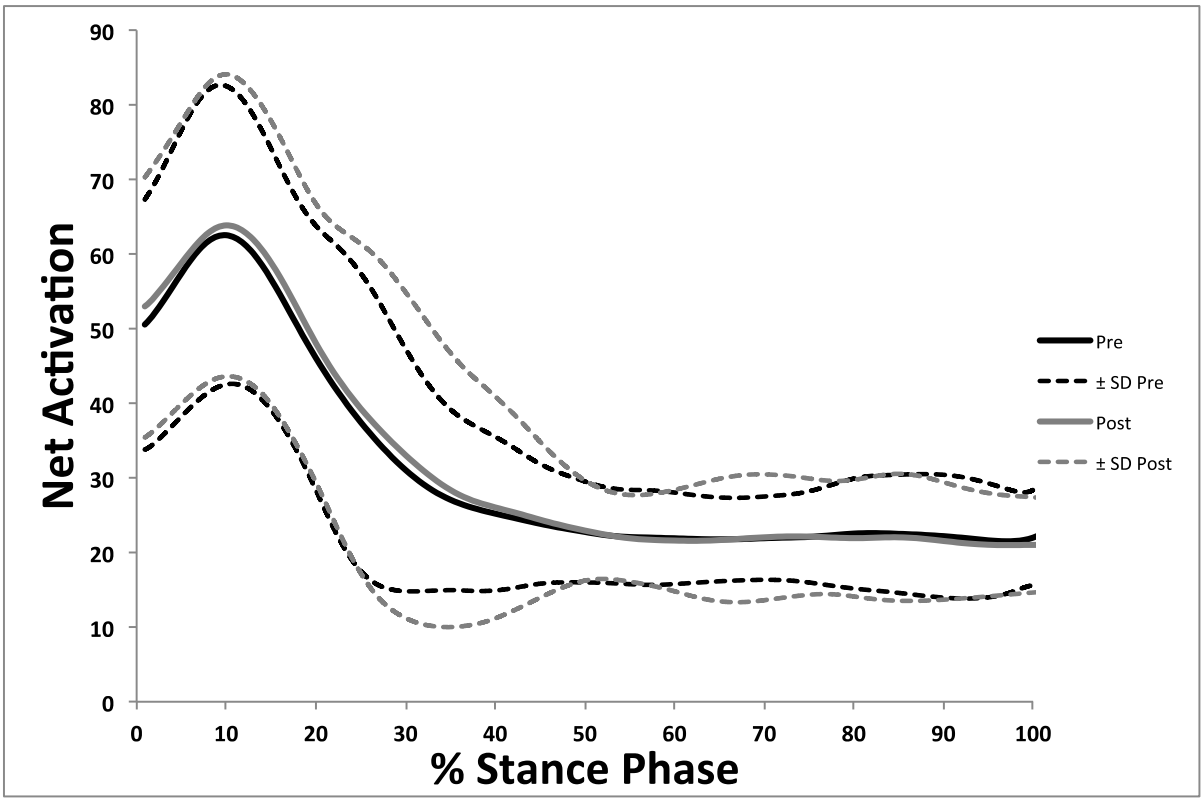


Figure E-3: Mean curves for net muscle activation during early stance for pre- and post-kneeling. Heel contact represents 0% of stance phase, toe-off represents 100% of stance phase. Positive values represent flexion moments.

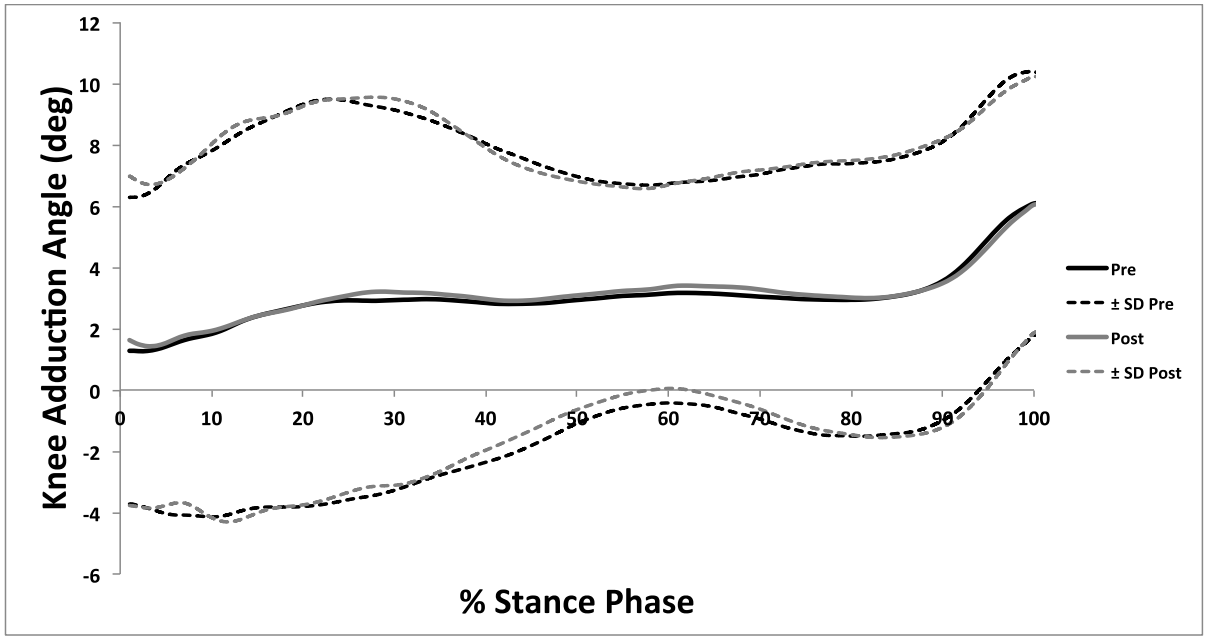


Figure E-4: Mean curves of knee adduction angle during stance phase of gait for pre- and post-kneeling. Heel contact represents 0% of stance phase, toe-off represents 100% of stance phase. Positive values represent adduction angles.

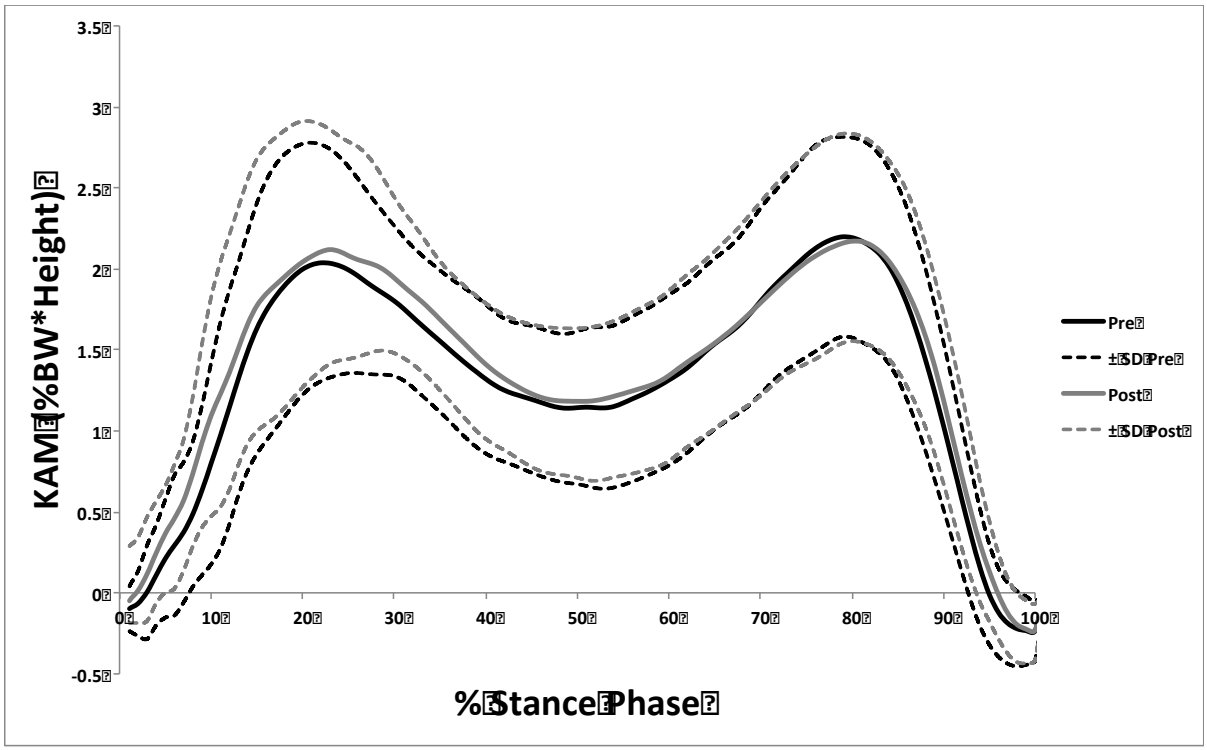


Figure E-5: Mean curves of knee adduction moment during stance phase of gait for pre- and post-kneeling. Heel contact represents 0% of stance phase, toe-off represents 100% of stance phase. Positive values represent adduction angles.