Neuromuscular Control of Gait and Squatting Following a Simulated Occupational Kneeling Exposure

by

Liana Michele Tennant

A thesis
presented to the University of Waterloo
in fulfillment of the
thesis requirement for the degree of
Master of Science
in
Kinesiology

Waterloo, Ontario, Canada, 2016

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 electronically available to the public.

Liana Tennant

Abstract

Frequent and prolonged occupational high knee flexion postures such as kneeling and squatting are associated with an increased risk for the initiation and development of knee osteoarthritis. In order to reduce the prevalence of knee osteoarthritis in this population, a better understanding of the link between these postures and the onset of joint degeneration is needed. The goal of this thesis was to investigate the effects of a 30-minute simulated occupational kneeling exposure on the mechanics of gait and squat transitions, as well as knee joint proprioception, in order to evaluate any negative adaptations that may be associated with an increased risk for knee osteoarthritis development. It was expected that increased joint loading during gait following the kneeling exposure would be linked to deficits in proprioceptive acuity. Greater frontal plane knee motion during squat transitions was also hypothesized. These hypotheses were based on the concept that a prolonged kneeling exposure may induce proprioceptive and neuromuscular control changes by way of ligamentous creep, muscle stretch, pain, or joint capsule deformation.

Forty healthy, young participants volunteered for this study (20M, 20F, age: 21.4 ± 2.5 years, height: 1.69 ± 0.10 m, mass: 68.8 ± 16.1 kg). Vastus medialis muscle activity was recorded with surface electromyography, external ground reaction forces were collected using force plates, and 3D lower limb kinematics were measured using an active motion capture system. A knee joint position sense task was used to evaluate proprioception. Gait was evaluated in both an unloaded condition and a loaded condition. In the loaded condition, participants carried a load normalized to 20% of their body mass in a crate held in both hands in front of the torso to simulate how loads are carried occupationally. Participants also performed squat transitions at both a slow and a fast pace. Participants then completed a 30-minute

simulated occupational kneeling exposure, following which proprioception, gait, and squatting measures were re-collected. All values were collected for a third time, 30 minutes from the time the kneeling exposure ended.

The kneeling exposure resulted in a significant increase in both the peak and mean knee frontal plane motion during the squat transition. The average baseline values for both peak and mean absolute frontal plane knee deviation (peak: 0.207 m (SD 0.110 m); mean: 0.084 m (SD 0.048 m)) were significantly smaller than post-kneeling values (peak: 0.229 m (SD 0.107 m), $p = .0057$; mean: 0.091 m (SD 0.048 m), $p = .0186$). The knee joint was still deviated during squat transitions 30 minutes post-kneeling (peak: 0.227 m (SD 0.108 m), $p = .0267$; mean: 0.093 m (SD 0.049 m), $p = .0061$). During gait, vastus medialis activation onset was delayed with respect to initial contact following the kneeling exposure (pre: -0.159 s (SD 0.034 s); post: -0.152 s (SD 0.035 s), $p = .0004$; 30post: -0.148 s (SD 0.032 s), $p = .0003$); however, the biological significance of this small change in activation onset is questionable. The kneeling protocol did not elicit a change in knee proprioception, or in the measures of external loading evaluated during gait.

Greater frontal plane knee motion following the kneeling exposure suggests a negative adaptation in movement control that may act to increase the risk of traumatic joint injury that could lead to secondary knee osteoarthritis. The lack of change in proprioceptive acuity implies that changes in knee joint position sense are not responsible for the observed change in squat control. In addition, the dynamic kneeling exposure used in the current study seems to reduce the potential for inducing adaptations in motor control during gait. This reasoning is based on a comparison to previous work that found alterations in gait mechanics following a 30-minute static full-flexion kneeling exposure. Further research is needed on the effects of prolonged and

cyclic kneeling on ligamentous creep, joint laxity, and neuromuscular control during gait, as well as other occupationally relevant tasks such as squat transitions.

Acknowledgements

I would not have been able to complete this project without the incredible support of a number of wonderful people:

To my supervisor, Dr. Stacey Acker – We have been working together for a while now, almost four years! Thank you for mentoring in me and believing in me, and allowing me to take my project where I felt it needed to go. I have grown from an undergrad that was nervous to work with the equipment into an experienced researcher under your guidance.

Dr. Jack Callaghan – Thank you for your valuable feedback and input on this project. Thank you also for the mentoring and guidance you have provided over the last couple of years as I worked on (and continue to work on) figuring out 'what next.'

Drs. Steve Prentice and Jim Frank – Thank you for your guidance on this project. Your input has made this a stronger study and I appreciate your contributions.

A big thank you goes out to all my lab mates, friends, and colleagues. Special acknowledgement goes to Dave and Helen – thanks for being such great friends, I cannot imagine having gone through this experience without you!

Thank you also to the undergrads that assisted with the data collection and some of the more monotonous components of data processing. Thanks for being troopers.

Thank you to the Department of Kinesiology staff who helped make the completion of this project that much smoother.

I would also like to acknowledge my financial support from provincial (OGS) and federal (NSERC) funding bodies.

Finally, thank you to my family, who definitely got more involved with this project than I originally anticipated! Thank you to my Aunt Michele for her sewing contributions. Mom, Dad, Ryan, thanks for supporting me through my academic career, and always being willing to lend an ear (or sometimes a hammer!) throughout the trials and tribulations of this project. Thank you!

Table of Contents

List of Figures

List of Tables

List of Abbreviations

AAE = absolute angular error

ACL = anterior cruciate ligament

APDF = amplitude probability distribution function

BW = body weight

BW/s = body weight / second

EMG = electromyography

HST = heel-strike transient

KAM = knee adduction moment

LCL = lateral collateral ligament

MCL = medial collateral ligament

MPF = mean power frequency

MVC = maximal voluntary contraction

OA = osteoarthritis

PCL = posterior cruciate ligament

ROL = rate of loading

ROM = range of motion

SD = standard deviation

VAS = visual analog scale

VM = vastus medialis

%BW*H = % body weight * height

%GC = % gait cycle

1.0 Introduction

The growing incidence of both tibiofemoral and patellofemoral osteoarthritis is a serious concern. Not only does knee osteoarthritis impair mobility and decrease quality of life, it is also a significant economic burden on society (Hunter, Schofield, & Callander, 2014; Uhlig, Slatkowsky-Christensen, Moe, & Kvien, 2010; Xie et al., 2008). Workers who regularly adopt high knee flexion postures (> 120° flexion), such as kneeling and squatting, demonstrate an increased risk for the development of knee osteoarthritis. For example, floor layers and tile setters are at an increased risk for the development of knee osteoarthritis (Coggon et al., 2000; Cooper, McAlindon, Coggon, Egger, & Dieppe, 1994). The risk is even greater in occupations that require kneeling and squatting as well as heavy lifting or carrying (Amin et al., 2008; Cooper et al., 1994; Ezzat & Li, 2014). The link between occupational physical demands and osteoarthritis risk is troubling because the process of cartilage degeneration is irreversible (Vigorita, Ghelman, & Mintz, 2008). Therefore, preventing occupation-mediated knee osteoarthritis is imperative.

Traditionally, knee osteoarthritis has been viewed as an outcome of 'wear-and-tear,' – an inevitable consequence of repetitive joint use. However, there is evidence to suggest that the mechanism of knee osteoarthritis initiation and progression is actually much more complicated. Because motor control is modifiable and an important component of joint load attenuation, researchers have hypothesized that alterations in neuromuscular control could act to either promote or reduce progressive joint degenerative changes in the knee. Based on this proposed mechanism, it is possible that time spent in an occupational kneeling posture could result in gait adaptations, such as increased medial knee joint loads or greater rates of external loading at the knee, which have been associated with an increased risk for knee joint injury and osteoarthritis

(Birmingham, Hunt, Jones, Jenkyn, & Giffin, 2007; Miyazaki et al., 2002). Changes in motor control during gait are important risk-related outcomes in this occupational group because workers spend may spend approximately 40 – 60% of the workday in upright, weight-bearing activities, including gait (Jensen, Rytter, & Bonde, 2010; Tennant, unpublished work). While the mechanism linking occupational kneeling to adaptations in gait is unknown, prolonged kneeling may alter proprioceptive sensation in the lower limb, perhaps due to ligamentous creep, muscular and joint capsule stretch or prolonged compression, pain, or similar mechanism known to alter neuromuscular control.

Therefore, the goal of this project was to determine whether, in a young, healthy, and uninjured population, a simulated occupational kneeling exposure altered gait characteristics associated with joint loading and knee osteoarthritis risk, including the peak knee adduction moment, vertical loading profile, and quadriceps activation patterns. Transitions into and out of a squat posture were also analyzed to identify changes in frontal plane knee motion that would indicate changes in neuromuscular control, and possibly the risk for traumatic knee injury. Finally, the effects of the kneeling exposure on knee joint proprioception were evaluated to determine if any changes in neuromuscular control could be linked to changes in proprioceptive sensation. By investigating these variables this project contributes to a better understanding of the potential mechanism of knee osteoarthritis initiation and progression in individuals whose occupations require combined high knee flexion postures, such as kneeling and squatting, and lifting or carrying.

2.0 Purpose and Hypotheses

The literature reviewed (3.0) suggests that gait may be altered following a kneeling exposure. Changes in neuromuscular control during gait may act to increase the risk for knee osteoarthritis in a number of ways:

a) Load surfaces of the joint that are not conditioned for load.

b) Alter the magnitude of loading, thereby increasing the risk for joint injury (e.g. meniscal damage).

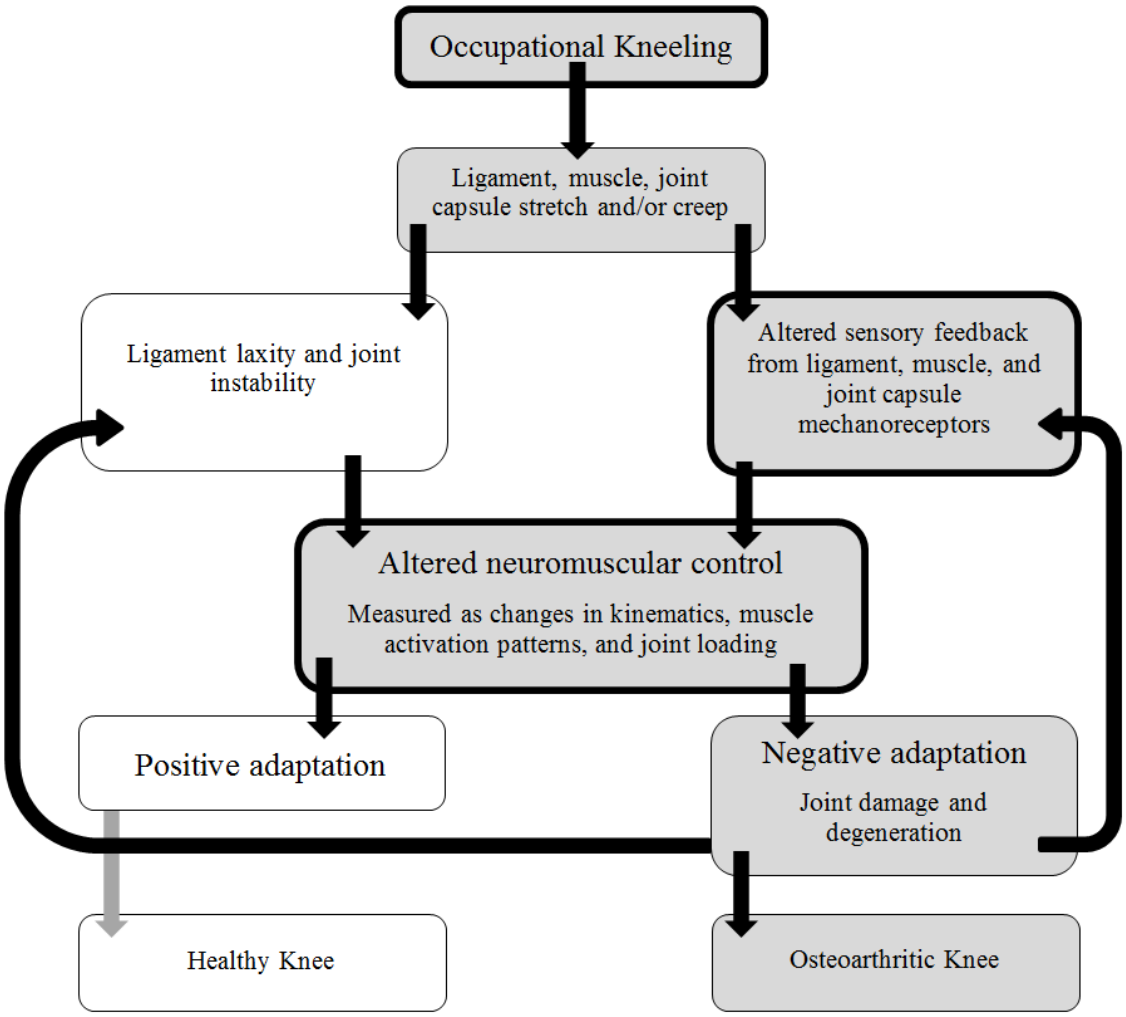
c) Impair normal force attenuation mechanisms at the knee.

In this study, kinematics and external forces were measured; however, joint translations and direct measures of joint contact force and location could not be determined. Therefore, the changes described in (a) above are outside the scope of the project. To evaluate changes in the magnitude of loading at the knee (b), indirect methods were employed. The peak external knee adduction moment was used as a surrogate measure of medial knee joint loading. The peak rate of loading was used as a surrogate measure of overall joint load (i.e. across both the medial and lateral compartments of the knee) that takes into account the fact that joint cartilage is viscoelastic, and therefore affected by the rate at which load is applied. Finally, to address (c), measures of quadriceps activation onset and magnitude were evaluated because pre-activation of the quadriceps prior to initial contact during gait is thought to contribute to force attenuation at the knee. Although not a direct assessment of any of the above risk factors, an evaluation of knee joint control while transitioning to-and-from the floor using a squat technique was used to identify changes in neuromuscular control. Characteristics of squat performance have not been evaluated for knee osteoarthritis risk specifically, beyond the potential injury risk associated with large joint forces in high-flexion squats. However, frontal plane knee motion has been found to

be related to traumatic injury risk during certain dynamic tasks. In addition, squatting may be used as a transition from standing to kneeling, or vice versa, in an occupational setting. Therefore, it was important to evaluate a measure of change in this high knee flexion task that is also required in occupations that require frequent and prolonged kneeling.

While acknowledging that osteoarthritis is a complex disease with a multitude of interacting risk factors, because of the role of sensory afferent input as a contributor to neuromuscular control, it is possible that impaired proprioceptive sensation also contributes to knee osteoarthritis risk. In the proposed mechanism (Figure 2.1Figure 2.1 – follow the pathway of shaded boxes along the right side), an occupational kneeling exposure may result in knee ligamentous, muscular, and/or joint capsule creep or stretch, or pain, which may alter sensory feedback. These alterations in sensory feedback could result in abnormal patterns of joint loading and motor control during gait or in transitions into and out of a high-flexion squat. This proposed mechanism provides the rationale for the outcome variables measured in this study: Greater medial knee joint loading, an increased rate of external loading, or altered quadriceps activation timing or magnitude during gait might indicate that alterations in neuromuscular control following an occupational kneeling exposure could contribute to joint damage and the initiation and progression of knee osteoarthritis. Any negative adaptations would likely be amplified when the worker carries heavy tools and materials while ambulating. A lack of change in gait outcome variables after the kneeling exposure, but an increase in frontal plane knee motion during squat transitions might indicate that a kneeling exposure increases the risk for traumatic injury, such as meniscus tears, because the knee joint is not tracking in a joint-sparing way.

This study is not longitudinal in nature and therefore determining a causal relationship between this mechanism (Figure 2.1, highlighted pathway) and the development of knee osteoarthritis is not possible. Similarly, while it is not within the scope of this investigation to quantify ligamentous creep, it is possible to measure changes in external loads, knee joint position sense (proprioception), muscle activation patterns, and joint kinematics. Should significant differences in these measures be observed in a young and healthy population, it can be determined whether a simulated occupational kneeling exposure results in changes to factors of neuromuscular control that could increase the risk for the development of knee osteoarthritis. This is a novel contribution to the literature because it is unknown whether measures of knee joint load such as the external knee adduction moment or rate of loading at initial contact are altered following a simulated occupational kneeling exposure. Similarly, the effects of such an exposure on squat mechanics are also undocumented. Finally, it is also unknown whether an occupational kneeling exposure changes knee proprioception.



adapted from Kajaks & Costigan (2015)

Figure 2.1 The proposed mechanism of joint damage resulting from occupational kneeling. The mechanism pathway explored in this project highlighted in grey. The specific components of the pathway that are explored in this project are bold-outlined.

Based on the proposed mechanism of knee osteoarthritis development, this project aims to answer a number of specific questions (Table 2.1).

Table 2.1

Questions, Hypotheses, and Rationale

Question	Hypotheses	Rationale
	Gait	
1. How is the peak knee adduction moment altered during gait following a simulated occupational kneeling exposure?	a) The peak knee adduction moment will be greater post-kneeling. b) Changes in the peak knee adduction moment will persist at thirty minutes post-kneeling. c) A carried load will increase the peak knee adduction moment.	The peak knee adduction moment is considered a compartment-specific measure of load over the medial surface of the tibiofemoral joint, with a higher moment associated with greater medial compartment loading (Birmingham et al., 2007). Because medial knee osteoarthritis is more prevalent in occupational kneelers (Cooper et al., 1994), it is worthwhile to evaluate the external knee adduction moment as a surrogate measure of medial knee load.
2. Is the vertical loading profile during gait altered following a simulated occupational kneeling exposure?	a) The peak vertical loading rate will be greater following an occupational kneeling exposure. b) Changes in the peak rate of loading will persist at thirty minutes post-kneeling. c) A carried load will increase the peak rate of loading.	The peak vertical loading rate is the maximum instantaneous rate of change of force in the vertical direction at initial contact. It is a surrogate measure of the load over the entire surface of the tibiofemoral joint (Hunt et al., 2010) that reflects the fact that joint cartilage is viscoelastic and therefore is affected by the rate of load application. Although occupational kneelers exhibit a greater incidence of medial tibiofemoral knee osteoarthritis, increased risk is observed in both compartments (Cooper et al., 1994). Therefore, a surrogate measure of overall knee load is beneficial.

Question	Hypotheses	Rationale
3. Do parameters of quadriceps muscle activation during gait change following a simulated occupational kneeling exposure?	a) The onset of vastus medialis activation will be delayed with respect to initial contact post-kneeling in both loading conditions. b) The magnitude of vastus medialis activation during the impact phase will be lower post-kneeling in both loading conditions. c) Changes in vastus medialis onset and activation magnitude will persist at thirty minutes post-kneeling.	Two variables of vastus medialis activation have been associated with increased loading rates and greater prevalence of heel-strike transients – magnitude of quadriceps pre-activation and onset of quadriceps pre-activation (Liikavainio et al., 2007). In heel-strikers, the quadriceps activate less and turn on later compared to non-heel-strikers. Therefore, although loading rate was measured, as well as the incidence of heel-strike transients, quadriceps pre-activation onset and magnitude gives some additional insight into the neuromuscular control mechanisms responsible for any observed changes in loading rate.
Squat Transitions		
4. Does neuromuscular control, as evaluated by the performance of squat transitions, demonstrate deficits following a simulated occupational kneeling exposure?	a) Absolute peak and mean deviations of the knee joint center from the plane created by the hip, ankle, and midfoot will be greater following a simulated occupational kneeling exposure. b) Observed changes will persist at thirty minutes post-kneeling.	Squat transitions are also performed frequently by individuals in occupations that require frequent and prolonged kneeling (Tennant, unpublished work). Since alterations in gait have been found following a kneeling exposure (Kajaks & Costigan, 2015), it is possible that movement patterns during other occupationally relevant tasks may be similarly affected. Although movement patterns during squats have not been directly linked to knee osteoarthritis risk, certain movement characteristics, such as frontal plane knee motion, could be linked to traumatic knee injuries (Hewett et al., 2005), which in turn could lead to knee osteoarthritis development. Therefore, analysis of squat transitions is an important inclusion.

Question	Hypotheses	Rationale
	Knee Proprioception	
5. Is proprioceptive acuity at the knee, as evaluated by a measure of joint position sense, impaired following a simulated occupational kneeling exposure?	a) The average absolute angular error of knee joint position sense will increase following a simulated occupational kneeling exposure. b) Observed changes in knee joint position sense will persist at thirty minutes post-kneeling.	Proprioception has been hypothesized to be linked to the control of force attenuation at the knee and may contribute to knee osteoarthritis onset (Knoop et al., 2011). Therefore this is an important measure that may help to explain any observed changes in gait or neuromuscular control patterns.

3.0 Literature Review

This literature review will establish a number of concepts that are important to the understanding of the project. First, muscle activation is the main mechanism of force attenuation at the knee joint (3.1.1); in particular, quadriceps pre-activation is an important determinant of knee force and loading rate at initial contact of gait (3.1.1). Second, excessive impulsive load (3.1.1) and therefore muscle dysfunction (3.1.2, 3.2.3) may contribute to knee osteoarthritis risk. Third, knee osteoarthritis has biomechanical contributions (3.2.1) that can be linked to frequent and prolonged occupational kneeling (3.1.2). The review will also discuss the role of proprioception in coordinating muscular pre-activation (3.1.1) and the ways in which proprioception can be evaluated (3.2.2). In addition, the need for attention to proprioceptive changes as a potential mechanism through which kneeling exposures may result in altered gait parameters that could contribute to knee osteoarthritis risk will be established (3.2.2, 3.4). Finally, previous work that has evaluated alterations in gait following a prolonged static kneeling exposure and in individuals who work in high knee flexion postures will be summarized (3.3).

3.1 Knee Osteoarthritis

3.1.1 Knee joint anatomy and force attenuation mechanisms in gait.

In order to understand how occupational kneeling combined with lifting and carrying might contribute to the development of knee osteoarthritis, it is important to address the normal structure and load bearing function of the knee. The knee is a synovial joint with three bony articulating surfaces – the distal end of the femur, the proximal end of the tibia, and the patella. A number of uni- and bi-articulate muscles that allow for knee flexion and extension, internal and external rotation, and abduction and adduction, guide joint movement. Five ligaments

restrain motion at the knee: two cruciate, two collateral, and the recently identified anterolateral ligament (Claes et al., 2013; Vincent et al., 2012). The medial and lateral menisci are cartilaginous structures found between the femoral condyles and the tibial plateau. The menisci contribute to joint stabilization, lubrication, nutrition of the articular cartilage, and proprioception (McDermott, Masouros, & Amis, 2008). The entire joint is surrounded by an external fibrous capsule and an internal synovial membrane. (Moore & Dalley, 2006)

In a healthy, non-osteoarthritic knee, articular cartilage acts as a low-friction wear-resistant tissue that allows for smooth and controlled movement (Pearle, Warren, & Rodeo, 2005), and contributes to the transmission and distribution of joint loads (Griffin & Guilak, 2005). The health of the articular cartilage is maintained through a delicate cycle of the synthesis and turnover of the cartilage extracellular matrix (Lin, Willers, Xu, & Zheng, 2006). Because the articular cartilage is avascular, the transport of nutrients and metabolic waste is facilitated through joint loading and joint movement (O'Hara, Urban, & Maroudas, 1990). *In vitro* studies have suggested that static compressive loading, as might occur in a high knee flexion posture, suppresses metabolic activity and degrades the extracellular matrix (Griffin & Guilak, 2005; Lin et al., 2006). In contrast, dynamic compression of articular cartilage at specific frequencies can be beneficial for cartilage health (Griffin & Guilak, 2005). Articular cartilage in some models demonstrates potential for adaptive capabilities (Liu et al., 2013); however, adult human articular cartilage does not appear to have a high capacity to repair structural damage (Brandt, Dieppe, & Radin, 2009; Griffin & Guilak, 2005).

Historically, cartilage, bone, and the menisci were all thought to contribute to force attenuation and load bearing at the knee (Englund et al., 2009; Hoshino & Wallace, 1987; Kurosawa, Fukubayashi, & Nakajima, 1980; Seedhom, 1979; Seedhom & Hargreaves, 1979;

Walker & Erkman, 1975). While it is true that subchondral bone can attenuate about 30% of the load at the knee (Imhof et al., 2000), it is now understood that articular cartilage and the fibrocartilaginous menisci contribute very little to force attenuation. The knee joint cartilage and menisci are mostly responsible for assisting in the distribution of force over the articulating surfaces by improving the congruency of the joint surface and increasing the contact area (Andrews, Shrive, & Ronsky, 2011; McDermott et al., 2008).

Although cartilaginous tissues have minimal force attenuation properties, recent literature suggests that appropriate activation of the leg musculature prior to impact appears to be the main mechanism of force attenuation at the knee during gait (Radin & Rose, 1986; Radin, Yang, Riegger, Kish, & O'Connor, 1991). Generally, during gait, external loads are anticipated and the quadriceps muscles activate prior to foot contact to distribute the force across the entire joint surface and decrease the rate of loading at the joint via eccentric contraction (Felson, 2004a; Lindstedt, LaStayo, & Reich, 2001). However, the magnitude and timing of quadriceps pre-activation varies, resulting in different loading patterns and forces (Whittle, 1999). For example, some individuals utilize a braking strategy where the leg decelerates using the ground, with minimal quadriceps pre-activation at the beginning of the stance phase (Jefferson, Collins, Whittle, Radin, & O'Connor, 1990). This lack of pre-activation results in a period of impulsive loading, referred to as a heel-strike transient (Figure 3.1) (Jefferson et al., 1990).

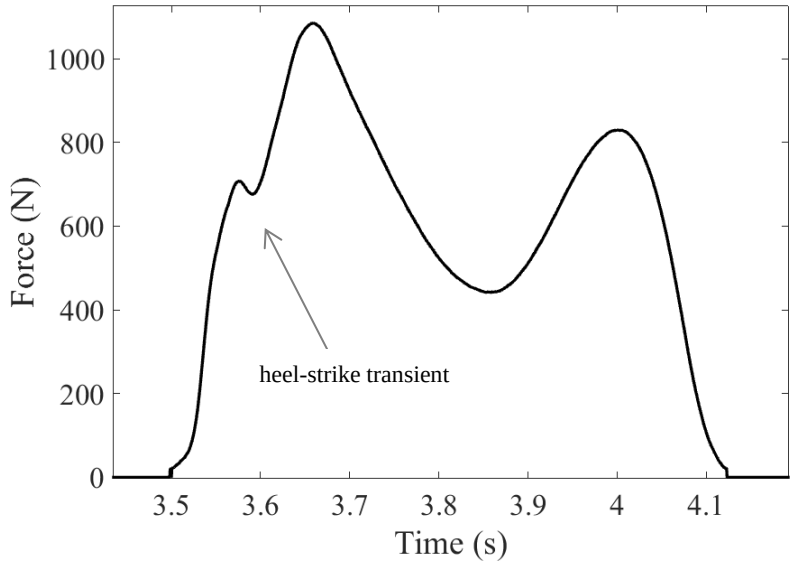


Figure 3.1 Vertical component of the ground reaction force from a single stride of normal, shod gait with visible heel-strike transient.

Although a number of different methods exist to identify heel-strike transients – which may affect reports of prevalence – the heel-strike transient deceleration pattern has been estimated to occur in one-third of the adult population (Radin et al., 1986). More recently, a barefoot study found that 22 out of 25 participants demonstrated regular heel-strike transients (Verdini, Marcucci, Benedetti, & Leo, 2006). Heel-strike transients may occur due to poor muscle coordination, such as delayed activation onset of rectus femoris and tibialis anterior, and inadequate biceps femoris activation (Verdini et al., 2006), but may also be simply a result of poor leg strength (Mikesky, Meyer, & Thompson, 2000). The high rate of loading associated with these transients has been hypothesized to promote degenerative changes to knee articular cartilage (Collins & Whittle, 1989; Liikavainio et al., 2007; Radin et al., 1984).

A second method of deceleration is a quadriceps dominant strategy (Jefferson et al., 1990). In this second method, the quadriceps group activates prior to initial contact. Therefore, in this strategy, force is thought to be attenuated by muscle as the eccentric contraction of the

quadriceps performs negative work (the muscle lengthens while exerting tension) and converts the mechanical energy to heat (Alexander, 1991; Brandt et al., 2009; Jefferson et al., 1990; Radin et al., 1991). This results in a reduced rate of loading at the knee. See Figure 3.2 for a comparison of each of the deceleration methods. Note that in the non-heel-striker, peak vastus medialis activation onset precedes initial contact of the foot.

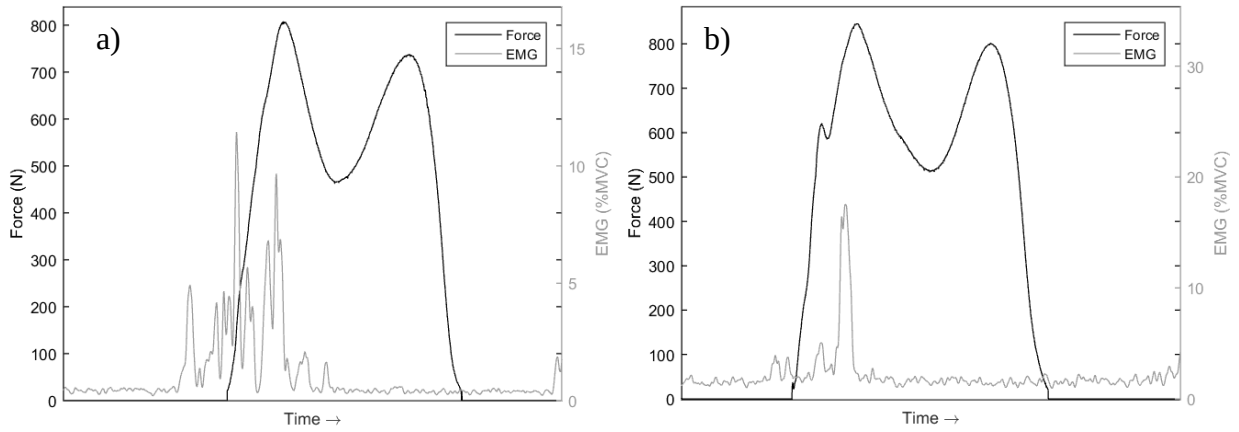


Figure 3.2 Comparison of the vertical ground reaction force and MVC normalized VM activation patterns of a non-heel-striker (a), and a heel-striker (b), both walking at 1.4 m/s.

Regardless of the braking strategy, coordinated muscle activation depends on neural inputs from the joints, muscles, tendons, and deep tissue proprioceptors (Table 3.1) (Hewett, Paterno, & Myer, 2002). This interaction between the sensory afferent pathway and the motor efferent pathway is termed the sensorimotor system (Lephart & Fu, 2000). Because of the intimate relationship between sensory input and motor output, accurate afferent information is necessary for normal motor control function and appropriate force attenuation mechanisms (Riskowski, Mikesky, Bahamonde, Alvey III, & Burr, 2005). Based on the understanding that sensory and motor functions are highly integrated, this project evaluated both gait and squat transition mechanics, as well as knee joint position sense. In this way, the findings of the project

may more insightful than an isolated evaluation of either component (sensory or motor function) in isolation.

Table 3.1

Knee Joint Proprioceptors

Receptor	Location	Stimulus Specificity
Musculotendinous mechanoreceptors		
Muscle spindles	Muscle fibres (intrafusal)	*Ia/1° afferents* Velocity and length sensitive, particularly to rapidly changing stimuli *II/2° afferents* Length sensitive ONLY
Golgi tendon organs	Tendons	Actively generated muscle force
Articular mechanoreceptors		
Pacinian corpuscles*	Ligaments, menisci, capsule	Small, dynamic changes in tissue deformation
Ruffini endings	Ligaments, menisci, capsule	Joint angle (especially at end ROM), velocity, intra-articular pressure
Golgi receptors	Ligaments, menisci, capsule	Joint angle (especially at end ROM)
Bare nerve endings*	Tissues in and surrounding the knee, including ligaments	Excessive tissue deformation, pain, inflammation

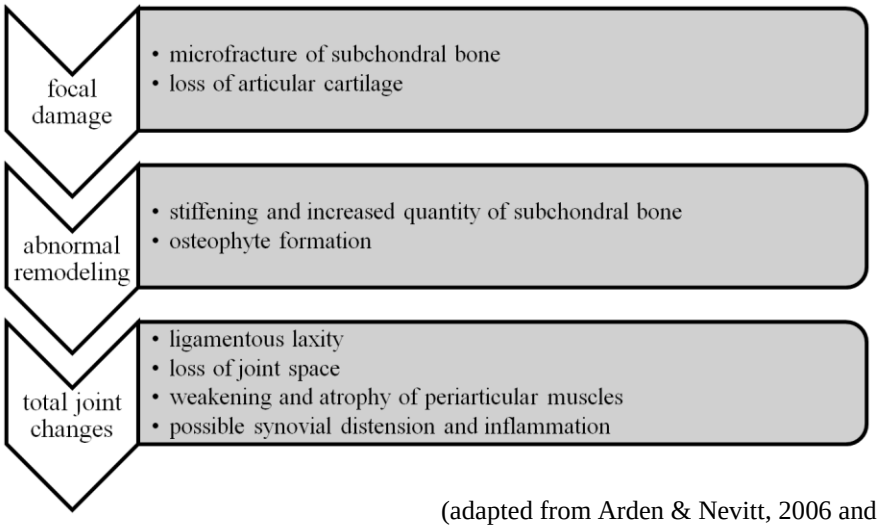
* although found in the joint, these receptors can also be found in the muscle

(adapted from Knoop et al., 2011; Rothwell, 1994)

3.1.2 What is knee osteoarthritis and who is at risk?

Defining osteoarthritis is a difficult task. Due to the wide array of factors and pathways that are hypothesized to contribute to joint degradation, osteoarthritis may be best described as the pathological and clinical outcome of a range of disorders, which often results in impaired mobility and general disability (Brandt, Dieppe, & Radin, 2008; Guccione et al., 1994; Nuki, 1999). Although a common pathophysiological progression encompassing all causes of knee

osteoarthritis does not exist (Brandt et al., 2009), attempts have been made to map the pathophysiology of mechanically mediated osteoarthritis (Figure 3.3). Initial changes generally include softening (chondromalacia), fibrillation, and erosion of habitually load-bearing articular cartilage, and microfracture and subsequent thickening of the subchondral bone (Radin & Rose, 1986). The remodeled subchondral bone is stiffer than before, resulting in further degenerative changes (Anderson, Brown, & Radin, 1993) which can be seen radiographically as decreased joint space width (Hunter et al., 2006) due to the combination of eroding articular cartilage and the formation of osteophytes. Eventually, over the course of many years, "total joint failure" may occur (Felson, 2004b) in that all of the structures of the joint – cartilage, bone, ligament, muscle, tendon, synovium, and joint capsule – are dysfunctional (Arden & Nevitt, 2006; Buckwalter & Mankin, 1997; Vigorita et al., 2008). Clinical symptoms of individuals with knee osteoarthritis may include knee joint instability (sometimes referred to as the feeling that the knee is 'giving way') (Fitzgerald, Piva, & Irrgang, 2004), pain, crepitus, inflammation and swelling, and decreased range of motion (Kuettner & Goldberg, 1995).



(adapted from Arden & Nevitt, 2006 and Felson, 2004b)

Figure 3.3 The pathogenesis of knee osteoarthritis.

Fortunately, although osteoarthritis results in irreversible joint damage, multiple studies have indicated that the disease may remain at a steady state for many years with little to no worsening of radiographic or clinical symptoms (Felson, 2004b; Vigorita et al., 2008; Watt & Dieppe, 1990; Watt, 2009). However, should dysfunction become debilitating, invasive surgical interventions such as total knee arthroplasty may be considered. Because many end-stage knee osteoarthritis treatments require expensive and invasive surgery, prevention of the initiation and progression of the disease is critical, especially in groups that are known to be at increased risk.

Epidemiologic studies give an indication of the frequency and distribution of disease, which can in turn help guide researchers to potential causes of a disease and identify at-risk populations. When studying knee osteoarthritis, understanding the prevalence of the disease is complicated by the fact that knee osteoarthritis is not simple to diagnose. A number of classification methods have been suggested to define osteoarthritis. Some authors have suggested that osteoarthritis is a spectrum or family of diseases (Guilak, 2011) and that different joints display unique risk factors (Dieppe & Kirwan, 1994). More commonly, osteoarthritis is classified as either idiopathic (primary), or localized (secondary). Idiopathic osteoarthritis is thought to develop because of metabolic disease, endocrine disorders, calcium deposition diseases, or neuropathy (Vigorita et al., 2008), and is often found in multiple joints of the body, commonly in the knee, hip, metacarpal, and intervertebral joints (Arden & Nevitt, 2006). The interest of this project lies in localized osteoarthritis, which is osteoarthritis that occurs in a joint due to local factors. Unfortunately, many epidemiologic studies do not make this distinction. Another part of the difficulty in defining and classifying osteoarthritis is that radiographic disease severity does not necessarily correlate with symptoms and disability (Arden & Nevitt, 2006; Dieppe & Lohmander, 2005; Sharma, Kapoor, & Issa, 2006). In fact, some individuals

may show evidence of substantial osteoarthritic disease but do not present with joint symptoms (Radin, 2004). In light of these inconsistencies, numerous of definitions have been used in the literature (Table 3.2).

Table 3.2

Classifications of Knee Osteoarthritis used in the Literature

Osteoarthritis Definition	**Description**
Clinical Osteoarthritis	Osteoarthritis is diagnosed based on a combination of symptoms (e.g. pain, disability) and the findings of a physical examination, which may or may include medical imaging (Arden & Nevitt, 2006).
Radiographic Osteoarthritis	The presence of osteoarthritis is based on analysis of radiographic images of the joint. The diagnosis of osteoarthritis is usually based on criteria such as evidence of osteophytes, narrowing of the joint space, sclerosis of subchondral bone, and bone contour deformity. The Kellgren and Lawrence scale is commonly used (Sharma et al., 2006) and ranks the joint on a scale of 0-4. A '0' on the scale indicates no radiographic evidence of osteoarthritis, while a '4' suggests severe osteoarthritis (Kellgren & Lawrence, 1957). However, neither Kellgren and Lawrence, nor subsequent users of this scale were consistent in the definition of the five grades (Schiphof, Boers, & Bierma-Zeinstra, 2008).
Symptomatic Osteoarthritis	An individual has frequent pain in the joint, often defined as most days of the month. Some authors have chosen to define symptomatic knee osteoarthritis as pain or stiffness in the context of radiographic evidence (Segal et al., 2009).
Asymptomatic Osteoarthritis	It has been well documented that an individual may present with significant radiographic (structural) osteoarthritis, but indicate little or no pain or disability (Felson, 2004b; Garstang & Stitik, 2006; Hannan, Felson, & Pincus, 2000; Radin, 2004).

(adapted from Lawrence et al., 2008)

Based on the differences in osteoarthritis classification strategies, it is important to recognize that the results of epidemiologic studies may be affected by the methods selected to determine the presence of disease. Nevertheless, it is clear that osteoarthritis is the most prevalent form of arthritis (Lawrence et al., 2008). Most people over 70 years of age have radiologic evidence of osteoarthritis in some joints (Dieppe & Lohmander, 2005). According to a World Health Organization report from 2000, globally, knee osteoarthritis prevalence ranges from 2,369 – 20,238 per 100,000 for adult males, and 6,211 – 30,208 per 100,000 for adult females, depending on the demographic (Gibson et al., 1996; Solomon, Beighton, & Lawrence, 1975). In addition, knee osteoarthritis is generally more common in women compared to men (Arden & Nevitt, 2006; Felson et al., 2000). This discrepancy between men and women exists until about 80 years of age, when rates become equal (Arden & Nevitt, 2006). Knee osteoarthritis affects more than 20 million people in the United States and is considered one of the leading causes of disability in the elderly (Guccione et al., 1994; Pearle et al., 2005). As mentioned previously, because the process of knee osteoarthritis is irreversible (Vigorita et al., 2008) and the personal and societal costs of the disease are high (Hunter et al., 2014; Uhlig et al., 2010; Xie et al., 2008), disease prevention and identification of at-risk groups is critical.

Specifically, certain populations and occupational groups are at increased risk for the development of knee osteoarthritis. For example, men and women in the Beijing Osteoarthritis Study (China) demonstrated a higher prevalence of knee osteoarthritis than individuals in the Framingham study (United States) (Zhang et al., 2004). It was determined that a significant proportion of this difference in prevalence could be accounted for by the time spent in non-occupation related squatting postures at age 25 in the Beijing Study participants (Zhang et al., 2004). Other studies have also found an increased incidence of knee osteoarthritis in Asian as

compared to Western populations (Yoshida et al., 2002). In addition, African Americans living in the United States are also at increased risk for the development and progression of knee osteoarthritis, although the reason for this difference is not known (Anderson & Felson, 1988; Kopec et al., 2013; Mazzuca et al., 2007). Kneeling and squatting associated with certain religious practices may also be linked to an increased risk for knee osteoarthritis, although the mechanisms are not clear (Chokkhanchitchai, Tangarunsanti, Jaovisidha, Nantiruj, & Janwityanujit, 2010). Another group that may be at increased risk for the development of knee osteoarthritis is elite athletes (Kuijt, Inklaar, Gouttebarge, & Frings-Dresen, 2012; Kujala, Kaprio, & Sarna, 1994; Tveit, Rosengren, Nilsson, & Karlsson, 2012); however, the evidence is conflicting (Iosifidis, Tsarouhas, & Fylaktou, 2014), and rates of knee osteoarthritis in athletes may approach rates seen in the general population once injuries sustained through sport participation are accounted for (Shrier, 2004).

Certain occupational groups also demonstrate an increased prevalence of radiographic and symptomatic knee osteoarthritis. Miners (Kellgren & Lawrence, 1952), shipyard workers (welders, sheet metal workers, general labourers) (Felson, 1994; Partridge & Duthie, 1968), and floor layers and tile setters (Coggon et al., 2000; Cooper et al., 1994; Jensen, Rytter, Marott, & Bonde, 2012; Manninen, Heliövaara, Riihimäki, & Suomalainen, 2002; Sandmark, Hogstedt, & Vingård, 2000; Tanaka, Smith, Halperin, & Jensen, 1982; Thun et al., 1987) all have a demonstrated increased risk of tibiofemoral or patellofemoral osteoarthritis. The physical demands in these occupations that are considered risk factors include heavy lifting or carrying, and kneeling or squatting (or stair-climbing – Cooper et al., 1994), which result in a marked increase in the risk for knee osteoarthritis, especially when both types of exposures are combined (Amin et al., 2008; Cooper et al., 1994; Ezzat & Li, 2014) (Table 3.3). Knee-straining postures

and heavy lifting and carrying have also been independently associated with an increased risk of meniscal tears, which may also contribute to knee osteoarthritis development in occupational kneelers (Baker, Bennell, Stillman, Cowan, & Crossley, 2002).

Table 3.3

Interactions between Occupations Involving Heavy Lifting and Repetitive Knee Flexion on the Risk of Knee Osteoarthritis

		Kneeling, squatting or stair-climbing	
		No	Yes
Heavy Lifting	No	1.0 [203]	2.5(1.1-5.5) [45]
	Yes	0.2(0.1-1.6) [9]	5.4(1.4-21.0) [13]

Data is presented as: odds ratio (95% confidence interval) [number of participants] (adapted from Cooper et al., 1994)

Although most research suggests a link between occupations that involve repetitive knee-bending and heavy loads, not all studies have found this association (Ezzat & Li, 2014; McWilliams, Leeb, Muthuri, Doherty, & Zhang, 2011). While different definitions of osteoarthritis presence may account in part for this discrepancy, how the exposure to knee-straining postures is quantified may also contribute to the dissimilar outcomes. For example, some studies use a cut-off of greater than 30 minutes to define prolonged exposure; however, one or two hours per day has also been used, as well as the number of days per week the worker is exposed to loads, and even non-numerical measures such as ‘always, often, or never’ descriptors. In a meta-analysis, publication bias was highlighted as a potential contributor to an overestimation of the risks associated with occupational knee osteoarthritis (McWilliams et al., 2011). Based on the literature, it is clear that although there are conflicting findings, occupational exposure is a significant risk factor for the development of knee osteoarthritis that deserves further research attention.

3.2 The Etiology of Knee Osteoarthritis

A number of factors contribute to the initiation and progression of knee osteoarthritis, including those that are biological and biomechanical in nature. Many authors have generated hypotheses on the etiology and pathogenesis of osteoarthritis in an attempt to address the variety of contributing variables. However, it is impossible to state with certainty, which, if any, provides the best insight into osteoarthritis initiation and development (Figure 3.4 a, b, c, d). Biomechanical factors that are thought to contribute to the risk of knee osteoarthritis development include obesity, occupation, sports activity, joint injury, ligamentous laxity, impaired proprioception, muscle dysfunction, and joint malalignment and developmental abnormalities (Garstang & Stitik, 2006). This section will address the biomechanical factors that are thought to contribute to the initiation and progression of knee osteoarthritis, with specific attention to causes associated with occupational knee-loading in high knee flexion postures (> 120° of flexion) such as kneeling and squatting. Acknowledgment and further details on the role of biological factors in knee osteoarthritis can be found in Appendix A: Biological Contributors to Knee Osteoarthritis.

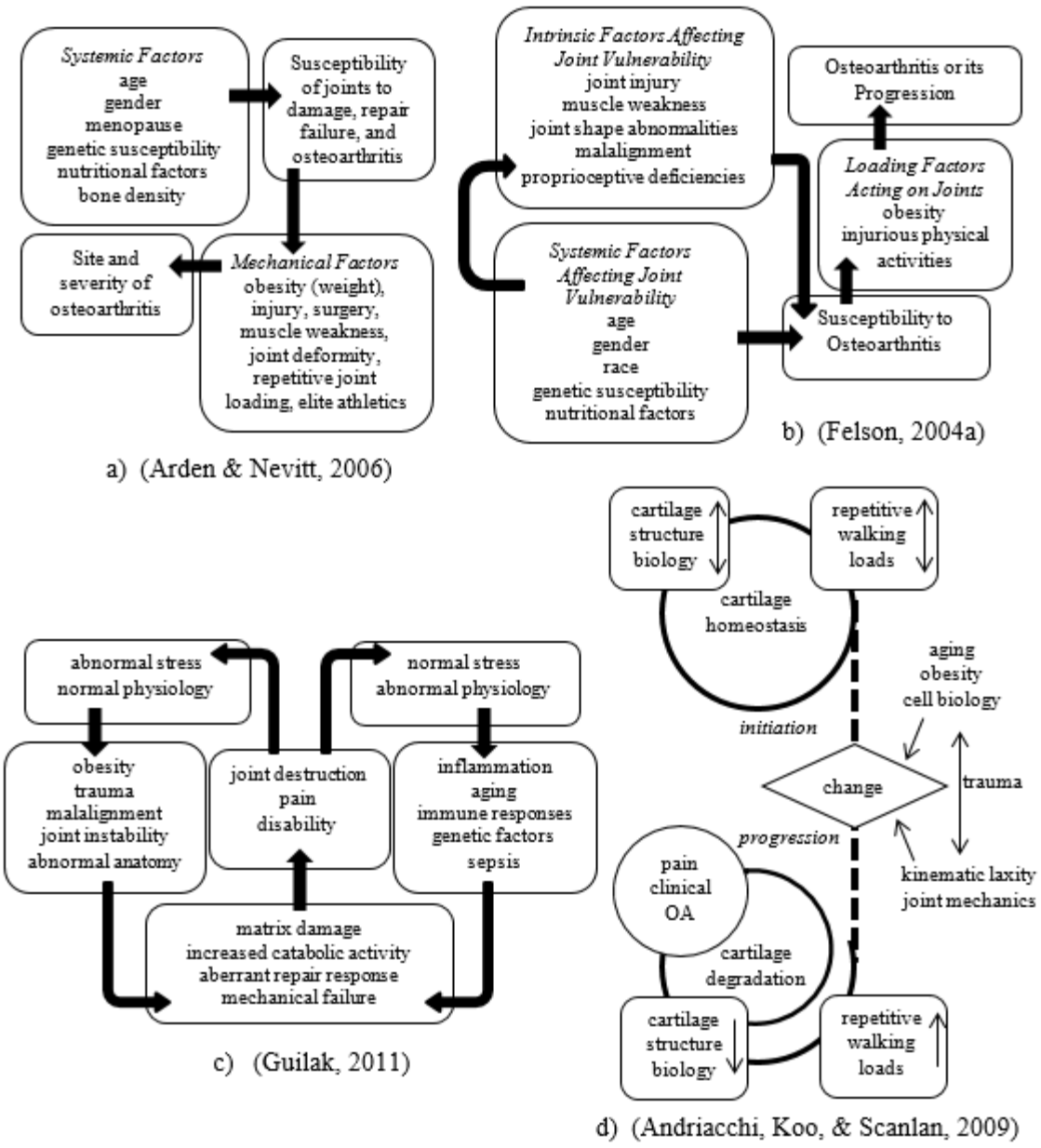


Figure 3.4 Examples of proposed mechanisms and modifying factors of knee osteoarthritis initiation and development.

NOTE: all figures are adapted from the respective authors cited.

3.2.1 Biomechanical contributions to knee osteoarthritis in occupational high knee flexion postures.

Although there is no clear consensus as to whether osteoarthritis is mechanically driven and biologically mediated, or vice versa, it is accepted that knee osteoarthritis is a disease with mechanical contributions. Two main theories exist to explain why mechanical factors contribute to the initiation and progression of knee osteoarthritis. The first theory is commonly referred to as the wear-and-tear theory. The wear-and-tear theory suggests that repetitive knee use alone is sufficient to result in the gradual wearing away of the articular cartilage (Hurley, 1999; Lane, 1995; Shrier, 2004). When applying this theory to occupations that require kneeling and squatting postures, joint damage is hypothesized to occur through two possible mechanisms:

1. Repeated loading in a high knee flexion posture directly results in degradation of the cartilage (Cooper et al., 1994), which upsets the balance between cartilage anabolism and catabolism.
2. Repeated loading increases the risk of meniscal tears (Cooper et al., 1994). Meniscal tears impair the healthy dispersion of load across the knee thereby promoting further joint injury and degradation of tissue (Badlani, Borrero, Golla, Harner, & Irrgang, 2013; Englund et al., 2009; Englund, Guermazi, & Lohmander, 2009; Kim, Bae, & Lim, 2013; Muthuri, McWilliams, Doherty, & Zhang, 2011).

To address the hypothesis that repeated loads in knee-straining postures causes cartilage degeneration, research has shown that tibiofemoral loading may be high when the knee is highly flexed, such as in kneeling and squatting (Dahlkvist, Mayo, & Seedhom, 1982; Nagura, Dyrby, Alexander, & Andriacchi, 2002; Thambyah, Goh, & De, 2005). It is also understood that static loading, which may occur in prolonged kneeling or squatting, is detrimental to the health of knee

joint cartilage (Griffin & Guilak, 2005; Lin et al., 2006). Therefore, it is possible that repeated loading associated with prolonged occupational high knee flexion postures may contribute to the development of knee osteoarthritis. To address the second hypothesis, workers who kneel frequently are at greater risk for the development of symptomatic meniscal tears compared to workers in non-knee-straining occupations (Jensen et al., 2012; McMillan & Nichols, 2005; Reid, Bush, Cummings, McMullin, & Durrani, 2010; Snoeker, Bakker, Kegel, & Lucas, 2013). Therefore, this difference in meniscal tear risk may contribute to why occupational kneeling is associated with an increased risk for knee osteoarthritis development.

While the wear-and-tear theory seems to be a viable explanation for occupation-mediated knee osteoarthritis, this theory has been criticized for being too simplistic (Hurley, 1999). In challenging the traditional view, it has been hypothesized that muscle dysfunction is a contributor to knee osteoarthritis development. The muscle dysfunction hypothesis is based on the understanding that appropriate muscle contraction is a significant mechanism of force attenuation at the knee during upright weight-bearing activities such as walking and running (as discussed in 3.1.1). Should quadriceps activation fail to efficiently attenuate force at the knee – whether due to an impairment in proprioception, muscle fatigue or atrophy, or injury – greater forces would be transmitted to the bone, cartilage, and menisci. In particular, large-amplitude impulse forces are more likely to cause injury to the bone and articular surfaces than loads applied at slower rates, even when the force is higher (Brandt et al., 2009). In addition, forces do not need to be supra-physiologic in magnitude – a physiological load applied at a high rate is sufficient to cause micro-damage (Radin, 2004). Since micro-fracture of the subchondral bone is thought to be one of the initiating factors of osteoarthritis development (i.e. micro-fracture of the

bone may occur prior to cartilage degeneration), quadriceps muscle dysfunction may therefore contribute to knee osteoarthritis risk. (Shrier, 2004)

How can the theory of muscle dysfunction be associated with knee osteoarthritis risk in occupational knee-straining work, when damage is thought to occur during upright, weight-bearing activities? Although estimates have suggested that workers in knee-straining occupations may spend between 38-66% of the work day on their hands and knees (Jensen et al., 2010), when the workers are not working on the floor, they may be carrying work materials (e.g. tiles, flooring planks, adhesives, portable generators, pails of water or quickset, underlay or subflooring, tools, etc. …). This is important to note because occupations that require both kneeling and heavy lifting or carrying are associated with an elevated risk for the development of knee osteoarthritis (Cooper et al., 1994). Therefore, should working in knee-straining postures cause quadriceps dysfunction, normal force attenuation mechanisms during gait may be altered, thereby resulting in higher rates of loading at the knee, especially when workers are carrying heavy equipment. Although not all authors agree (Henriksen et al., 2006), high rates of loading during gait are commonly associated with increased injury risk (Brandt et al., 2009), thereby theoretically linking occupational kneeling to the initiation and progression of knee osteoarthritis through a muscle dysfunction mechanism. Thus, measurement of the external rate of loading and knee moments during gait following an occupational kneeling exposure are beneficial to determine if knee joint loads are in fact increased. The following sections will further explore the role of proprioception in knee osteoarthritis, the proposed role of the quadriceps muscle group with respect to knee joint health, how alterations in proprioception and quadriceps function could lead to abnormal or excess knee joint loads, and how impaired proprioception and

changes in neuromuscular control could occur as a result of occupational high knee flexion postures.

3.2.2 The role of proprioception and knee osteoarthritis.

The role of joint proprioception in the etiology and pathogenesis of knee osteoarthritis is debated, and complicated by the fact that there is no standard definition for proprioception. Proprioception has been defined in a number of ways – most simply, proprioception is a conscious or unconscious perception of body position and movement (Knoop et al., 2011). Proprioception may also be considered the summation of all neural inputs from muscles, tendons, and joints at the central nervous system which, combined, regulate reflexes and motor control (Hewett et al., 2002). As previously discussed (3.1.1) the knee joint contains a number of mechanoreceptors (Rothwell, 1994) which contribute to joint position and movement sense and are important for coordinated muscle activation patterns and appropriate force attenuation mechanisms at the knee.

Proprioception is implicated in knee osteoarthritis in a number of ways. First, proprioception is thought to protect the knee against injurious movements through reflex responses (Knoop et al., 2011). For example, in animal studies, stretch of the ACL causes a change in the sensitivity of muscle spindle reflex responses (Johansson, Sjölander, & Sojka, 1990; Solomonow et al., 1987). Proprioception also contributes to knee stabilization in static and dynamic movement (Knoop et al., 2011; Schipplein & Andriacchi, 1991). Lastly, proprioceptive afferent information is a critical component of motor learning and the coding of motor control patterns of movement (Hunter, 2009). Therefore, if a protective neuromuscular control mechanism is disturbed, or if afferent information provides inaccurate feedback, the knee may be exposed to high impulse loads during daily activities such as gait (Arden & Nevitt, 2006;

Liikavainio et al., 2007; Segal & Glass, 2011). Based on the understanding of the importance of proprioceptive afferent information in motor control, it has been suggested that poor proprioceptive acuity impairs protective mechanisms during gait and may contribute to joint damage (Knoop et al., 2011). However, whether impaired proprioception is a precursor to knee osteoarthritis development, a symptom of the disease, or both, is not known.

Regardless of whether proprioception is measured by joint position sense (Garsden & Bullock-Saxton, 1999; Hassan, Doherty, Mockett, & Doherty, 2002; Hortobágyi, Garry, Holbert, & Devita, 2004; Hurley, Scott, Rees, & Newham, 1997; Marks, Quinney, & Wessel, 1993) or movement sense (Barrack, Skinner, Cook, & Haddad Jr., 1983; Hewitt, Refshauge, & Kilbreath, 2002; Koralewicz & Engh, 2000; Pai, Rymer, Chang, & Sharma, 1997), most studies indicate that knee osteoarthritis patients have impaired proprioception at the knee compared to controls. Deficits in proprioception at the knee joint may also increase as the disease progresses, and be equally impaired in the unaffected knee in persons with unilateral osteoarthritis (Garsden & Bullock-Saxton, 1999), although these relationships are less clear (Knoop et al., 2011). While it is evident that knee osteoarthritis is associated with impaired joint proprioception, a causal relationship has not been experimentally established. One longitudinal study assessed the proprioceptive acuity of individuals with knee osteoarthritis as well as those with known risk factors for knee osteoarthritis (e.g. obesity, previous knee injury or surgery – whether occupational risk factors were included was not reported). The findings of that study suggested that proprioceptive deficits are not a risk factor for the development and progression of the disease (Felson et al., 2009; Segal, Glass, Torner et al., 2010). Instead, the authors argued that the loss of mechanoreceptors and associated changes in proprioceptive sense are a result of degeneration of the joint subsequent to osteoarthritis initiation.

Although it is possible that proprioception is not associated with knee osteoarthritis onset or progression (Felson et al., 2009; Segal, Glass, Felson et al., 2010), because proprioception is thought to be an important determinant of joint health (Chang, Lee, Zhao, Ren, & Zhang, 2014) a number of studies have investigated the potential benefit of training programs to improve proprioception in individuals with knee osteoarthritis. For example, studies have shown that physiotherapist-supervised proprioceptive training in patients with knee osteoarthritis may improve knee position and movement senses, and improve symptoms of pain and disability (Diracoglu, Baskent, Celik, Issever, & Aydin, 2008; Hurley & Scott, 1998; Lin, Lin, Chai, Han, & Jan, 2007). Therefore, while the mechanism of proprioceptive decline associated with knee osteoarthritis is not known, these findings suggest that poor proprioceptive acuity at the knee contributes to the functional disability and pain experienced as a result of knee osteoarthritis, and that proprioception is modifiable.

A number of other factors have also been shown to alter knee joint proprioception in addition to training and disease. For example, muscle mechanoreceptors are thought to have a significant contribution (Hiemstra, Lo, & Fowler, 2001; Lattanzio & Petrella, 1998; Proske, Wise, & Gregory, 2000). Therefore, researchers have attempted to determine whether muscle stretching affects knee proprioception. Although stretching has only been shown to evoke short-lasting changes in passive muscle stiffness (Madding, Wong, Hallum, & Medeiros, 1987; Rosenbaum & Hennig, 1995; Toft, Espersen, Kalund, Sinkjaer, & Hornemann, 1989), the subjective improvement in stretch-tolerance following a stretching regimen suggests an adaptive sensory effect even though the muscle itself is no more extensible (Ben & Harvey, 2010; Björklund, Hamberg, & Crenshaw, 2001; Larsen et al., 2005). When tested, studies indicate that stretching induces a variety of proprioceptive effects. A study on proprioceptive-neuromuscular

facilitation stretching of the quadriceps and hamstrings found that participants were slower to detect knee movement following the stretch (Streepey et al., 2010). The observed decrease in proprioceptive acuity during this type of stretching, which requires the individual to contract the muscle while the muscle is lengthened, corresponds with early findings that this stretch/activation combination causes decreased sensitivity of the muscle spindles (Gregory et al., 1990). In contrast to these findings, a study on the effects of static stretching found that there were no effects of stretch when testing the knee at 20° of flexion. In addition, the absolute angular error of joint angle reproduction at a testing angle of 45° actually improved by between 2° and 2.8° following static stretching of the quadriceps, hamstrings and adductor muscles (Ghaffarinejad, Taghizadeh, & Mohammadi, 2007). Therefore, while the outcomes of these studies are inconsistent, muscle stretching does appear to affect knee joint proprioception.

Ligaments also contribute to knee joint proprioception. While ligaments were historically considered to function solely as mechanical restraints, ligaments are also important sensory organs that monitor joint proprioceptive information (Johansson, Sjolander, & Sojka, 1991a; Johansson, Sjolander, & Sojka, 1991b; Solomonow, 2006). In practice, the role for ligamentous contribution to joint proprioception is supported by studies that have demonstrated proprioceptive deficits individuals with lax joints (Barrack, Skinner, Brunet, & Cook, 1984; Marks et al., 1993), and in individuals who are ACL-deficient (Carter, Jenkinson, Wilson, Jones, & Torode, 1997).

Other factors that affect proprioception include fatigue (Björklund, Crenshaw, Djupsjöbacka, & Johansson, 2000), attention (Bennell, Wee, Crossley, Stillman, & Hodges, 2005; Goble, Mousigian, & Brown, 2012), and age. In fact, age has been shown to result in proprioceptive deficits regardless of disease status (Barrack et al., 1983; Marks et al., 1993;

Skinner, Barrack, & Cook, 1984); however, the concept that proprioception decreases with age has been challenged by a study using a weight bearing test of proprioception which did not indicate a significant proprioceptive decline with age (Bullock-Saxton, Wong, & Hogan, 2001).

It is likely that the rampant discrepancies in study outcomes in terms of whether proprioception is a modifying factor in knee osteoarthritis risk are at least partially the result of the methods used to measure proprioception. No standardized method exists to measure proprioception and a multitude of techniques are used (Lephart & Fu, 2000). Also, studies that measure joint position sense – often tested as the ability to reproduce a target knee angle – and studies that measure motion sense – the threshold to detection of passive movement of the joint – are poorly correlated (Grob, Kuster, Higgins, Lloyd, & Yata, 2002; Stillman & McMeeken, 2001). This difference has been hypothesized to exist because active tests (such as a position sense test where the participant actively produces then re-produces a target knee angle) stimulate both the muscle spindles and joint mechanoreceptors, whereas passive tests (motion detection tests) minimally stimulate muscle spindles (Felson et al., 2009). In addition, tests of proprioception in weight bearing postures may be affected by confounding factors such as balance and muscle strength (Knoop et al., 2011). Clearly, position sense is not equal to movement sense, and it is ill advised to treat studies that test these two different aspects of proprioception as the same measure.

Furthermore, measures of proprioception may also be limited by the inherent nature of the tests used. Most methods to measure proprioception are a conscious perception of position sense or movement detection threshold; however, during normal daily activities such as gait, conscious perception is generally not required (Bennell, Wrigley, Hunt, Lim, & Hinman, 2013). In addition, even if an individual is able to accurately perceive proprioceptive information,

perception does not necessarily translate into efficient use of this information by the nervous system to control movement (Bennell, Hinman, Wrigley, Creaby, & Hodges, 2011). Also, the change in angular error observed between patient and control groups can be very small (Table 3.4). For example, in knee osteoarthritis patients, values can range from less than 1° to 4°, making it difficult to determine if these changes are valid and relevant, or simply due to measurement error. Therefore, caution should be taken when measuring proprioception based on current methods.

Table 3.4

Proprioceptive Acuity in Knee Osteoarthritis Patients in Non-Weight Bearing Tests of Knee Position Sense

Study	Target Knee Angle	Age-matched Control Group Error	Osteoarthritis Patient Group Error	Difference
Baert et al., 2013	20°	2.40 ± 1.81°	2.84 ± 1.56°	0.44°
Barrack et al., 1983	65 – 85°	4.6 ± 2.2°	8.6 ± 4.8°	4°
Bayramoglu, Toprak, & Sozay, 2007	45°	5.20 ± 3.80°‡	6.29 ± 4.12°‡	1.09°
Bennell et al., 2003	20°, 40°	-	2.3 – 3.2°	-
Birmingham et al., 2001	30 – 60°	-	2.89 ± 1.14°	-
Erden, Otman, Atilla, & Tunay, 2003	30°	1.34 ± 0.56°	2.38 ± 1.46°	1.04°
Felson et al., 2009	0 – 90°	-	3.9° *	-
Hall, Mockett, & Doherty, 2006	20 – 50°	5.10 ± 3.02°	5.84 ± 3.20°†	0.74°
Hassan, Mockett, & Doherty, 2001	0 – 90°	7.9° (6.9, 8.9) ǂ	12.0° (10.5, 136.6) ǂ	4.1° (2.2, 6.1) ǂ
Hortobágyi et al., 2004	15, 30, 45, 60, 75°	15°: 3.1 ± 1.3°	15°: 6.7 ± 2.8°	15°: 3.6°
Hurley et al., 1997	0 – 90°	30°: 2.8 ± 1.3°	30°: 6.0 ± 2.2°	30°: 3.2°
Lund et al., 2008	not reported	2.29° (2.00, 2.57) ǂ	3.57°(3.22, 3.92) ǂ	1.28° (0.84, 1.73) ǂ

‡ right leg data reported here only
ǂ variance is presented as a 95% confidence interval
* median angular error of 2,440 knee osteoarthritis patients
† radiographic knee osteoarthritis AND knee pain

Evidently, proprioception is a complicated sense that is affected by a number of factors and may be difficult to accurately quantify. At the same time, proprioception is an important component of motor control that deserves research attention. While current methods of measuring proprioception are not ideal, and research is conflicting as far as the contribution to joint degeneration, proprioception is an important measure to investigate based on the concept

that altered sensory afferent input could contribute to the mechanism of change leading to alterations in neuromuscular control. Because muscle activation onset and magnitude have been shown to alter joint loading patterns (3.1.1), proprioceptive deficits induced by an occupational kneeling exposure – whether through ligamentous, muscular, or capsular stretch, or another mechanism – could promote injurious loading patterns during gait. Therefore, in addition to measuring the knee adduction moment and vertical loading profile during gait, a measure of knee joint position sense was also included in an attempt to better understand the potential neuromuscular control mechanism linking occupational kneeling and the development of knee osteoarthritis.

3.2.3 Quadriceps dysfunction: Symptom vs. cause of knee osteoarthritis.

In healthy individuals, muscle contributes to force attenuation at the knee (Hurley, 1999; Segal & Glass, 2011). As discussed previously (3.1.1), appropriate force attenuation relies partially on the quadriceps to control the rate of loading at the knee to prevent damage to the subchondral bone and articular surfaces (Herzog, Longino, & Clark, 2003; Jefferson et al., 1990). Therefore, it is possible that quadriceps strength and activation patterns may act to either promote or protect against degenerative joint disease. For example, the incidence of knee pain is lower in individuals who use eccentric quadriceps contraction to control the rate of joint loading at initial contact of gait (Hurley, 1999). In addition, quadriceps weakness and atrophy are seen in individuals with knee osteoarthritis when compared to healthy controls (Hurley et al., 1997; Lewek, Rudolph, & Snyder-Mackler, 2004; O'Reilly, Jones, & Doherty, 1997; Sharma, Pai, Holtkamp, & Rymer, 1997; Slemenda et al., 1997). Clearly, the quadriceps muscle group is linked to knee joint health; however, it is unknown whether quadriceps group atrophy or dysfunction are potential causes of knee osteoarthritis, or if they occur as a result of the disease.

The research is conflicting as to whether quadriceps weakness and changes in neuromuscular control are symptoms or initiating factors of knee osteoarthritis. For example, knee pain and inflammation can cause neuromuscular inhibition and atrophy of type II muscle fibres (Felson, 2004b), and these effects can occur even when pain is not present (Rutherford, Jones, & Newham, 1986; Stokes & Young, 1984). In addition, quadriceps strength has not been shown to be protective against radiographic knee osteoarthritis initiation (Hunt et al., 2010; Roos, Herzog, Block, & Bennell, 2011). These findings would suggest that muscle dysfunction is a symptom of joint degeneration.

In contrast, some researchers believe that quadriceps weakness and dysfunction contributes to knee osteoarthritis risk. Longitudinal studies on human participants have found that quadriceps weakness may precede knee osteoarthritis onset (Hootman, Fitzgerald, Macera, & Blair, 2004; Segal et al., 2009; Slemenda et al., 1998), and quadriceps strength may protect against symptomatic knee osteoarthritis in women (Segal et al., 2009). Beyond the investigation of simple muscle strength, quadriceps activation patterns may be a more important determinant of knee osteoarthritis risk (Hurley, 1999; Segal, Findlay, Wang, Torner, & Nevitt, 2012). For example, animal models suggest that quadriceps muscle dysfunction precedes joint degenerative changes (Herzog et al., 2003). In addition, poor neuromuscular control of the quadriceps was found in knee osteoarthritis patients in a study measuring force production accuracy in concentric and eccentric contractions (Hortobágyi et al., 2004). Although the authors were unable to determine whether neuromuscular control deficits precede or follow joint degeneration, they suggested that neuromuscular activation and muscle physiology of the knee extensors may be more important than muscle strength in knee osteoarthritis risk. Therefore, while the evidence is

very limited, there is some indication that neuromuscular control of the quadriceps may be an important determinant of knee osteoarthritis risk.

Based on the body of research, while quadriceps strength and neuromuscular dysfunction may be outcomes of the disease, there is also evidence to suggest that these factors may contribute to osteoarthritis initiation. Therefore, measures of quadriceps onset timing and magnitude of activation during gait are important to gain insight into changes in neuromuscular control following a simulated occupational kneeling exposure. Because altered neuromuscular control can affect joint loading during gait, changes in quadriceps activation after kneeling may indicate a role for quadriceps dysfunction in increased rates of knee osteoarthritis observed in occupational kneelers.

3.3 The Effects of Occupational High Knee Flexion Postures on Gait Parameters

This project is not the first to analyze the effects of a prolonged kneeling exposure on gait parameters. In the first known study, 10 participants were asked to hold a static, full-flexion kneeling posture for three, 10-minute cycles with 5 minutes of rest between cycles (Kajaks, 2008; Kajaks & Costigan, 2015). Participants were instrumented unilaterally with kinematic motion tracking markers and EMG sensors were used to record the activation of the lower limb muscles. Gait measurements were taken before and after the kneeling protocol. The prolonged, full-flexion kneeling exposure resulted in significant differences in all gait parameters (knee adduction moment, flexion moment, and knee flexion angle) and in muscle co-contraction and total muscle activity (a measure of the integrated EMG signal). While the authors did report a muscle co-contraction index, changes in the external loading rate or the timing of muscle activation onset – both of which have been shown to be related to joint loads during gait (Brandt et al., 2009) – were not reported. In addition, the kneeling exposure was static and therefore not

likely a realistic representation of an occupational kneeling exposure, the number of participants was low, and only males were tested. The study was also limited in that it was purely exploratory – mechanisms to explain the observed effects, while proposed, could not be causally determined. As well, gait speed was not controlled for. While in some respects allowing participants to select their preferred speed may be advantageous, changes in gait speed can affect measures such as the knee adduction moment (Baliunas et al., 2002; Lelas, Merriman, Riley, & Kerrigan, 2003).

In a more recent study, the gait kinematics of workers who are exposed to knee-straining postures (KS) – defined as kneeling and squatting – were compared to workers who are not exposed to knee-straining postures (n-KS) (Gaudreault, Hagemeister, Poitras, & de Guise, 2013). When walking on a treadmill, KS workers demonstrated a smaller knee angle range-of-motion in the sagittal plane, with a greater mean flexion angle at foot contact. In addition, KS workers' knees were adducted throughout the entire gait cycle (stance and swing), whereas n-KS workers' knees exhibited both abduction and adduction angles. While this study provided evidence for kinematic differences between the two groups of workers, they were unable to report on the adduction moment during gait. The external knee adduction moment has been associated with medial knee osteoarthritis progression (Miyazaki et al., 2002) and may have provided valuable insight into the risk for osteoarthritis development between groups. The authors acknowledged that future work is needed to better determine the causal relationship between knee-straining postures and observed kinematic differences between the groups. Given the extremely limited data available on the effects of occupational knee-straining postures on gait parameters, this is clearly a topic in need of research attention.

3.4 Potential Mechanisms Linking Occupational High Knee Flexion Postures with Changes in Neuromuscular Control

There are a number of possible mechanisms by which an occupational kneeling exposure could alter neuromuscular control, including ligamentous creep or muscle stretch (2.0, Figure 2.1). Of the potential mechanisms, ligamentous creep may be the most likely to be responsible for any neuromuscular control changes following a kneeling exposure. Although, ligamentous creep has not been directly linked to proprioceptive deficits (i.e. specifically joint position or movement sense deficits), it has been described as causing 'feedback signal corruption' (Solomonow, 2006). In addition, as previously noted (3.2.2), individuals with lax joints or who are ACL-deficient exhibit impaired knee joint proprioception (Barrack et al., 1984; Carter et al., 1997; Marks et al., 1993). Occupational kneeling postures have been shown to exert tension in the ligaments, which could induce a creep response, and in turn affect neuromuscular control and joint proprioception. This section will discuss in detail ligament function in high knee flexion postures, how sensory signals may be altered by ligamentous creep, and the potential for muscular stretch to induce similar changes.

Specific knowledge of the effects of a full-flexion kneeling posture on muscle and ligaments is almost non-existent. Fortunately, however, data on ligament function is becoming more readily available as the role of ligaments in the high flexion range has come under investigation in applications towards improving total knee arthroplasty function in kneeling (Belvedere et al., 2012; Hosseini et al., 2015; Park, Hosseini, Tsai, Kwon, & Li, 2015; Park et al., 2006). In the most straightforward sense of ligament function, it is understood that certain fibres of both the anterior and posterior cruciate ligaments are thought to always be in a state of tension (Fuss, 1989; Van Dommelen & Fowler, 1989) in order to contribute to joint stability

throughout the full joint range of motion (Solomonow, 2006). In particular, ligaments act as 'check-reins' near or at the end ranges of joint motion. Therefore, it is reasonable to hypothesize that joint ligaments would experience some tension when the knee is fully flexed, such as in a kneeling posture. For the PCL, this is evident as one mechanism of PCL rupture is when an individual falls to the floor when the knee is hyperflexed and the ankle is plantarflexed (Prentice, 2011), which would suggest that high forces act on the PCL in high knee flexion postures.

At least one cadaveric study has examined the forces on knee joint ligaments in high knee flexion postures. In this study, forces acting on ligaments were evaluated while including simulated muscle loads from the quadriceps and hamstrings throughout the knee range of motion, up to 150° of knee flexion (Li et al., 2004). At 150° of knee flexion the force on the ACL peaked at 30 N (this peak force occurred when only the quadriceps load was applied). Results from the same study revealed that force on the PCL at the highest degree of flexion was approximately 35 N when accounting for muscular loading. For both cruciate ligaments, peak forces were not observed in the high flexion posture, but at more extended knee positions. The effects of supporting body weight, as would be observed in a kneeling posture, were not evaluated. A similar study modelled ligamentous loads from 60° to 140° of knee flexion and validated the model against a cadaveric knee (Yang, Wickwire, & Debski, 2010). Unlike the previous group, this study evaluated forces acting on the ligaments when applying external anterior-posterior and compressive loads, as opposed to applying forces based on muscle actuation. The highest experimental ligament loads were still found at smaller knee flexion angles, compared to the highest flexion angle evaluated (e.g. for both the ACL and PCL, forces were greatest at 90° flexion). Based on the cadaveric data, at 140° of knee flexion, the ACL exhibited the greatest loading magnitude (24.5 N), followed by the MCL (10.2 N), PCL (3.9 N),

and LCL (1.1 N). It has been hypothesized that forces at high knee flexion are reduced in the PCL due to a loss in biomechanical advantage of this ligament in restricting posterior tibial translation and compression of tissues (Hofer, Gejo, McGarry, & Lee, 2011). Based on this work, it is apparent that ligamentous loads, while perhaps not maximal in high knee flexion postures, are still present, and prolonged application of these loads may have the potential to induce ligamentous creep.

Ligamentous laxity or creep that could occur as a result of a prolonged kneeling exposure may have effects on neuromuscular control. Solomonow (2006) refers to creep as causing 'feedback signal corruption,' that can induce errors in movement control and precision. The proposed mechanism of these corrupted signals is due to acute inflammation of the ligament following micro-rupture of the collagen fibers, which can occur with static or repetitive loading of a ligament, even when loading is within the physiological limits (Solomonow, 2006; Solomonow, 2009). There are a number of examples of neuromuscular changes resulting from ligamentous creep. For example, a study that induced ACL laxity found that ligament creep caused alterations in muscle activation patterns and force production capacity of the knee flexors and extensors (Chu et al., 2003). Similarly, experimentally induced PCL creep altered muscle activation magnitude and co-activation patterns during isokinetic knee flexion and extension tasks (Cheng, Zhang, Shan, & Wang, 2014). Unfortunately, the studies that induced ACL/PCL creep did not evaluate the creep recovery response, and no data exists on whether ligamentous creep occurs after a kneeling exposure. However, the creep response of ligaments of the lumbar spine has been evaluated. These studies indicate that prolonged loading can induce creep that persists even in healthy young participants (McGill & Brown, 1992), and cyclic loading may also result in persistent creep and neuromuscular disorder (Courville et al., 2005). Therefore, it is

possible that a similar creep recovery response would be observed in ligaments of the knee, which may induce similar neuromuscular control changes.

While data on the effects of high knee flexion postures on ligaments is limited, there is arguably even less evidence for neuromuscular control changes associated with muscle stretching protocols. However, as previously briefly noted (3.2.2), muscle stretching may exert some effect on knee joint proprioception. In addition, a recent study evaluating the effects of a stretching protocol on knee joint laxity found that a relatively short static stretching protocol (two rounds of a series of four stretches, where each stretch was held for 20 seconds) induced a significant increase in anterior tibial translation compared to a control group when measuring laxity with an arthrometer (Baumgart, Gokeler, Donath, Hoppe, & Freiwald, 2015). Although the effects of laxity induced by stretching on neuromuscular control were not evaluated in that study, based on the findings of proprioceptive deficits in individuals with lax joints (Barrack et al., 1984; Marks et al., 1993), and neuromuscular control changes observed following experimentally induced ligamentous laxity (Cheng et al., 2014; Chu et al., 2003; Sbriccoli, Solomonow, Zhou, Lu, & Sellards, 2005), it could be hypothesized that similar neuromuscular changes would be found after the stretching protocol.

3.5 Summary of the Relevant Literature

Knee osteoarthritis causes pain and disability and is a significant economic burden on society (Hunter et al., 2014; Xie et al., 2008). Occupations that require prolonged kneeling, especially when combined with lifting or carrying, appear to put workers at significantly greater risk for knee osteoarthritis development than kneeling or carrying alone (Amin et al., 2008; Cooper et al., 1994; Ezzat & Li, 2014). It is possible that occupational kneeling affects gait parameters such as the external knee adduction moment, loading rate, and quadriceps activation.

This proposed mechanism has some basis in the muscle dysfunction theory of knee osteoarthritis, which proposes that poor neuromuscular control results in impulsive joint loads at the knee. These impulsive loads may cause microfractures of the subchondral bone and subsequent cartilage degeneration and eventually total joint dysfunction (Shrier, 2004). Based on the understanding of the interdependency of sensory afferent information and motor control, proprioceptive deficits (e.g. via ligament creep) may be linked to any observed alterations in motor control observed during gait. Therefore, the effects of occupational kneeling on gait and knee joint proprioception deserve further investigation to evaluate the potential changes in upright, weight-bearing activities, which may contribute to the increased risk for knee osteoarthritis documented in this population.

4.0 Methods

4.1 Study Population

Twenty female and twenty male participants were recruited (Table 4.1 for demographics). It was important to collect both male and female participants because there are known differences in proprioception, neuromuscular control, and incidence of knee osteoarthritis between the sexes (Arden & Nevitt, 2006; Bennell et al., 2013; Moore et al., 2002; Nicolella et al., 2012). In addition, both men and women may be employed in occupations that require high flexion knee postures, such as kneeling. Therefore, measurement and analysis of both groups is necessary to determine the effects of an occupational kneeling exposure on the outcome measures.

Table 4.1

Participant Demographics

	All	**Male**	**Female**
Age (years)	21.4 (2.5) [18, 28]	21.4 (2.4) [18, 26]	21.4 (2.6) [18, 28]
Weight (kg)	68.8 (16.1) [49.1, 136.0]	76.6 (18.7) [52.7, 136.0]	61.0 (7.1) [49.1, 76.6]
Height (m)	1.69 (0.10) [1.52, 1.92]	1.77 (0.10) [1.52, 1.92]	1.62 (0.10) [1.53, 1.79]
Arm span (m)	1.70 (0.11) [1.51, 1.98]	1.79 (0.08) [1.64, 1.98]	1.61 (0.06) [1.51, 1.74]
Preferred walking speed (treadmill) (m/s)	1.19 (0.19) [0.82, 1.74]	1.23 (0.19) [0.88, 1.74]	1.14 (0.18) [0.82, 1.49]
Ely's angle (°)	56 (18) [26, 113]	54 (21) [26, 113]	58 (16) [31, 84]
Self-reported activity (days/week)	3.9 (1.6) [1, 7]	4.3 (1.6) [1, 7]	3.6 (1.5) [1, 7]

Values are reported: mean (standard deviation) [min, max].

All participants were be between the ages of eighteen and thirty and had no current lower extremity injury, nor did they admit to current or previous knee ligament or meniscus injuries, conditions that impair balance, or regular exposure to knee-straining postures. For a more detailed description of the exclusion criteria, see Appendix B: Participant Exclusion Factors.

4.2 Experimental Design

Data collections took place in the Biomechanics of Human Mobility Lab, which is located in room 1405 of Burt Mathews Hall at the University of Waterloo. Participants wore gym shoes (i.e. low-heeled running shoes) and loose athletic shorts. After providing informed consent, participants completed a health screening questionnaire (Appendix C: Participant Screening Questionnaire), a short questionnaire about their footwear, mood, and physical activity (Appendix D: Participant Information Questionnaire), and a battery of leg dominance tests. The leg dominance test included kicking a ball, picking up an object from the floor, stamping out an imaginary fire, and tracing the outline of an object on the floor with their foot (Schneiders et al., 2010). Data were only collected from the dominant leg. Participant height, mass, and age were recorded. Participants were then instrumented with surface electrodes over the vastus medialis and performed maximal voluntary contractions as well as a quiet rest trial (4.5). Vastus medialis was selected as a representative muscle to evaluate quadriceps function because it has been reported to contribute to the presence or absence of heel-strike transients in gait (Liikavainio et al., 2007), and the signal-to- noise ratio for the EMG signal of rectus femoris can sometimes be too small during gait to accurately and reliably identify activation onset, which was one of the variables of interest. These trials were followed by instrumentation and digitization using the active motion capture system.

Hip flexibility was measured using Ely's test (Peeler & Anderson, 2008; Prentice, 2011) to account for any differences in potential muscle stretch from the kneeling exposure between participants. Specifically, Ely's test is intended as a measure of rectus femoris tightness. Participants lay prone on a massage table with the muscles relaxed while the investigator palpated the ASIS of the ipsilateral hip. When the participant indicated that they were relaxed,

the investigator slowly brought the participant's foot towards their buttocks through passive knee flexion and the investigator noted the point at which they felt the ASIS begin to reduce pressure on the fingertips (Burns, personal communication, fall 2014; Prentice, 2011), indicating a lift of the ipsilateral hip from the table. The time of this feeling of reduced pressure was noted using an external trigger. The trial was collected using the 3D motion capture system so that the knee flexion angle at the instant of reduced pressure could later be recorded following kinematic processing.

Next, baseline values were collected. A baseline value of knee proprioceptive acuity was measured using the average of three trials of an active knee angle reproduction test (4.6). Participants were given at least three trials to familiarize themselves with the procedure prior to instrumentation and before testing began. Tests of knee proprioception were followed by gait trials. Gait trials were constrained to 1.4 m/s (± 0.05 m/s) and were conducted on an 8.5 m walkway. This velocity was selected because it has been shown to be a comfortable walking pace for both men and women in their twenties (Bohannon, 1997; Kumar et al., 2015). In addition, gait velocity is known to affect the external force (Andriacchi, Ogle, & Galante, 1977), the rate of loading (Chang et al., 2012) and the moments at the knee (Baliunas et al., 2002; Lelas et al., 2003); therefore, it was necessary to keep gait speed consistent at all measurement time points to reduce the potential effects of walking speed on the outcome measures. It should be acknowledged that by controlling for gait speed, the introduction of an external load may alter the cadence of gait. Gait speed was determined using photoelectric timing gates spaced equally on either side of the force platforms (3-meter inter-gate distance). Only trials with clean contact of the dominant foot on a single force plate and a walking speed of 1.4 m/s (± 0.05 m/s), as indicated by the timing gates, were used in analysis. A minimum of three gait trials were

collected where the participant walked without a load. A minimum of three trials were also collected where the participant walked while carrying a load normalized to 20% of body mass. The load was placed in a crate held in the hands, close to the body to simulate how loads are carried occupationally. This load was selected because it has been commonly used in the literature in studies of load carriage, and has been found to be sufficient to elicit changes in neuromuscular control, such as prolonged activation of certain lower limb muscles and increased external knee moments (Cook & Neumann, 1987; Ghori & Luckwill, 1985; M. Hall, Boyer, Gillette, & Mirka, 2013). Central-stabilization within the crate prevented the load from shifting, and participants were required to carry the crate at sufficient height to prevent interference with hip range of motion (Figure 4.1). The order of unloaded and loaded carries was randomized.

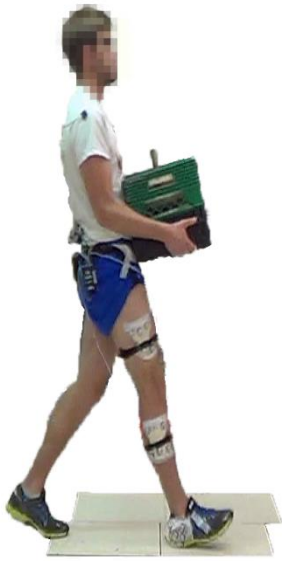


Figure 4.1 The loaded gait task.

Finally, participants performed squat transitions to-and-from the floor as a measure of neuromuscular control (Figure 4.2). Individuals in occupations that require working on the floor must transfer their body (and materials), up and down from the ground many times per day (Figure 4.3). Unpublished data from the Biomechanics of Human Mobility Lab indicates that for

floor installers who work without a partner or team, transitions occur, on average, every 2 – 3 minutes. For example, one floor layer from the study sample transitioned up and down from the floor 120 times during four hours of work. In addition, specific knee joint movement profiles may be associated with injury risk (Frost, 2013; Hewett et al., 2005). Therefore, participants performed a squat transition task where they were asked to descend to and rise from the floor five times as a measure of neuromuscular control.

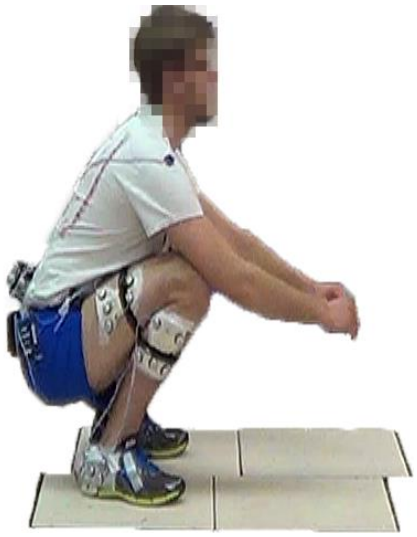


Figure 4.2 An example of the high knee flexion posture during the squat transition used to evaluate neuromuscular control changes following a kneeling exposure.

Because workers may elect to use either a flat foot (Figure 4.2), or a flexed foot method (where the metatarsals remain in contact with the floor but the heels are raised) in order to squat to the floor, and it is possible that some participants may not be able to reach the floor using either of the methods, participants were allowed to select their preferred method of movement. The only instruction given by the investigator was to squat as low to the floor as they possibly could. As mentioned, ankle posture was not constrained, nor was stance width. The squat was also performed at two speeds, whereby participants achieved the appropriate speed by keeping pace with an auditory metronome (44 BPM – slow, and 88 BPM – fast (Almosnino, Kingston, &

Graham, 2013)). For the current study, participants had a one beat static hold at both the top and bottom of the squat transition to facilitate identification of a true 'stop' and 'start' point for both ascending and descending phases of the squat. Therefore, the slow pace dictated a squatting rate of approximately 9 squats/minute, and the fast pace, 17 squats/minute. Slow squats were always performed first in the event that the participant was unable to squat at the fast pace. All participants completed a minimum of five practice squats at each pace prior to instrumentation.

Figure 4.3 Examples of squat transitions seen in occupation.

Following the collection of baseline data, participants completed a 30-minute simulated occupational kneeling exposure. A 30-minute exposure was selected because it is the minimum length of time that has been associated with an increased risk for the development of knee osteoarthritis in kneeling occupations (Cooper et al., 1994) and has been shown to be sufficient to elicit changes in gait kinetics (Kajaks & Costigan, 2015). The kneeling task in the current

study incorporated periods of full-flexion kneeling, as well as single-arm supported kneeling, in an attempt to incorporate postures that are common for floor installers (Figure 4.4).

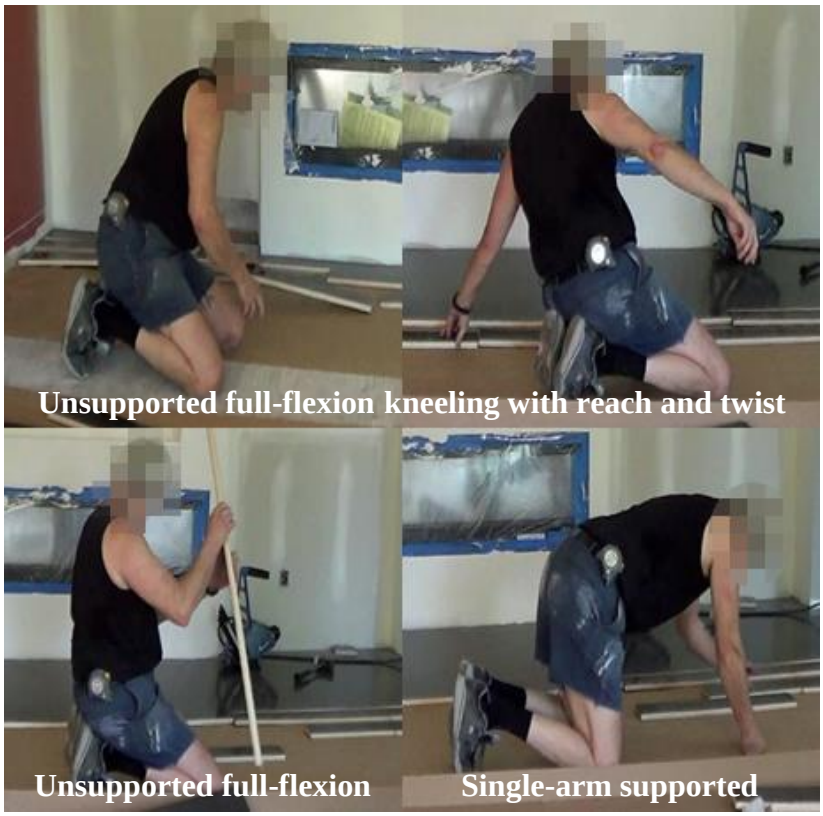


Figure 4.4 Examples of postures adopted in occupations that require kneeling.

The kneeling exposure was designed such that participants alternated between 30 seconds of full-flexion kneeling, and 2 minutes of forward, single-arm supported kneeling. Cycling between full-flexion and single-arm supported kneeling was repeated for 10 minutes. After 10 minutes, participants were given a 30-second standing break. Participants then completed two additional 10-minute exposures, to total 30 minutes. During the single-arm supported kneeling component, participants worked on a card-sorting task. The width of the activity-space for the card-sorting task was normalized to each individual participant's arm-span, and divided into four equal quadrants (Figure 4.5). For each 2-minute block spent in single-arm supported kneeling, the first minute was spent in one of the four quadrants, and the second minute was spent in

another quadrant. Quadrant order was randomized, and equal time was spent in each quadrant over the course of the entire exposure.

Although shoulder reach zone standards are reported for reach dimensions (CSA, Z1004-12, 2012), specific adherence to these reach zones was not applied when defining the work space due to the nature of the fact that participants were kneeling, and therefore reach distances will differ as compared to reaching when in a standing or seated posture at a desk (Figure 4.6). Estimates of the standard reach zones with respect to the experimental set-up used are provided for comparison purposes (Figure 4.5). Participants were encouraged to use as much space as possible within the quadrant. During the full-flexion kneeling component the participants sat quietly and were instructed to attempt to achieve the highest degree of knee flexion possible (i.e. buttocks resting on heels, or approaching heels), without excessive backward trunk lean. Participants were not instructed about ankle posture and were encouraged to assume whichever posture was more comfortable. See Appendix E: Example Kneeling Exposure Protocol for further details.

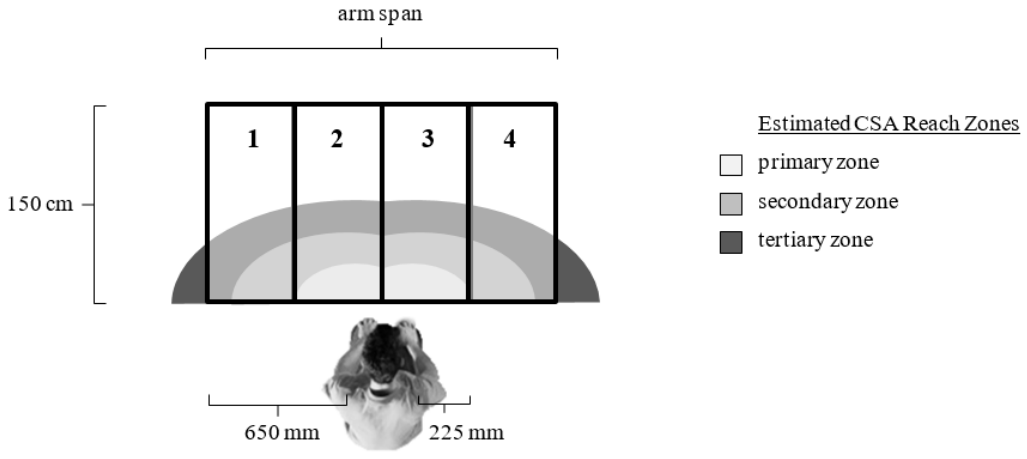


Figure 4.5 Card sorting task set-up.

Note that workspace width values given are based on the average participant measurements - for the study, the workspace was normalized to each participant's individual anthropometrics. CSA Standards for reach zones are approximated.

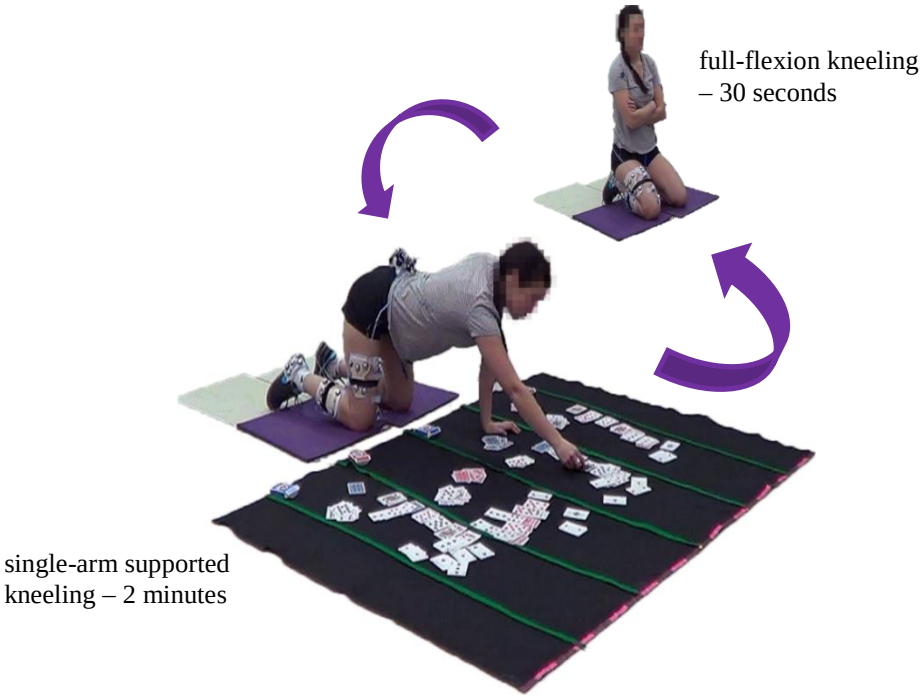


Figure 4.6 An illustration of the card sorting task activity cycle.

In order to reduce the effects of knee pain, thin foam mats (1 mm thickness when compressed) were placed on the force plates under the participants' knees. Pain was assessed using a 100-mm visual analog scale before the kneeling trial began and at five minute intervals during the kneeling exposure task. The pain measure was always recorded while the participant rested in full-flexion kneeling (Figure 4.6 – full-flexion kneeling posture, Appendix F: Pain Diagram). Immediately following the end of the kneeling exposure, knee joint proprioception (three trials), gait, and squats were measured again. Loaded and unloaded gait trials were randomized, with at least three trials collected in each condition. At least three trials were also recorded for the squat transitions. The participant then rested quietly while seated until thirty minutes had passed since the end of the kneeling exposure. Following the rest period, proprioception, gait, and squat measures were repeated to determine the transiency of the effects, again, with at least three trials collected for each condition (Figure 4.7).

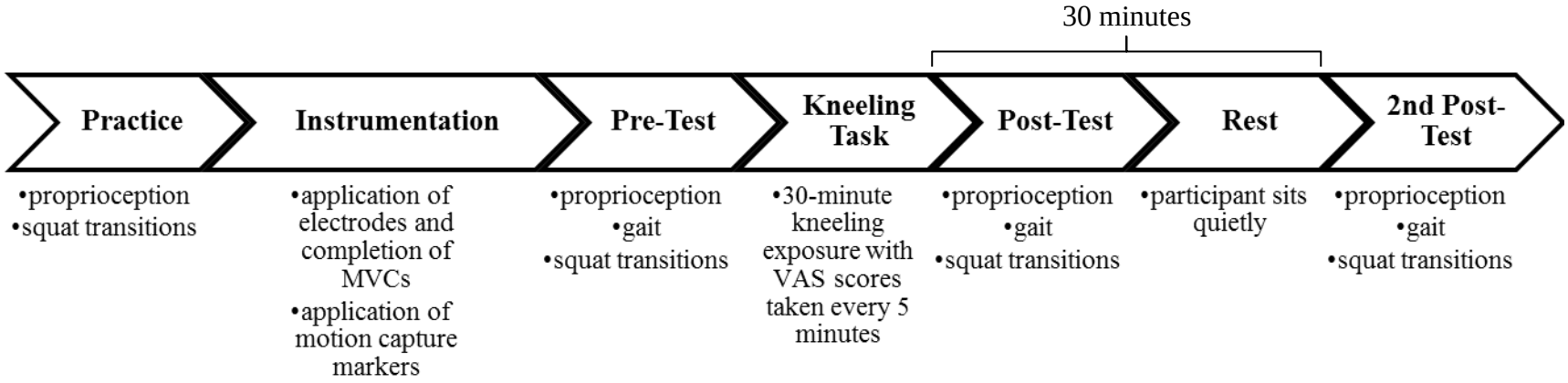


Figure 4.7 The experimental design.

4.3 Kinematics

Lower limb kinematic data were measured using a 6-bank, 18 camera 3D Optotrak system (Optotrak Certus & 3020, NDI, Waterloo, ON, CA). The collection volume was calibrated using a rigid cube (16 infrared diodes) during a 60-second calibration trial. The global coordinate system was defined following the ISB standard (Wu & Cavanagh, 1995) using a digitizing probe to facilitate data processing in Visual 3D (V 4.96.13, C-Motion, Germantown, MD, USA). Figure 4.8 illustrates the laboratory set-up.

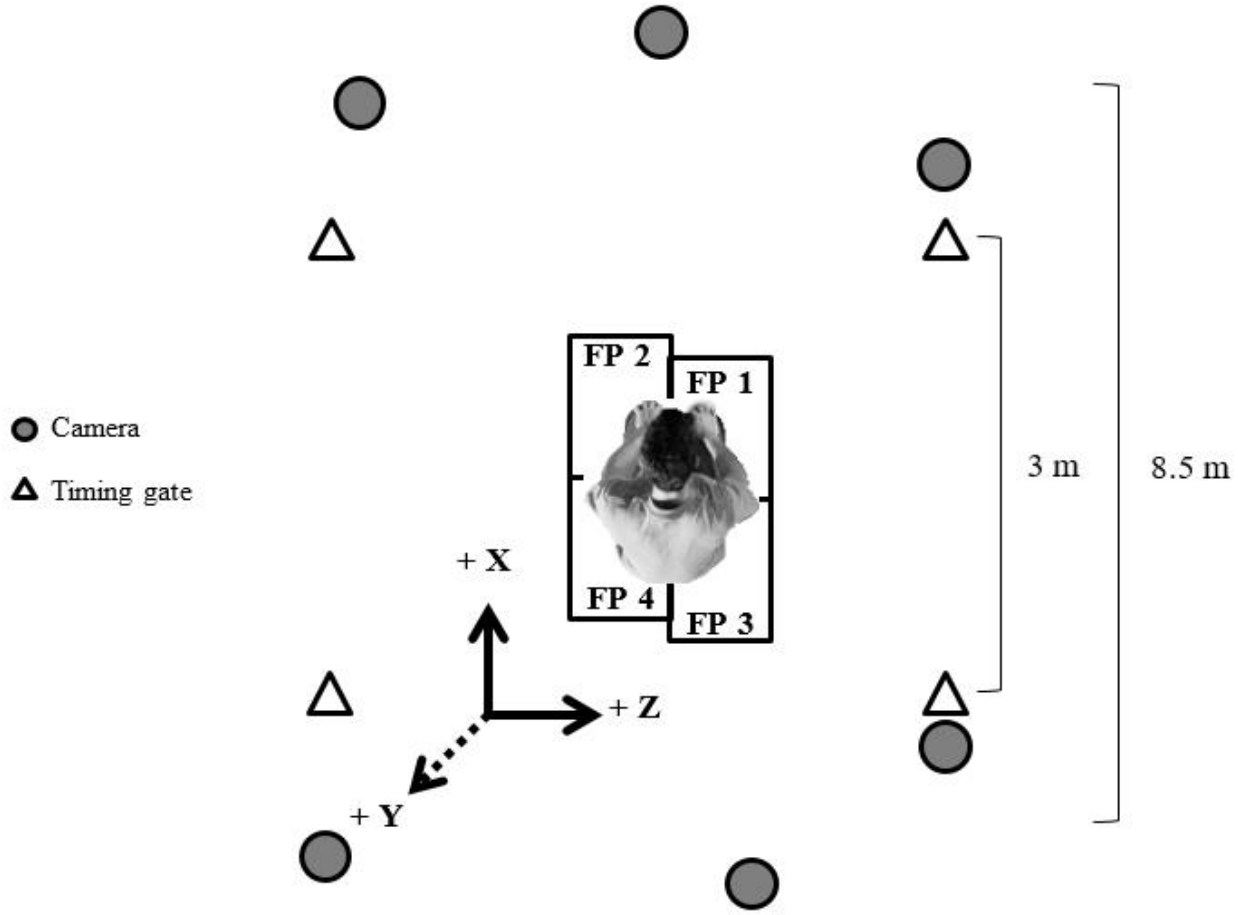
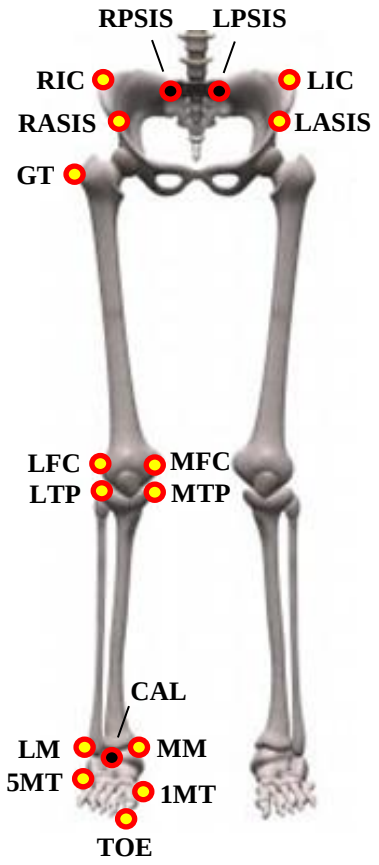


Figure 4.8 The laboratory set-up.
NOTE: the location of the global coordinate system indicates the origin. Force plates are indicated by 'FP' and the respective force plate number.

Rigid bodies, each with five, non-collinear infrared emitting diodes, were securely attached to the lateral aspect of the foot, shank, and thigh of the participant's dominant leg. An additional cluster was securely attached to the skin over the sacrum. Bony landmarks were digitized using a digitizing probe to identify relevant anatomical locations and allow for the creation of 3D segment coordinate systems (Figure 4.9).



Segment	*Marker*	*Definition*
Pelvis	RASIS*	right anterior superior iliac spine
	LASIS*	left anterior superior iliac spine
	RIC	right iliac crest
	LIC	left iliac crest
	RPSIS*	right posterior superior iliac spine
	LPSIS*	left posterior superior iliac spine
Thigh	GT*	greater trochanter
	LFC	lateral femoral condyle
	MFC	medial femoral condyle
Shank	LTP	lateral tibial plateau
	MTP	medial tibial plateau
	LM*	lateral malleolus
	MM*	medial malleolus
Foot	1MT*	1st metatarsal head
	5MT*	5th metatarsal head
	CAL	calcaneous
	LM*	lateral malleolus
	MM*	medial malleolus
	TOE	tip of the great toe

Figure 4.9 Anatomical landmarks for 3D segment coordinate system definitions.
The right leg is the dominant leg in this example. Markers used to define the segment are denoted with an asterisk (*).

The digitization procedure took place with the participant standing in the anatomical position. Following digitization, the static reference trial for model generation was recorded, also in the anatomical position. In order to facilitate the determination of hip and knee joint centers, functional movement trials were collected. The hip joint center trial incorporated hip

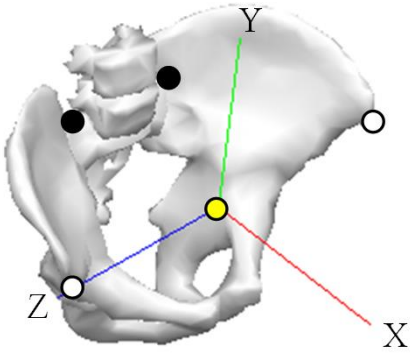
flexion, extension, abduction, adduction, and circumduction motions for 10 cycles, with participants instructed to limit range of motion to less than maximum (Begon, Monnet, & Lacouture, 2007). For the knee joint center trial, the participant was instructed to flex and extend the knee for 30 seconds, from full extension to approximately 90° of flexion. Functional joints were calculated in Visual 3D using joint calculation algorithms (Schwartz & Rozumalski, 2005). The knee joint center and axis of rotation calculated by the algorithm were used to create new medial and lateral knee markers. This was done by projecting the medial and lateral femoral condyle markers (LFC & LMC, Figure 4.9) onto the axis of knee rotation.

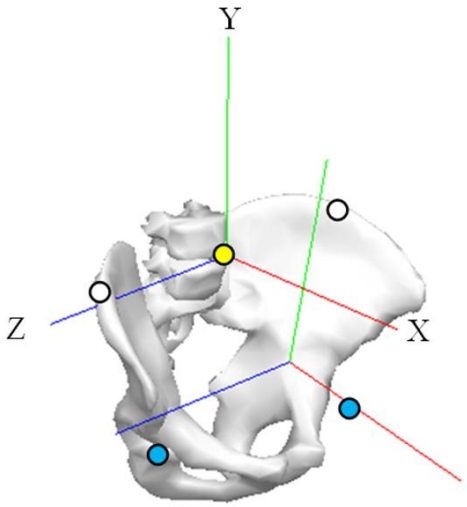
Coordinate systems for each of the segments complied with ISB recommendations (Wu & Cavanagh, 1995), with the x-axis indicating the anterior/posterior direction, the y-axis indicating the superior/inferior direction, and the z-axis being perpendicular to both following the right-hand rule (Table 4.2). A 'Coda' pelvis was selected to define the pelvis coordinate system because the ASIS and PSIS bony landmarks are usually readily identifiable on young, healthy individuals. This is compared to the sacrum, pubis, or tops of the iliac crest, which are required landmarks when using other pelvis coordinate system definitions, and may be more difficult to landmark accurately if excess soft tissue is present. Additional 'virtual' pelvis and foot segment coordinate systems were built to facilitate joint angle calculations that are biologically relevant. As such, these secondary coordinate systems were considered for use in calculating kinematics only, and were not used in calculating kinetics. For the hip, a kinematic-only segment was required because the Coda pelvis induces an anterior tilt of the local coordinate system (because the transverse plane is defined by the ASIS and PSIS markers, see Table 4.2). For the foot, a kinematic-only segment was required because it was preferred to normalize the ankle angle in degrees of plantar- or dorsi-flexion with respect to the reference

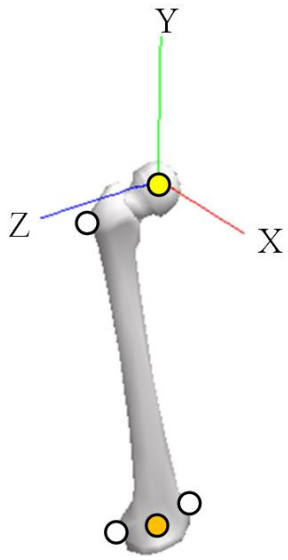
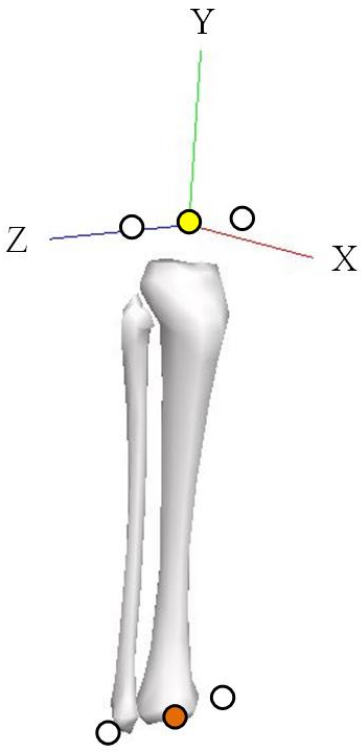
posture. For example, without the kinematic-only segment, the ankle angle in the standing reference posture would be expressed as approximately 90°.

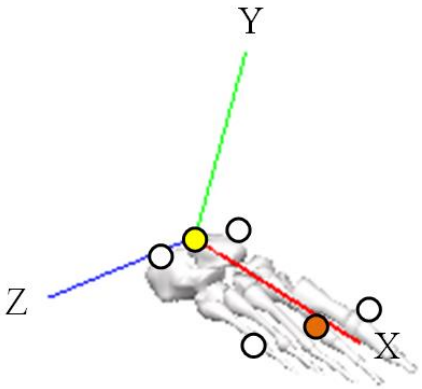
Table 4.2

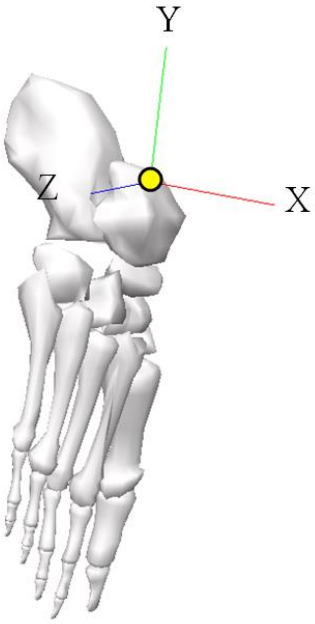
Body Segment Coordinate Systems for the Lower Limb

Segment	**Coordinate System Setup**
Coda Pelvis  	Origin: The origin is defined as the midpoint between ASIS markers. XZ plane: The XZ-plane is the plane defined by the left and right ASIS and PSIS markers. X-axis: The X-axis is defined as the vector from the distal segment endpoint to the proximal segment endpoint (midpoint of the PSIS markers to the midpoint of the ASIS markers), at the origin. Note that for the purposes of calculating hip angles, defining the X-axis based on the ASIS and PSIS markers induces an anterior pelvic tilt. Therefore, a secondary "virtual pelvis" was used to calculate hip joint angles. Y-axis: The Y-axis is the vector perpendicular to the XZ-plane at the origin. 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-by-Y, at the origin.

Segment	Coordinate System Setup
'Virtual' Pelvis 	Origin: The origin is defined as the midpoint between iliac crest markers. Iliac crest markers were defined as a vertical projection from the hip joint centers estimated by the Coda pelvis (highlighted in blue). YZ plane: The YZ-plane is the plane defined by the left and right hip joint center and iliac crest markers. Y-axis: The Y-axis is defined as the vector from the distal segment endpoint to the proximal segment endpoint (midpoint of the hip joint centers to the midpoint of the iliac crest markers), at the origin. 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-by-Y, at the origin.
Thigh	Origin: The origin is defined as the hip joint center, as calculated using the functional trials. YZ plane: The YZ-plane is the plane defined by the greater trochanter, the hip joint center, and the lateral and medial knee makers (NOTE: the lateral and medial knee markers are created from the projections of the lateral and medial femoral condyle markers onto the knee axis of rotation, as estimated from the functional knee joint trial).

Segment	Coordinate System Setup
	Y-axis: The Y-axis is defined as the vector from the distal segment endpoint to the proximal segment endpoint (midpoint of the knee markers to the functional hip joint center), at 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 from the hip joint center.
Shank 	Origin: The origin is defined as the midpoint of the lateral and medial knee markers. YZ-plane: The YZ-plane is the plane defined by the lateral and medial knee markers and the lateral and medial malleoli. Y-axis: The Y-axis is defined as the vector from the distal segment endpoint to the proximal segment endpoint (midpoint of the malleoli markers to the midpoint of the knee markers), at the origin. 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-axis by the Y-axis, from the knee joint center.

Segment	Coordinate System Setup
Foot  	By default, Visual 3D determines the frontal plane as the plane defined by the segment endpoints. In the case of the virtual pelvis, femur, and tibia, this is correct, and the frontal (YZ) plane is defined by the markers. However, in the foot, the plane defined by the markers is actually the transverse plane. Therefore, the default coordinate system created by Visual 3D is manually rotated in the software (as shown corrected in the figure), so that the transverse plane is the XZ-plane and the frontal plane is the YZ-plane. Origin: The origin is defined as the midpoint between lateral and medial malleoli markers. YZ-plane (original): The YZ-plane is the plane defined by the lateral and medial malleoli and the 1st and 5th metatarsals. Y-axis (original): 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, at the origin. X-axis (original): 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-by-Y, from the origin. The local coordinate system is then rotated manually in Visual 3D such that the YZ-plane is the frontal plane.

Segment	Coordinate System Setup
'Virtual' Foot  	The virtual foot is defined based on the same markers as the shank segment, but tracks to the foot cluster markers. YZ-plane: The YZ-plane is the plane defined by the lateral and medial knee markers and the lateral and medial malleoli. Y-axis: The Y-axis is defined as the vector from the distal segment endpoint to the proximal segment endpoint (midpoint of the malleoli markers to the midpoint of the knee markers), at the origin. 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-axis by the Y-axis, from the knee joint center.

Note: All coordinate system lines indicate the direction of the positive (+) axis.
Circles represent the relevant bony landmarks, segment origins, and midpoints (white – anterior, black – posterior, origin – yellow, midpoint – orange, joint center – blue)

Kinematic data were sampled at 100 Hz and filtered using a 2nd order dual-pass low-pass Butterworth filter with a cut-off frequency of 6 Hz (Winter, Patla, & Frank, 1990). Joint angles were calculated in Visual 3D with a ZXY (flexion/extension – abduction/adduction – axial rotation) Cardan sequence. The ankle angle was calculated using the virtual foot, with the shank as the reference segment. The knee angle was calculated using the shank, with the thigh as the reference segment. The hip angle was calculated using the thigh, with the virtual pelvis as the reference segment. Gait event data were also calculated in Visual 3D in order to determine event timing. A footswitch attached under the heel of the shoe of the dominant limb was used to verify the initial contact event generated by Visual 3D. Kinematic data were padded by ensuring at

least 1 second of collected data before and after proprioception tests, the analyzed strides of gait, and squat transitions (Howarth & Callaghan, 2009). All kinematic post-processing was completed in Matlab (R2015a, MathWorks, Natick, MA, USA).

4.4 Kinetics

Moment and force data from the force plate were sampled at 2100 Hz (OR6-7, AMTI, Watertown, MA, USA). The force plate amps have a built in 2nd order low-pass critically damped filter of 1050 Hz. In order to facilitate identification of heel-strike transients, and to calculate the external rate of loading and knee adduction moment, force data was processed in two ways: unfiltered (no additional filtering), and dual-pass low-pass filtered using a 2nd order Butterworth filter at a cut-off of 100 Hz (Kristianslund, Krosshaug, & Van den Bogert, 2012). Unfiltered force data was used to determine the presence or absence of heel-strike transients (Whittle, 1997), as well as calculate joint moments. A heel-strike transient was identified, if, during the rising phase of the vertical ground reaction force, the force peaked then decreased by more than 1.2% of the peak vertical force magnitude of the rising phase (similar to the methods described by Hunt et al., 2010) (Figure 4.10).

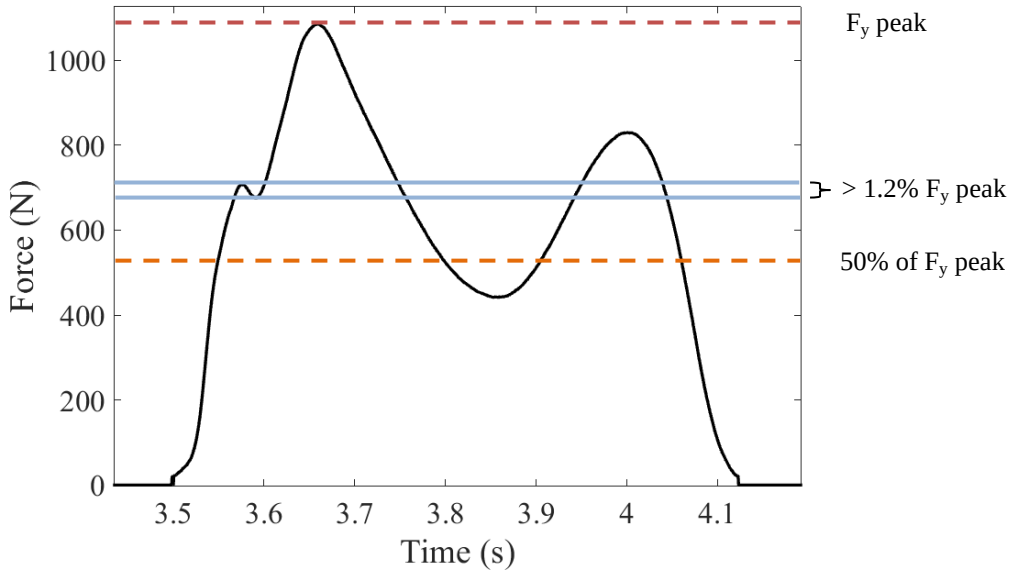


Figure 4.10 Illustration of the method used to identify the occurrence of a heel-strike transient.
NOTE: 'Force' is the vertical ground reaction force.

The external knee adduction moment was calculated in Visual 3D and was resolved into the tibial coordinate system (Mündermann, Dyrby, Hurwitz, Sharma, & Andriacchi, 2004). Knee moments were normalized as a percentage of participant body weight multiplied by height in order to remove the potentially confounding effects of sex (Moisio, Sumner, Shott, & Hurwitz, 2003). The resulting measure of the external knee adduction moment is unit-less. The peak knee adduction moment during weight acceptance of the stance phase of gait (i.e. the peak moment during first half of the stance phase, or the first peak of the double peaked curve) was reported for statistical analysis. The maximum vertical loading rate was determined by the peak value of the first derivative of the force data filtered at 100 Hz (Mikesky et al., 2000). For this measure, the force data was normalized to body weight. Force data was also used for event timing using the 'Automatic Gait Events' function in Visual 3D. The minimum force threshold was set at the program default of 20 N (Zeni et al., 2008).

4.5 Electromyography

Surface electromyography (EMG) was used to record muscle activation of the vastus medialis of the dominant leg. The skin over the muscle belly was prepared by shaving the skin, followed by light abrasion with an exfoliating gel (NuPrep Skin Prep Gel, Weaver and Company, Aurora, CO, USA) and rubbing alcohol. Surface Ag-AgCl electrodes (Ambu ® Blue Sensor N, Denmark) were applied with a two-centimeter inter-electrode distance over the muscle belly and in line with the direction of the muscle fibres, approximately two finger widths medial and 3 – 4 finger widths superior to the superolateral border of the patella, following the SENIAM guidelines (Hermens et al., 1999) (Figure 4.11a). Signals were measured using a wireless EMG system (Wave Plus EMG, Cometa, Cisliano, Italy). This system has a built-in non-modifiable 1000x signal amplification and a bandpass filter of 10 – 500 Hz. A reference electrode is not required using this system. Appropriate electrode placement was confirmed through manual muscle test contractions to ensure a clean signal.

Participants completed a 5-minute warm-up that consisted of walking at a comfortable pace on a treadmill. Next, maximum voluntary contractions (MVCs) were recorded to measure maximal neural drive. Participants were then seated in a knee extension machine with the knee flexed at an angle of 45° from full knee extension (Lin, Hsu, Chang, Chien, & Chang, 2008) (Figure 4.11b). Participants were instructed to isometrically contract their knee extensors against resistance, ramping up until they achieved their maximum effort. The participant was given at least one practice trial to familiarize themselves with the equipment and the testing procedure. During the testing procedure, the participant was given verbal encouragement. Two maximal voluntary contractions were recorded, with a minimum of 1-minute rest between trials, or until the participant indicated they were ready to complete a second trial. The participant was

asked if they felt if they achieved a maximum after each test. If the participant felt that they had not achieved their max, additional trials were taken until they were confident that they had successfully done so. A resting baseline trial with the participant lying supine was also recorded while the participant rested quietly with muscles inactive.

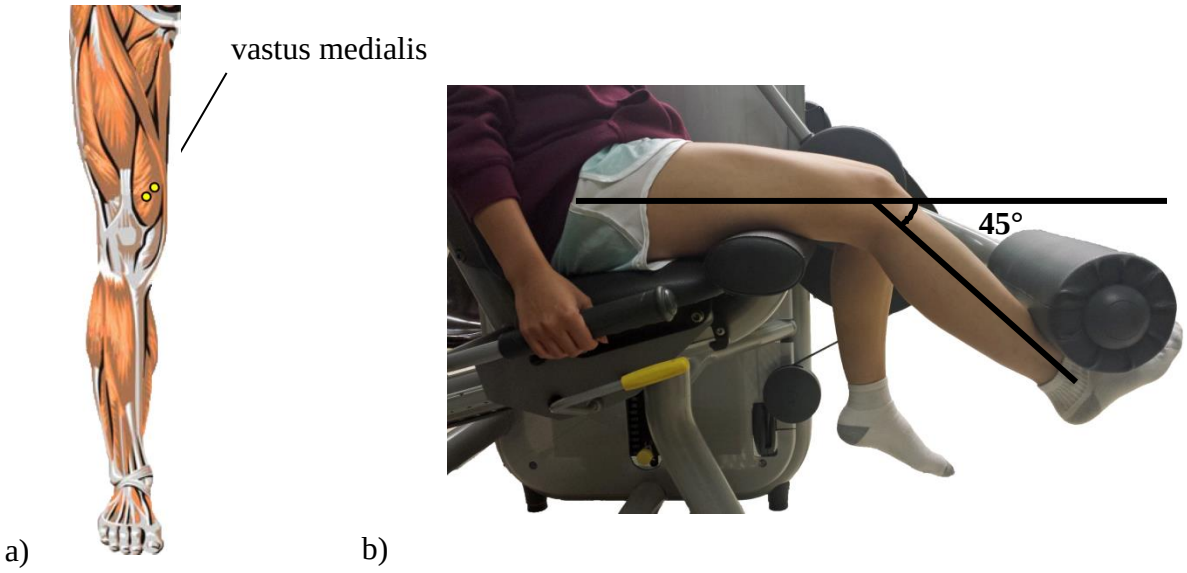


Figure 4.11 Vastus medialis testing set-up.
a) Electrode placement b) MVC testing posture.

4.5.1 Calculating quadriceps activation onset and magnitude.

A standard method to determine muscle activation onset does not exist in the literature; however, it is well understood that removing too much of the high frequency components of the EMG signal can delay the identification of onset time (Robertson, Caldwell, Hamill, Kamen, & Whittlesey, 2004). To determine activation onset, the EMG signal was processed using the following procedure: 1) DC bias removal, 2) 20 Hz dual-pass, high-pass Butterworth filter (Cholewicki & VanVliet Iv, 2002; De Luca, 1997; Potvin & Brown, 2004) to remove low-frequency noise contamination, 3) full-wave rectification 4) 50 Hz dual-pass, low-pass

Butterworth filter. Vastus medialis activation onset was defined as the first frame where the 50 ms moving average window of the processed signal exceeded baseline levels by at least three standard deviations, for 25 ms, within the 250 ms window before initial contact (Hodges & Bui, 1996). The baseline mean and standard deviation values used to determine the thresholds were calculated based on the quiet resting trial. Activation onset was reported as the time measured in seconds relative to the occurrence of foot initial contact, taking into account the built-in delay of 14 ms in the Cometa wireless EMG system. Activation onset was also reported as a percentage of the gait cycle to facilitate comparisons with similar data in the literature. The footswitch was used to identify the timing of the second heel-strike for normalization purposes.

To analyze vastus medialis activation magnitude, the EMG signal was linear enveloped using the following procedure: 1) DC bias removal 2) dual-pass, high-pass Butterworth filter at 20 Hz, 3) full-wave rectification, 3) dual-pass, low pass Butterworth filter with a cut-off of 6 Hz (Schmitz, Silder, Heiderscheit, Mahoney, & Thelen, 2009). The high-pass filter was included to eliminate low-frequency noise contamination that was apparent in some participants' data. Signals were then normalized to the peak of the linear enveloped EMG from the MVC trials. Vastus medialis activation magnitude was reported as the trapezoidal integrated value of the signal between 50 ms prior to, and 50 ms following initial contact, in order to capture EMG activity around the impact phase of gait (Nigg, Cole, & Bruggemann, 1995; Riskowski et al., 2005).

4.5.2 Evaluating the presence of fatigue.

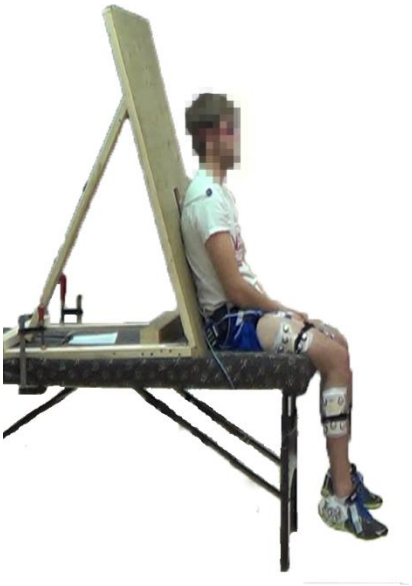
Efforts were taken to reduce the potentially confounding effects of fatigue, such as reminding participants that they may take a break should they choose to do so (such as during the loaded gait trials). However, it is understood that fatigue can affect both proprioception (Björklund et al., 2000; Miura et al., 2004) and neuromuscular control (Moore et al., 2002; Yeung, Au, & Chow, 1999). Therefore, it was necessary to quantify fatigue to account for this potentially confounding factor. Local fatigue was quantified for vastus medialis by analyzing the raw EMG waveform (DC bias removed) during the ascending phase of the slow squat transition. For each squat in succession, a 250 ms window centered on when the knee flexion angle was 80° was isolated, similar to previously validated methods for evaluating fatigue during dynamic contractions (Potvin, & Bent, 1997). In this way, only concentric contractions were evaluated. A fast Fourier transform was then performed on each 250 ms window to determine the mean power frequency (KinAnalysis, University of Waterloo, ON, CA). The mean power frequency values at each measurement time (pre/post/30post) were averaged (Cho & Kim, 2012), and expressed as a percentage of the baseline mean power frequency. Fatigue was considered to have occurred when the mean power frequency decreased by greater than eight percent of the baseline (Mastalerz et al., 2012).

4.6 Proprioception

Knee joint proprioception was measured as knee joint position sense using methods that have been previously validated (Beynnon et al., 2000). A non-weight bearing test was selected to better replicate proprioceptive input available prior to initial contact of gait, when the leading limb is non-weight bearing (Riskowski et al., 2005). The participant was seated in a chair such that the back was supported at a 70° angle, the thigh was approximately parallel to the floor, and

both legs were free to dangle (knee angle at approximately 80°) (Figure 4.12a). The backrest was constructed with a cutout to allow the fin-like marker cluster used to track the pelvis to pass through undisturbed and permit the participant to have their upper body fully supported (Figure 4.12b). A blindfold was used to occlude vision and the participant was instructed to relax their muscles as much as possible. The instructions to the participant were standardized (Appendix G: Proprioception Participant Instructions).

a)



b)

Figure 4.12 The knee joint position task set-up.
a) The resting posture b) Demonstrating the notch in the backrest to allow the pelvis cluster to pass through.

When the participant indicated that they were ready and focused, the investigator lightly held the participant's shoe on the dominant leg, and moved the participant's leg into extension at a rate of approximately 10°/s to a target flexion angle of 20°, measured initially using a goniometer. To allow for consistent repeated trials, a physical external cue was placed near the height of the participant's heel when the knee was at 20° (based on measurement using the goniometer) so that the investigator could feel with their hand on the participant's foot when the

target knee angle was reached. Only the investigator came in contact with the external cue, not the participant. Although proprioception is thought to be enhanced near end range of motion (Hogervorst & Brand, 1998), which may impair the ability of the test to detect changes, this angle was selected because it approaches the knee angle at initial contact of gait (Riskowski et al., 2005; Winter, 1991), and therefore is more functionally relevant than proprioception measured at the mid-range of joint motion (Riskowski et al., 2005). Once the target angle was reached, the investigator removed support and the participant held the posture for five seconds while focusing on the knee position. The participant's leg was then returned to the resting posture by the investigator. After five additional seconds, the participant was asked to actively replicate the knee angle with the ipsilateral leg and indicate to the investigator when they felt that the target angle had been reached. The participant held the re-test angle for five seconds.

All knee angles were measured and recorded using the Optotrak system with the marker placement strategy described previously (4.3). To facilitate the calculation of the average angle, the research assistant fired an external trigger once when the investigator placed the knee at the target angle, and again when the participant indicated that they reached what they felt was the target angle by notifying the research assistant with a verbal cue. Three trials were taken each time proprioception was measured. To determine proprioceptive acuity, the absolute angular error score was used. The absolute angular error score is the magnitude of the difference between the average target knee joint angle and the average re-test angle for each trial. An average absolute angular error was also calculated at each measurement point.

4.7 Frontal Plane Knee Motion during Squat Transitions

Kinematics of squat transitions to-and-from the floor were also measured. Squat performance was characterized by determining the peak and mean absolute deviation of the functional knee joint center with respect to the body-fixed plane created by the ankle joint (defined as the midpoint of the medial and lateral malleoli markers), the functional hip joint center, and the distal foot (defined as the midpoint of the fifth and first metatarsal heads) (Frost, Beach, McGill, & Callaghan, 2015) (Figure 4.13). Deviation was measured as the perpendicular distance of the knee joint center to the plane and reported as the absolute value, in meters. The absolute value was used because the goal of this measure was to quantify negative adaptations in movement patterns, and therefore were most interested in whether the deviation increased, and less so about the direction of the deviation.

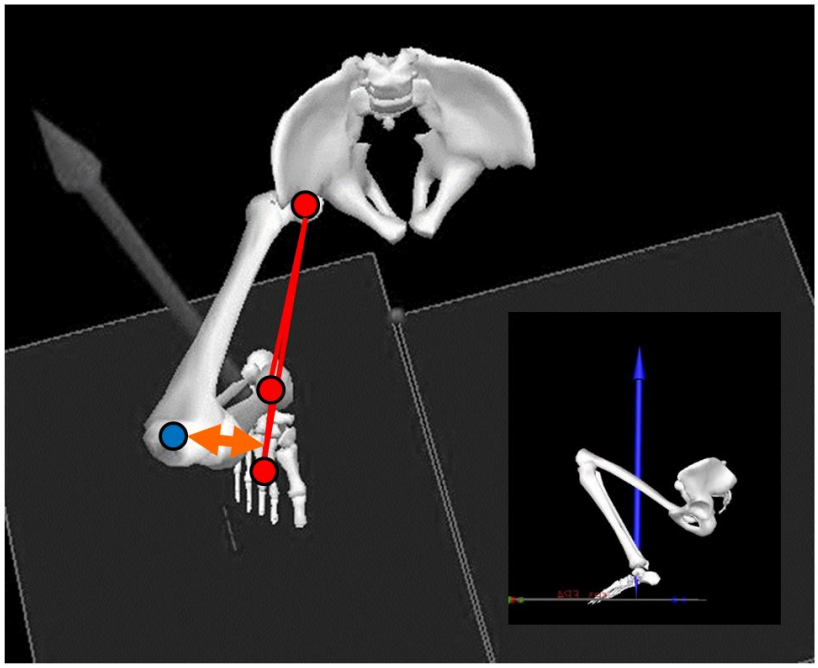


Figure 4.13 Illustration of the measure used to quantify medial/lateral deviation of the knee joint center in the frontal plane.
Red circles indicate the points used to create the body-fixed plane (red lines). The orange double-headed arrow represents the perpendicular distance of the knee joint center (blue circle) from the plane. Inset figure shows the sagittal view of the squat to illustrate squat depth.

Although frontal plane knee motion has been associated with an increased risk for traumatic knee injury during dynamic movements (Hewett et al., 2015), and not knee osteoarthritis risk, quantifying frontal plane knee motion may be used to determine whether the kneeling exposure caused changes in neuromuscular control that may not be evident during gait, if the effect sizes of changes in gait are small. In addition, as previously indicated, workers in professions such as floor laying perform frequent transitions to-and-from the ground as part of their occupation. While the quality of movement during a squat has never been directly linked to knee osteoarthritis risk, certain kinematic variables, including increased frontal plane motion, have been associated with an increased risk for traumatic knee injury, and therefore could be an indirect risk factor for knee osteoarthritis.

4.8 Statistical Analyses

Statistical analysis software (SAS Version 9.4, Cary, NC, USA) was used to test the proposed hypotheses (Table 2.1). All ANOVA testing used a mixed general linear model design. 'Trial' was included as an independent variable in order to evaluate the transiency of any of the observed effects, should the effects of kneeling washout before all trials at each time point were collected. Levene's test was used to evaluate the homogeneity of group mean variance, and Mauchly's test was used to test the hypothesis that the variances of the differences between conditions were equal (evaluates the assumption of sphericity). When Mauchly's test indicated a violation of the assumption of sphericity, the Greenhouse-Geisser corrected p-value was used to evaluate significance. Planned contrast analysis was used to determine the significance between means when significant interaction effects were found. Effect size was reported using Pearson's correlation coefficient *r*. (Field & Miles, 2010)

For measures of knee proprioception, significant effects were determined using a 3-way mixed model ANOVA (Table 4.3).

Table 4.3

Statistical Analysis Procedure for Absolute Angular Error

Factor Type	**Independent Variables**		**Dependent Variables**	**Statistical Model**
	Factor	***Level***		
Between	Sex	Male Female	Absolute Angular Error (AAE)	Mixed General Linear Model 3-way ANOVA (2x3x3)
Within	Time	Pre Post 30Post		
Within	Trial	Trial 1 Trial 2 Trial 3		

For gait measures of peak vertical loading rate, peak knee adduction moment, vastus medialis activation onset, and vastus medialis activation magnitude, significant effects were determined using a 4-way mixed model ANOVA (Table 4.4). Note, that for statistical analysis of activation magnitude, participant S41 was excluded because the recorded MVCs for this participant were not a true maximal effort based on the evaluation of activation magnitudes during gait, which were found to exceed peak EMG values recorded during the MVC trials.

Table 4.4

Statistical Analysis Procedure for Gait Variables

Factor Type	**Independent Variables**		**Dependent Variables**	**Statistical Model**
	Factor	***Level***		
Between	Sex	Male Female	*Kinetics* Peak Loading Rate Peak Knee Adduction Moment	Mixed General Linear Model 4-way ANOVA (2x2x3x3)
Within	Load	Load No Load		
Within	Time	Pre Post 30Post	*Electromyography* Vastus medialis activation onset Vastus medialis activation magnitude	
Within	Trial	Trial 1 Trial 2 Trial 3		

For squat measures of peak and mean deviation from the plane, significant effects were determined using a 5-way mixed model ANOVA (Table 4.5).

Table 4.5

Statistical Analysis Procedure for Squat Variables

Factor Type	**Independent Variables**		**Dependent Variables**	**Statistical Model**
	Factor	***Level***		
Between	Sex	Male Female	*Neuromuscular Control* Peak Deviation from the Plane Mean Deviation from the Plane	Mixed General Linear Model 5-way ANOVA (2x2x2x3x3)
Within	Speed	Fast Slow		
Within	Direction	Descent Ascent		
Within	Time	Pre Post 30Post		
Within	Trial	Trial 1 Trial 2 Trial 3		

5.0 Results

5.1 Quantitative Protocol and Kneeling Exposure Summary

All participants were able to complete the study protocol to satisfaction. Three participants were removed from analysis because of experimental technical errors and one for insufficient trials noted during post-processing. One participant indicated 'no' when asked in the pre-participation survey, "Would you consider yourself to be in a good mood today?" Mood is thought to be associated with the presence of heel-strike transients (Whittle, 1999); however, this participant did not present with heel-strike transients and therefore this should not be considered a confounding factor. During the knee joint position sense task, all participants were confirmed to be relaxed, with VM activation levels ≤ 10 %MVC (Rozzi, Lephart, & Fu, 1999).

During the kneeling exposure, most participants did not develop pain. Only participants S06, S10, S14, S16, and S46 exceeded an increase from baseline of 8 mm or greater, for at least one measured site, at the end of the exposure (Nelson-Wong, & Callaghan, 2014). All measures taken immediately post-kneeling were attained within the 30-minute window intended for the collection of these measures and rest (mean: 17.4 min (SD 4.86 min), maximum: 30 min, minimum: 10 min). Eight participants required five or more trials to capture a stride of gait that met all necessary requirements (i.e. foot isolation, correct speed) at the 'post' measurement time point in one or both of the load conditions.

Analysis of the mean power frequency of VM during the slow squat ascent indicated that five participants may have experienced onset of muscular fatigue during the course of the protocol (Table 5.1). Of these five participants, only one (S45) showed evidence of fatigue at the

'post' measurement time, suggesting that all significant changes found immediately following the kneeling exposure were not confounded by fatigue.

Table 5.1

Participants who demonstrated a Decrease in the Mean Power Frequency during the Ascent Phase of the Slow Squat

	% Change in MPF	
Participant	**Post**	**30Post**
S06	7.8	- 8.8
S12	- 2.4	- 8.2
S13	- 2.4	- 10.6
S39	- 4.1	- 8.7
S45	- 9.7	- 3.8

Only participants where MPF exceeded 8% of the baseline value are reported.

Efforts were made to ensure that the kneeling exposure was similar between participants by normalizing the workspace dimensions to the participant, cycling through defined quadrants in an effort to equalize exposure between the right and left knees, and encouraging the participant to use as much of the workspace as possible. Amplitude probability distribution functions were used to quantify the postures assumed by the participants to verify that the exposure was truly consistent (Figure 5.1, Figure 5.2, and Figure 5.3). Trends were similar between males and females and across participants, with the most deviation occurring in the plantar/dorsiflexion angle of the ankle, which was likely the result of the fact that participants were allowed to select whether they wanted to kneel with their ankles plantar- or dorsi-flexed (Figure 5.3). All participants with the exception of S34 had at least 93% marker cluster visibility for all segments across the exposure (Appendix H: Kneeling Exposure Marker Visibility for Calculation of APDFs).

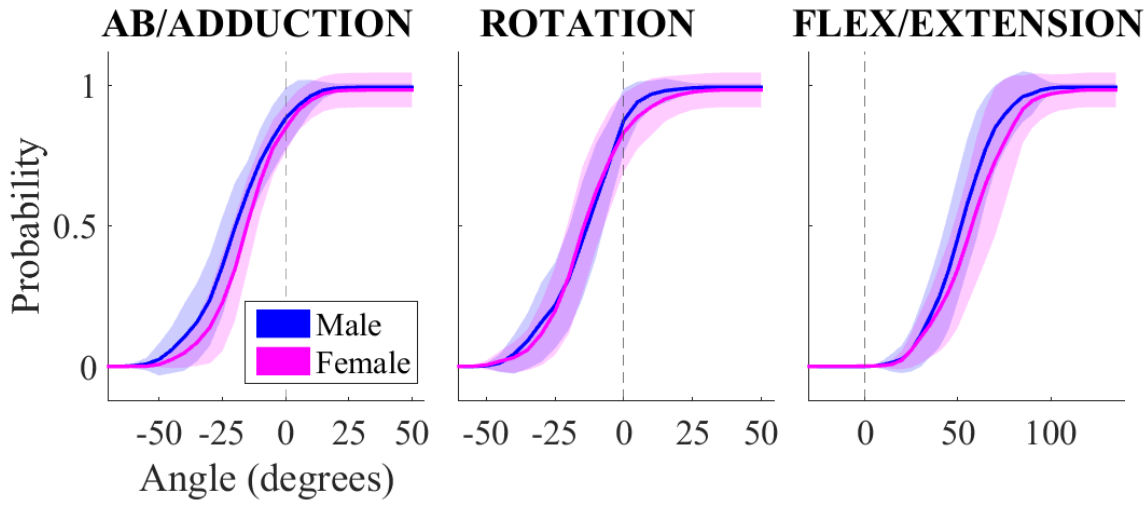


Figure 5.1 Hip angle APDF results for the prolonged kneeling exposure for males and females.
Hip adduction, internal rotation, and flexion are positive (+).

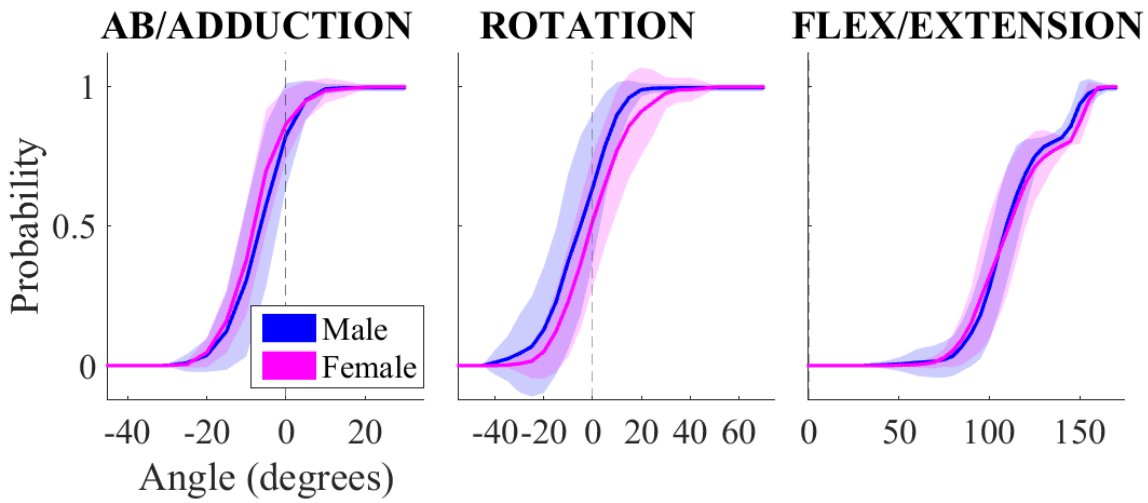


Figure 5.2 Knee angle APDF results for the prolonged kneeling exposure for males and females.
Knee adduction, internal rotation, and flexion are positive (+).

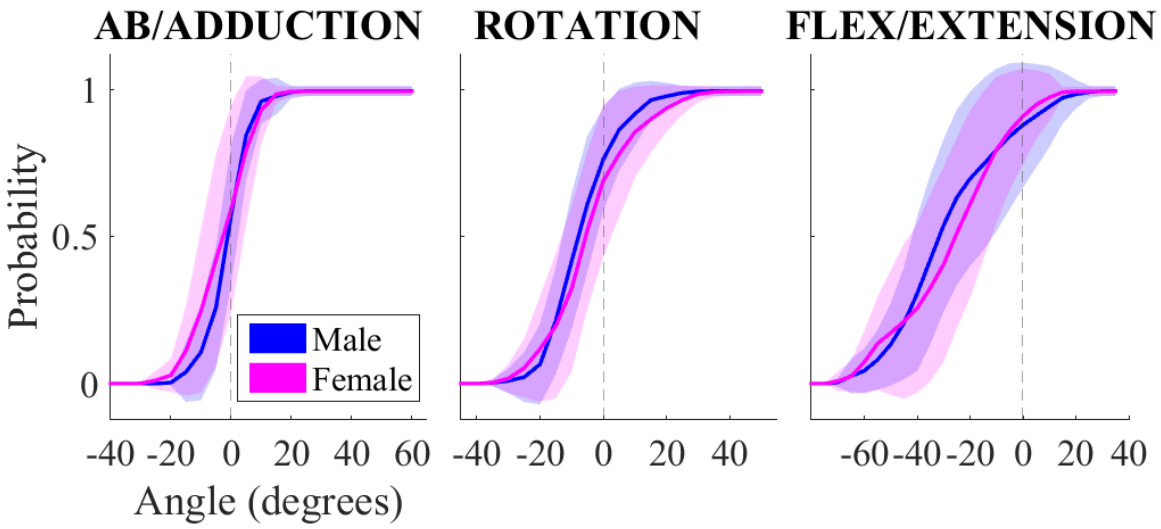


Figure 5.3 Ankle angle APDF results for the prolonged kneeling exposure for males and females. Ankle adduction, internal rotation, and plantarflexion are positive (+).

A summary of the statistics relevant for hypothesis testing is provided (Table 5.2), followed by a more detailed exploration in sections 5.2 – 5.4. The full results of the ANOVA, including comparisons not evaluated within the scope of this document, can be found in Appendix J: Full ANOVA Results.

Table 5.2

Questions, Hypotheses, and Statistical Results - Summary

Question	Hypotheses	Results	Status
	Gait		
1. How is the peak knee adduction moment altered during gait following a simulated occupational kneeling exposure?	a) The peak knee adduction moment will be greater post-kneeling.	a) There was no main effect of the kneeling exposure on the peak knee adduction moment ($F_{2,76}$ = 3.07, p = .0523).	rejected
	b) Changes in the peak knee adduction moment will persist at thirty minutes post-kneeling.	b) There was no main effect of the kneeling exposure on the peak knee adduction moment ($F_{2,76}$ = 3.07, p = .0523).	rejected
	c) A carried load will increase the peak knee adduction moment.	c) A carried load increased the peak knee adduction moment ($F_{1,38}$ = 113.56, p < .0001, r = .87).	accepted
2. Is the vertical loading profile during gait altered following a simulated occupational kneeling exposure?	a) The peak vertical loading rate will be greater following an occupational kneeling exposure.	a) There was no main effect of the kneeling exposure on the peak vertical rate of loading ($F_{2,76}$ = 1.96, p = .160).	rejected
	b) Changes in the peak rate of loading will persist at thirty minutes post-kneeling.	b) There was no main effect of the kneeling exposure on the peak vertical rate of loading ($F_{2,76}$ = 1.96, p = .160).	rejected
	c) A carried load will increase the peak rate of loading.	c) A carried load increased the peak vertical rate of loading ($F_{1,38}$ = 111.40, p < .0001, r = .86).	accepted

Question	Hypotheses	Results	Status
3. Do parameters of quadriceps muscle activation during gait change following a simulated occupational kneeling exposure?	a) The onset of vastus medialis activation will be delayed with respect to initial contact post-kneeling in both loading conditions.	b) The onset of vastus medialis post-kneeling was significantly delayed immediately post-kneeling, compared to onset at baseline ($F_{1,38}$ = 14.86, p = .0004, r = .53).	accepted
	b) The magnitude of vastus medialis activation during the impact phase will be lower post-kneeling in both loading conditions.	a) There was no main effect of the kneeling exposure on vastus medialis activation magnitude ($F_{2,74}$ = 2.61, p = .0803).	rejected
	c) Changes in vastus medialis onset and activation magnitude will persist thirty minutes post-kneeling.	c) The onset of vastus medialis 30 minutes post-kneeling was still significantly delayed compared to onset at baseline ($F_{1,38}$ = 15.98, p = .0003, r = .54).	onset: accepted
		There was no main effect of the kneeling exposure on vastus medialis activation magnitude ($F_{2,74}$ = 2.61, p = .0803).	magnitude: rejected

Question	Hypotheses	Results	Status
		Squat Transitions	
4. Does neuromuscular control, as evaluated by the performance of squat transitions, demonstrate changes following a simulated occupational kneeling exposure?	a) Absolute peak and mean deviations of the knee joint center from the plane created by the hip, ankle, and midfoot will be greater following a simulated occupational kneeling exposure.	a) The peak deviation of the knee joint center post-kneeling was significantly greater compared to peak deviation at baseline ($F_{1,38}$ = 8.59, p = .0057, r = .43). The mean deviation of the knee joint center post-kneeling was significantly greater compared to peak deviation at baseline ($F_{1,38}$ = 6.05, p = .0186, r = .37).	accepted for both
	b) Observed changes will persist at thirty minutes post-kneeling.	b) The peak deviation of the knee joint center post-kneeling was significantly greater compared to peak deviation at baseline ($F_{1,38}$ = 5.32, p = .0267, r = .35). The mean deviation of the knee joint center post-kneeling was significantly greater compared to peak deviation at baseline ($F_{1,38}$ = 8.43, p = .0061, r = .43).	accepted for both
		Knee Proprioception	
5. Is proprioceptive acuity at the knee, as evaluated by a measure of joint position sense, impaired following a simulated occupational kneeling exposure?	a) The absolute angular error of knee joint position sense will increase following a simulated occupational kneeling exposure.	a) There was no main effect of the kneeling exposure on the absolute angular error ($F_{2,76}$ = 0.01, p = .9882).	rejected
	b) Observed changes in knee joint position sense will persist at thirty minutes post-kneeling.	b) There was no main effect of the kneeling exposure on the absolute angular error ($F_{2,76}$ = 0.01, p = .9882).	rejected

5.2 Gait

5.2.1 Kinetics

Peak External Knee Adduction Moment

There was no main effect of the kneeling exposure on the peak knee adduction moment during gait ($F_{2,76}$ = 3.07, p = .0523) (Table 5.3, Figure 5.8). These results do not support hypotheses 1a and 1b that the peak knee adduction moment would increase following a kneeling exposure, and persist for 30 minutes post-kneeling.

Table 5.3

Peak External Knee Adduction Moment for All Participants, Males Only, and Females Only

	Unloaded			Loaded		
Time Point	**All**	**Male**	**Female**	**All**	**Male**	**Female**
Baseline (Pre-Kneeling)	2.46 (0.82)	2.06 (0.72)	2.86 (0.72)	3.00 (1.06)	2.53 (0.89)	3.47 (1.01)
Post-Kneeling	2.57 (0.85)	2.13 (0.70)	3.01 (0.76)	3.05 (1.06)	2.57 (0.92)	3.53 (0.98)
30 Minutes Post-Kneeling	2.52 (0.96)	2.03 (0.78)	3.01 (0.88)	3.06 (1.08)	2.63 (0.94)	3.49 (1.05)

Moments are reported as mean (SD) for both loaded and unloaded conditions, normalized to %BW*H.

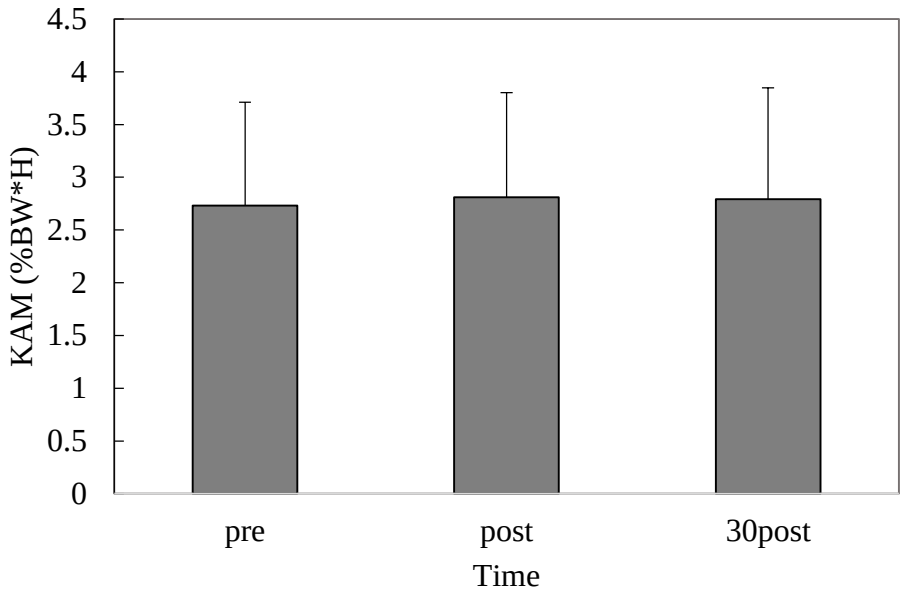


Figure 5.4 The effect of the kneeling exposure on the peak knee adduction moment.
The peak external knee adduction moment during gait is plotted at baseline (pre), post-kneeling, and 30 minutes post-kneeling time points. Error bars indicate +1 SD.

As expected, there was a main effect of the carried load. Across all conditions, the peak knee adduction moment was significantly greater when participants carried 20% of their body weight, compared to when they walked freely (loaded: mean 3.04 (SD 1.07) %BW*H vs. unloaded: mean 2.52 (SD 0.88) %BW*H, $F_{1,38} = 113.56$, $p < .0001$, $r = .87$) (Figure 5.9). This result supports hypothesis 1c that an external load would increase the peak knee adduction moment.

There was also a main effect of sex on the peak knee adduction moment. Females had significantly greater peak knee adduction moments across all conditions compared to males (female: mean 3.23 (SD 0.94) %BW*H vs. male: mean 2.33 (SD 0.86) %BW*H, $F_{1,38} = 12.27$, $p = .0012$, $r = .49$) (Figure 5.10). There was no main effect of trial on the peak knee adduction moment ($F_{2,76} = 0.42$, $p = .6569$). Three-way interaction effects were found between sex, load, and the kneeling exposure ($F_{2,76} = 3.17$, $p = .0474$), and sex, trial, and the kneeling exposure

($F_{2,76}$ = 2.45, p = .0482); however, these complex interactions are limited by the study size and therefore will not be explored within the scope of this document. No other signficant interaction effects were found.

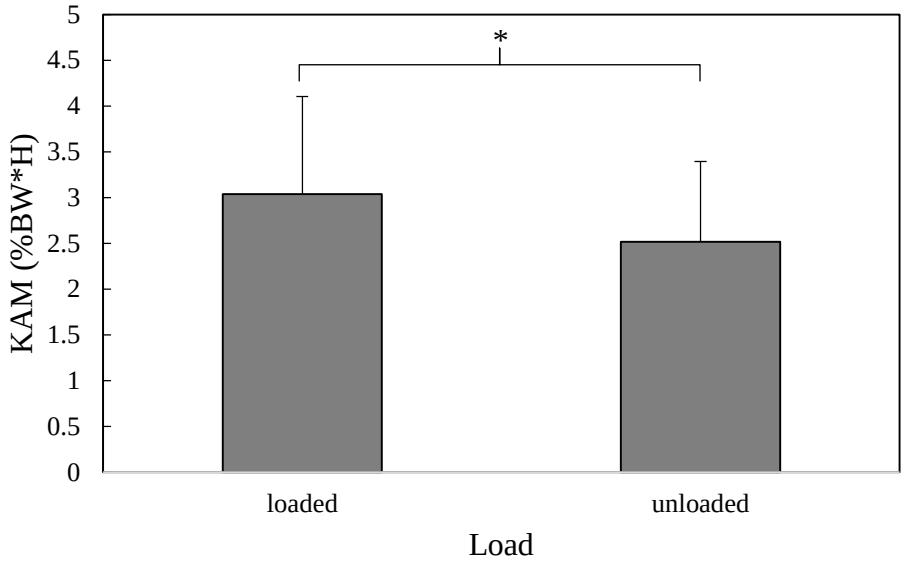


Figure 5.5 The effect of a carried load on the peak knee adduction moment.
The peak external knee adduction moment during gait is plotted in the loaded (20%BW load) and unloaded conditions. The asterisk (*) denotes a significant difference at p < .05. Error bars indicate +1 SD.

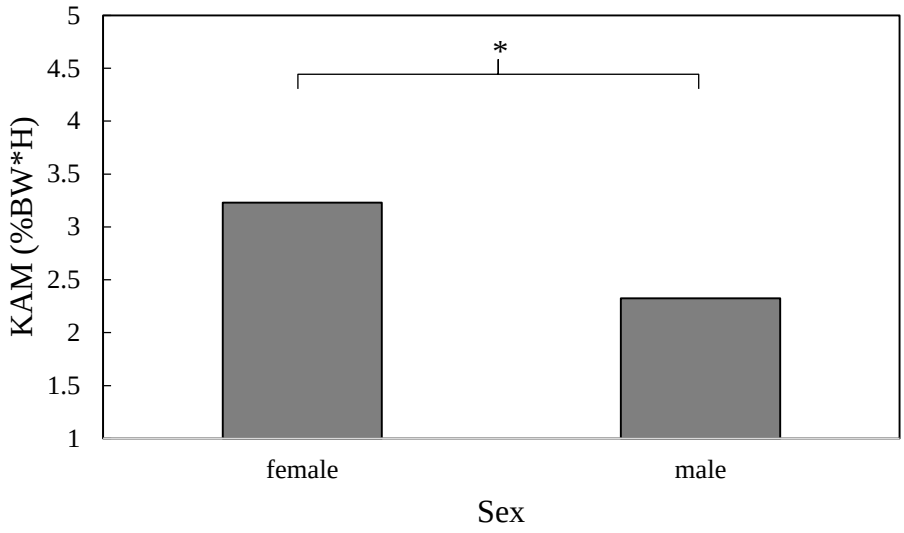


Figure 5.6 The effect of sex on the peak knee adduction moment.
The peak external knee adduction moment during gait is plotted for males and females. The asterisk (*) denotes a significant difference at p < .05. Error bars indicate +1 SD.

Although no main effects of the kneeling exposure on the peak knee adduction moment were found, some participants demonstrated trends that suggest a change occurred. In the loaded condition only, participants S03, S18, and S21 demonstrated an increase, and participants S08, S12 and S19 demonstrated a decrease in the peak knee adduction moment of at least 0.5 %BW*H immediately following the kneeling exposure (Figure 5.11).

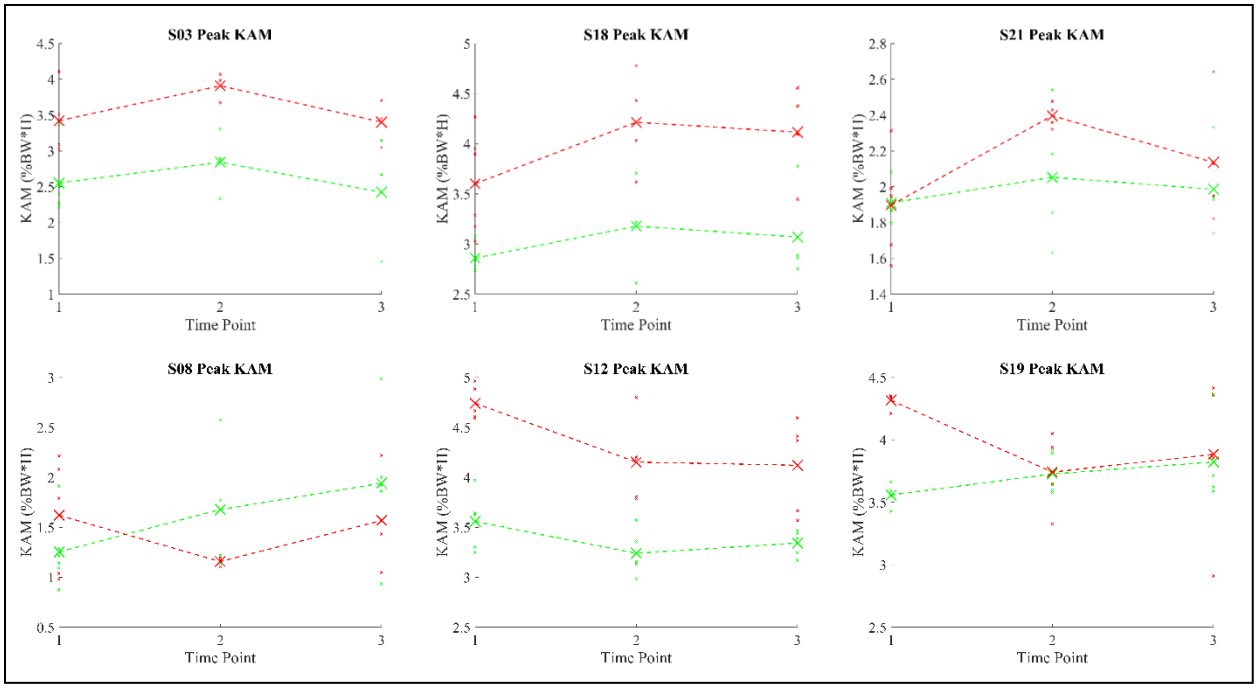


Figure 5.7 Participants who demonstrated a change in the peak KAM following the kneeling exposure.

Loaded gait trials are denoted by the red markers and unloaded gait trials are denoted by the green markers. Large, bold 'X's are the mean peak KAM for that time point, small 'x's' indicate each trial taken. Only participants whose average peak KAM difference between baseline and post-kneeling exceeded 0.5 %BW*H (either increased or decreased) in the loaded condition are shown. Time point 1, 2, and 3 refer to pre, post and 30 minutes post kneeling measurement points, respectively.

Peak Vertical Rate of Loading and Heel-Strike Transients

Contrary to hypotheses 2a and 2b, there was no main effect of the kneeling exposure on the peak vertical rate of loading ($F_{2,76}$ = 1.96, p = .16) (Table 5.4, Figure 5.12). The only factor that was found to have a main effect on the peak rate of loading was the carried load, which

supports hypothesis 2c. The peak rate of loading was significantly greater when the participants carried 20% of their body weight compared to when they walked without a load, across all conditions (loaded: mean 25.19 (SD 6.75) BW/s vs. unloaded: mean 20.43 (SD 5.55) BW/s, $F_{1,38}$ = 111.40, p < .0001, r = .86) (Figure 5.13). There was no main effect of sex ($F_{1,38}$ = 1.72, p = .172, r = .21) or trial ($F_{2,76}$ = 0.88, p = .4173). Although females had a tendency to demonstrate higher peak rates of loading compared to males (female: mean 23.83 (SD 7.13) BW/s, male: mean 21.79 (SD 5.90) BW/s), this difference was not statistically significant ($F_{1,38}$ = 1.72, p = .1970, r = .21). No significant interaction effects were found.

Table 5.4

Peak Vertical Rate of Loading for All Participants, Males Only, and Females Only

	Unloaded			**Loaded**		
Time Point	**All**	**Male**	**Female**	**All**	**Male**	**Female**
Baseline (Pre-Kneeling)	20.30 (5.40)	19.07 (4.66)	21.52 (5.84)	25.38 (6.24)	24.20 (5.02)	26.55 (7.10)
Post-Kneeling	20.19 (5.23)	19.26 (4.86)	21.11 (5.46)	24.66 (6.55)	24.17 (6.15)	25.16 (6.95)
30 Minutes Post-Kneeling	20.80 (6.03)	19.57 (5.86)	22.03 (5.99)	25.54 (7.43)	24.47 (5.59)	26.60 (8.83)

Values are reported as mean (SD) for both loaded and unloaded conditions, normalized to BW/s.

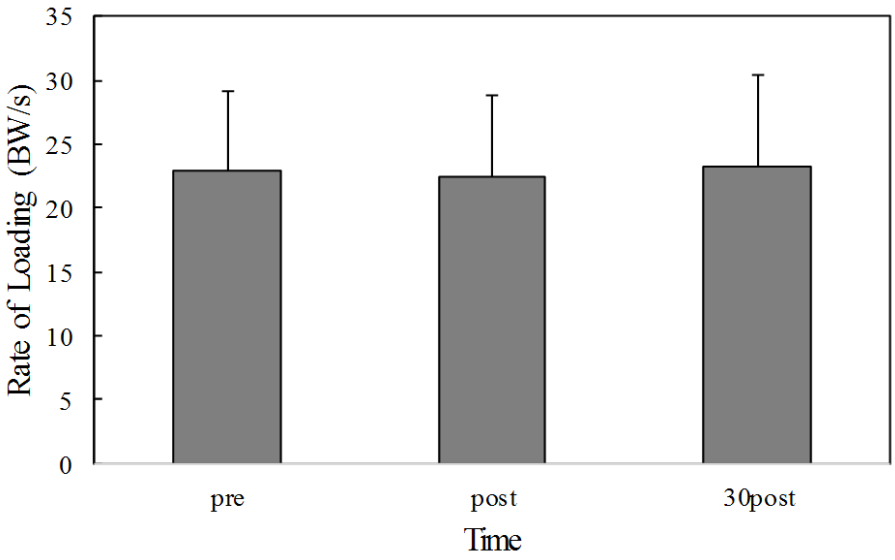


Figure 5.8 The effect of the kneeling exposure on the peak vertical rate of loading.
The peak vertical rate of loading during gait is plotted at baseline (pre), post-kneeling, and 30 minutes post-kneeling time points. Error bars indicate +1 SD.

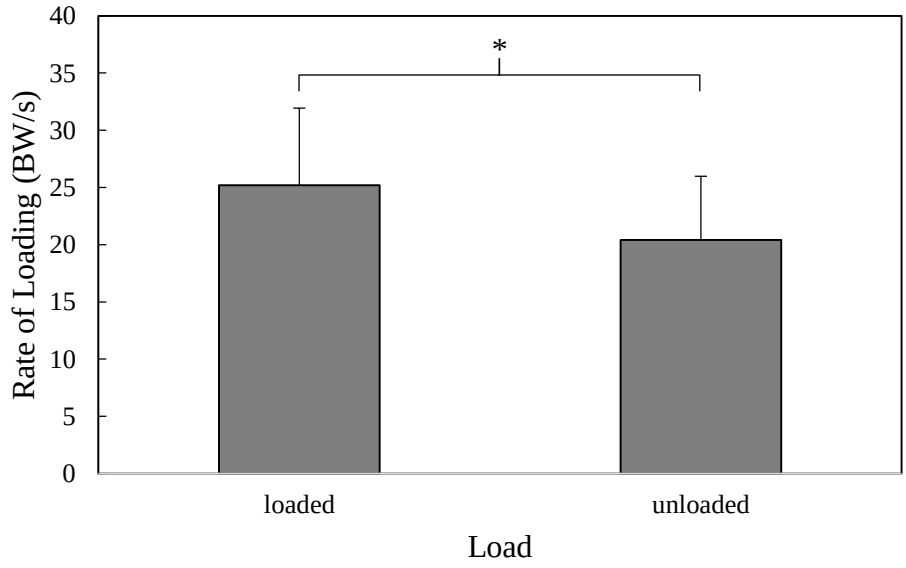


Figure 5.9 The effect of a carried load on the peak vertical rate of loading.
The peak vertical rate of loading during gait is plotted in the loaded (20%BW load) and unloaded conditions. The asterisk (*) denotes a significant difference at $p < .05$. Error bars indicate + 1 SD.

Heel-strikes were analyzed for all available gait trials in each condition, which ranged from a minimum of three, to a maximum of six trials. An increase in heel-strike transient

occurrence was considered to be a shift from less than 50% prevalence, to greater than 50% prevalence (Appendix I: Heel-Strike Transient Identification). Only two participants showed an increase in heel-strike transient occurrence immediately following the kneeling exposure (S09, S16). For participant S09, the increase was observed in both loaded and unloaded gait, and for S16, the increase was only observed in unloaded gait. One participant showed a decrease in heel-strike transient prevalence immediately following the kneeling exposure (S24).

5.2.2 Quadriceps Activation

Vastus Medialis Activation Onset

Muscle activation onset is reported relative to initial contact as both time (in seconds) and as a percentage of the gait cycle (%GC) (Table 5.5). There was a main effect of the kneeling exposure on the activation onset of vastus medialis ($F_{2,76}$ = 10.33, p = .0003) (Figure 5.14). Vastus medialis onset pre-kneeling (mean -0.159 s, (SD 0.034 s)) was significantly earlier than vastus medialis onset both post-kneeling (mean -0.152 s (SD 0.035 s), $F_{1,38}$ = 14.86, p = .0004, r = .53), and 30 minutes post-kneeling (mean -0.148 s (SD 0.032 s), $F_{1,38}$ = 15.98, p = .0003, r = .54). These findings support the hypotheses that a simulated occupation kneeling exposure would delay quadriceps activation onset immediately following the exposure (3a), with the effects persisting at 30 minutes post-kneeling (3c).

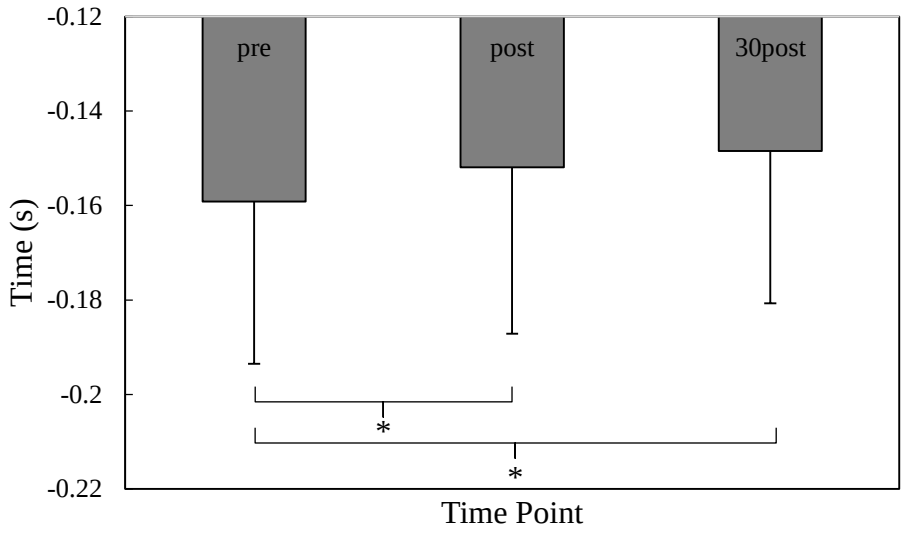


Figure 5.10 The effect of the kneeling exposure on vastus medialis activation onset.

Vastus medialis activation onset relative to initial contact of gait is plotted at baseline (pre), post-kneeling, and 30 minutes post-kneeling time points. The asterisk (*) denotes a significant difference at $p < .05$. Error bars indicate + 1 SD.

Table 5.5

Vastus Medialis Activation Onset for All Participants, Males Only, and Females Only

	Unloaded						Loaded					
	All		Male		Female		All		Male		Female	
Time Point	**s**	**%GC**	**s**	**%GC**	**s**	**%GC**	**s**	**%GC**	**s**	**%GC**	**s**	**%GC**
Baseline (Pre-Kneeling)	-0.156 (0.033)	-14.47 (3.10)	-0.158 (0.031)	-14.27 (2.79)	-0.154 (0.036)	-14.66 (3.39)	-0.162 (0.035)	-15.61 (3.39)	-0.164 (0.030)	-15.49 (2.85)	-0.160 (0.040)	15.74 (3.88)
Post-Kneeling	-0.146 (0.039)	-13.55 (3.66)	-0.144 (0.038)	-13.08 (3.45)	-0.148 (0.040)	-14.01 (3.83)	-0.156 (0.030)	-15.08 (2.92)	-0.159 (0.027)	-14.91 (2.35)	-0.157 (0.034)	-15.24 (3.41)
30 Minutes Post-Kneeling	-0.149 (0.032)	-13.84 (3.15)	-0.149 (0.031)	-13.60 (2.96)	-0.150 (0.034)	-14.09 (3.34)	-0.158 (0.033)	-14.14 (3.10)	-0.150 (0.027)	-14.21 (2.57)	-0.145 (0.037)	-14.07 (3.57)

Values are reported as mean (SD) for both loaded and unloaded conditions, as absolute time (s), and normalized to the length of the gait cycle (%GC) relative to initial contact.

There was also a main effect of load on the activation onset of vastus medialis ($F_{1,38}$ = 5.38, p = .0258, r = .35) (Figure 5.15). Vastus medialis onset occurred significantly earlier in the loaded condition, compared to the unloaded condition (loaded: mean -0.156 s (SD 0.033 s) vs. unloaded: mean -0.151 s (SD 0.035 s)). There was also a main effect of trial ($F_{2,76}$ = 6.74, p = .002); however, the difference was found between the first and third trials ($F_{1,38}$ = 11.00, p = .002, r = .47), and therefore will not be further investigated as this relationship is not relevant to the study hypotheses. No main effect of sex on VM onset was found ($F_{1,38}$ = 0.04, p = .08353, r = .03).

A significant 2-way interaction effect was found for load and the kneeling exposure ($F_{2,76}$ = 4.61, p = .0129). In both loading conditions, vastus medialis onset was delayed following the kneeling exposure; however, at 30 minutes post-kneeling, vastus medialis onset-delay remained approximately the same between the post and 30 minutes post-kneeling time points in the unloaded condition, whereas onset-delay continued to increase in the loaded condition ($F_{1,38}$ = 4.42, p = .0421, r = .32). Because the interaction occurred between post-kneeling and 30 minutes post-kneeling time points, this effect will not be explored as it does not pertain to the study hypotheses. Also, a significant 3-way interaction between trial, load, and the kneeling exposure was also found ($F_{4,152}$ = 2.77, p = .0293). Similarly, this interaction effect will not be explored as an analysis of a 3-way interaction effect would be better suited to a study with a larger sample size.

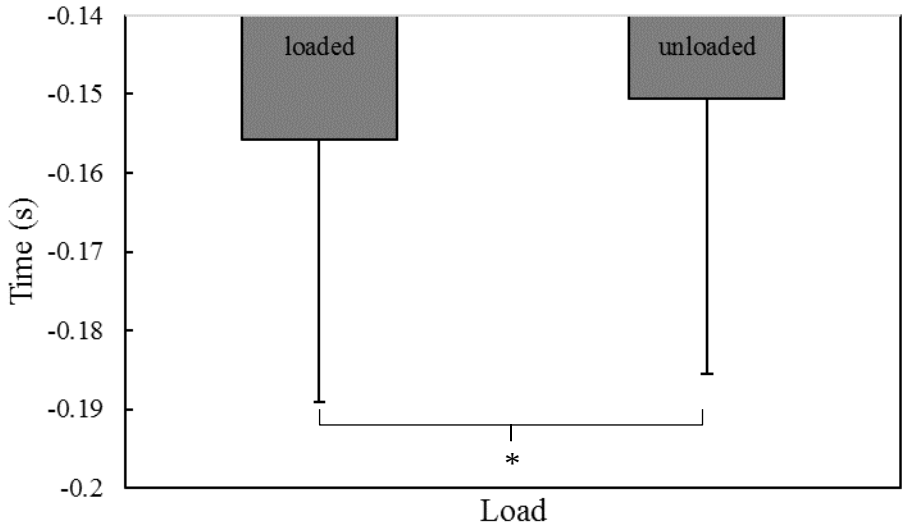


Figure 5.11 The effect of a carried load on vastus medialis activation onset.
Vastus medialis activation onset relative to initial contact of gait is plotted in the loaded (20%BW load) and unloaded conditions. The asterisk (*) denotes a significant difference at $p < .05$. Error bars indicate + 1 SD.

Vastus Medialis Magnitude of Activation

Contrary to hypothesis 3b and 3c, no main effects of the kneeling exposure were found for vastus medialis activation magnitude at initial contact ($F_{2,74} = 2.61$, $p = .0803$) (Figure 5.16). There was a main effect of load ($F_{1,37} = 41.02$, $p < .0001$, $r = .73$) such that the loaded gait trials elicited greater levels of muscle activation compared to unloaded gait trials (loaded: mean 2259.56 (SD 1244.51) vs. unloaded: 1874.66 (SD 1073.20)) (Figure 5.17). There was no main effect of trial number ($F_{2,74} = 1.10$, $p = .3371$, $r = 0.12$) or sex ($F_{1,37} = 3.32$, $p = .0766$, $r = .18$) on vastus medialis magnitude, nor were there any significant interaction effects.

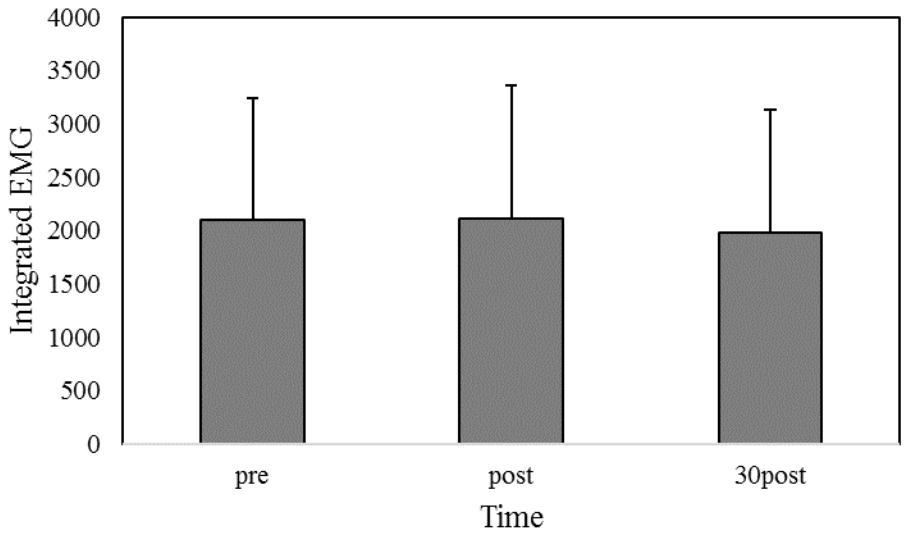


Figure 5.12 The effect of the kneeling exposure on vastus medialis activation magnitude.
Vastus medialis activation magnitude at initial contact of gait is plotted at baseline (pre), post-kneeling, and 30 minutes post-kneeling time points. Error bars indicate + 1 SD.

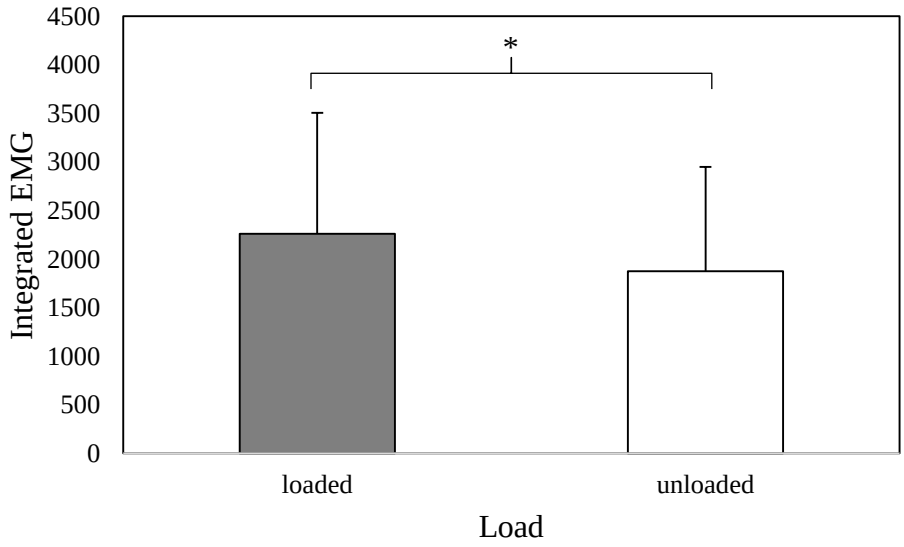


Figure 5.13 The effect of load on vastus medialis activation magnitude.
Vastus medialis activation magnitude at initial contact of gait is plotted in the loaded (20%BW load) and unloaded conditions. The asterisk (*) denotes a significant difference at $p < .05$. Error bars indicate + 1 SD.

5.3 Frontal Plane Knee Motion during Squat Transitions

Peak Deviation of the Knee Joint Trajectory

There was a main effect of the kneeling exposure on the peak deviation of the knee joint center from the plane ($F_{2,76}$ = 5.44, p = .0096) (Figure 5.18, Table 5.6). The peak deviation from the plane at baseline (mean 0.207 m (SD 0.110 m)) was significantly smaller than the peak knee deviation from the plane post-kneeling (mean 0.229 m (SD 0.107 m), $F_{1,38}$ = 8.59, p = .0057, r = .43). The peak deviation from the plane at the 30 minutes post time point was also significantly greater than the baseline measure (mean 0.227 m (SD 0.108 m), $F_{1,38}$ = 5.32, p = .0267, r = .35). These findings support hypotheses 4a and 4b that an increase in the peak frontal plane knee motion would occur following a kneeling exposure, and persist for 30 minutes post-kneeling.

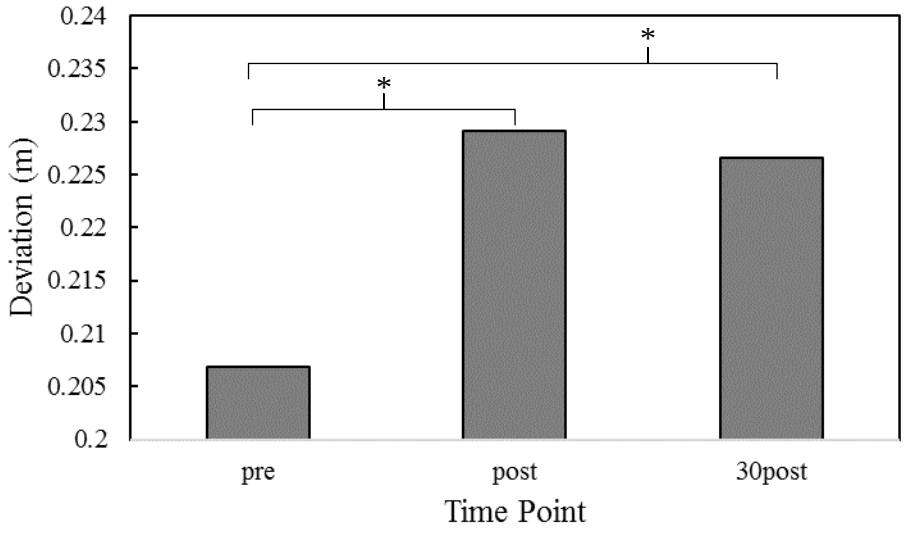


Figure 5.14 The effect of the kneeling exposure on peak knee deviation.
Peak knee joint center deviation from the body-fixed plane is plotted at baseline (pre), post-kneeling, and 30 minutes post-kneeling time points. The asterisk (*) denotes a significant difference at p < .05.

Table 5.6

Peak Knee Joint Center Deviation from the Plane for All Participants, Males Only, and Females Only

	Trial 1			Trial 2			Trial 3		
Time Point	**All**	**Male**	**Female**	**All**	**Male**	**Female**	**All**	**Male**	**Female**
Baseline (Pre-Kneeling)	0.200 (0.112)	0.200 (0.119)	0.200 (0.106)	0.209 (0.110)	0.200 (0.117)	0.217 (0.102)	0.212 (0.107)	0.206 (0.112)	0.217 (0.101)
Post-Kneeling	0.221 (0.110)	0.214 (0.115)	0.229 (0.106)	0.234 (0.105)	0.224 (0.109)	0.244 (0.101)	0.232 (0.107)	0.220 (0.109)	0.245 (0.103)
30 Minutes Post-Kneeling	0.222 (0.110)	0.214 (0.114)	0.231 (0.106)	0.229 (0.107)	0.219 (0.114)	0.240 (0.098)	0.228 (0.107)	0.221 (0.110)	0.235 (0.104)

Values are reported as mean (SD) for males and females, by trial, in meters.

Unexpectedly, there was also a main effect of trial ($F_{2,76}$ = 9.09, p = .0010). The first trial (mean 0.215, SD 0.111 m) had a significantly smaller peak deviation than both the second trial (mean 0.224 m (SD 0.108 m), $F_{1,38}$ = 12.65, p = .0010, r = .45) and the third trial (mean 0.224 m (SD 0.107 m), $F_{1,38}$ = 9.47, p = .0039, r = .5) (Figure 5.19). No main effect of squat speed ($F_{1,38}$ = 0.09, p = .7685, r = 0.05), squat direction ($F_{1,38}$ = 3.88, p = .0562, r = .30), or sex ($F_{1,38}$ = .23, p = .6353, r = .08) were found. A 2-way interaction between participant sex and squat speed was found ($F_{1,38}$ = 5.79, p = .0210, r = 0.36), as well as a significant 3-way interaction between squat speed, trial number, and the kneeling exposure ($F_{4,152}$ = 2.68, p = .0449); however, these effects do not pertain to the study hypotheses and therefore will not be explored within the scope of this document.

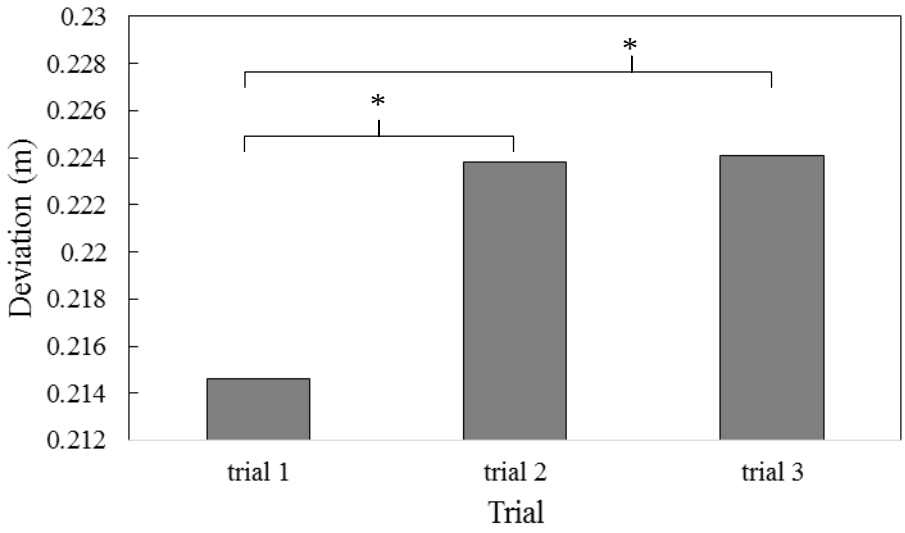


Figure 5.15 The effect of trial on peak knee deviation.

Peak knee joint center deviation from the body-fixed plane is plotted for the first, second, and third trials. The asterisk (*) denotes a significant difference at p < .05.

Mean Deviation of the Knee Joint Trajectory

There was a main effect of the kneeling exposure on mean knee joint deviation from the plane during the squat transitions ($F_{2,76}$ = 5.82, p = .0044). Mean deviation at baseline (mean

0.084 m (SD 0.048 m)) was significantly smaller than both the post-kneeling (mean 0.091 m (SD 0.047 m), $F_{1,38}$ = 6.05, p = .0186, r = .37) and 30 minutes post-kneeling (mean 0.093 m (SD 0.049 m), $F_{1,38}$ = 8.43, p = .0061, r = .43) values (Figure 5.20). These findings support hypotheses 4a and 4b that an increase in the mean frontal plane knee motion would occur following a kneeling exposure, and persist for 30 minutes post-kneeling.

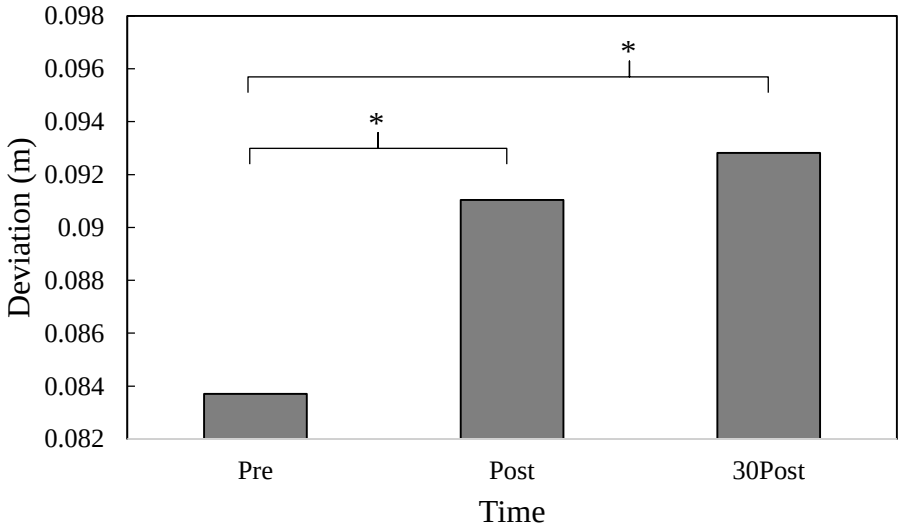


Figure 5.16 The effect of the kneeling exposure on mean knee deviation.
Mean knee joint center deviation from the body-fixed plane is plotted at baseline (pre), post-kneeling, and 30 minutes post-kneeling time points. The asterisk (*) denotes a significant difference at p < .05.

There was also a main effect of squatting speed on the mean knee deviation ($F_{1,38}$ = 9.85, p = .0033, r = .45). Slow squatting (mean 0.092 m (SD 0.051 m)) resulted in significantly greater mean knee joint trajectory deviation than fast squatting (mean 0.086 m (SD 0.045 m) (Figure 5.21). There was no main effect of the direction of the squat ($F_{1,38}$ = 1.12, p = .2969, r = .17), the trial number ($F_{1,38}$ = 0.72, p = .4885, r = .10), or the sex of the participant ($F_{1,38}$ = 0.17, p = .6853, r = .07). Significant 2-way interaction effects were found between squat speed and squat direction (ascent vs. descent) ($F_{1,38}$ = 7.06, p = .0114, r = .40), and between trial number and squat direction ($F_{2,76}$ = 5.82, p = .0045). A significant 4-way interaction effect was found for

variables of squat speed, direction, trial number, and the kneeling exposure ($F_{4,152}$ = 2.90, p = .0237). These significant 2- and 4-way interaction effects will not be explored within the scope of this document as they do not pertain to the study hypotheses and sufficient analysis may require a larger study sample size.

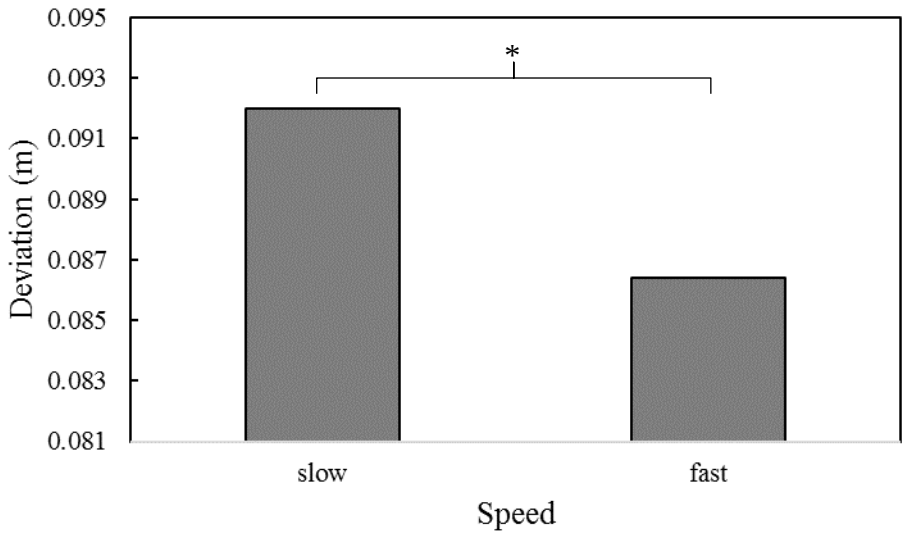


Figure 5.17 Effect of speed on mean squat deviation.
Mean knee joint center deviation from the body-fixed plane is plotted for squatting at the slow (44 bpm) and the fast (88bpm) paces. The asterisk (*) denotes a significant difference at p < .05.

There was a lack of agreement between knee joint angles and the knee joint center deviation measures during the squat transitions, such that increases in the deviation of the knee joint center did not necessarily coincide with changes in knee joint angles. For example, participant S16 demonstrated a trend towards an increase in the peak knee abduction angle (knee valgus) post-kneeling when squatting at the slow pace, but showed little to no change in the knee abduction angle in the fast squats (Figure 5.22). However, analysis of the knee deviation data suggests a trend towards an increase in deviation of the knee joint center in *both* the fast and slow squats following the kneeling exposure (Figure 5.23). In addition, the peak knee abduction angle occurs approximately at the halfway point of the transition, whereas the peak deviation

from the plane occurs at the bottom of the squat. Knee deviation and joint angle data during squat transitions for all participants can be found in Appendix K: Tri-Axial Knee Joint Angles and Knee Deviation during Squat Transitions.

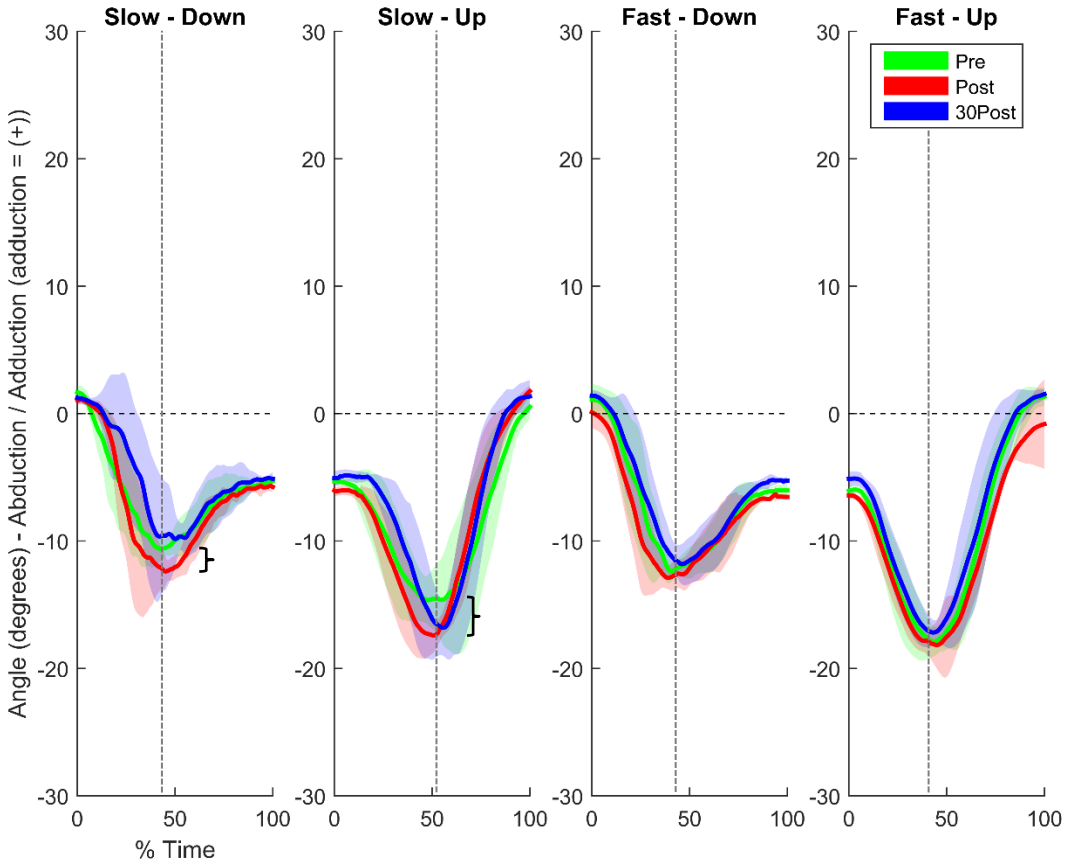


Figure 5.18 The knee abduction angle for S16 during fast and slow squats.

Data are time normalized to 101 data points between the start and end of each direction of movement (ascent and descent). Differences in the peak knee abduction angle between baseline and post-kneeling measures during slow squats are highlighted using brackets. The vertical dashed line indicates the approximate location of the peak knee abduction angle at all three time points. Shaded error bars indicate ± 1 SD.

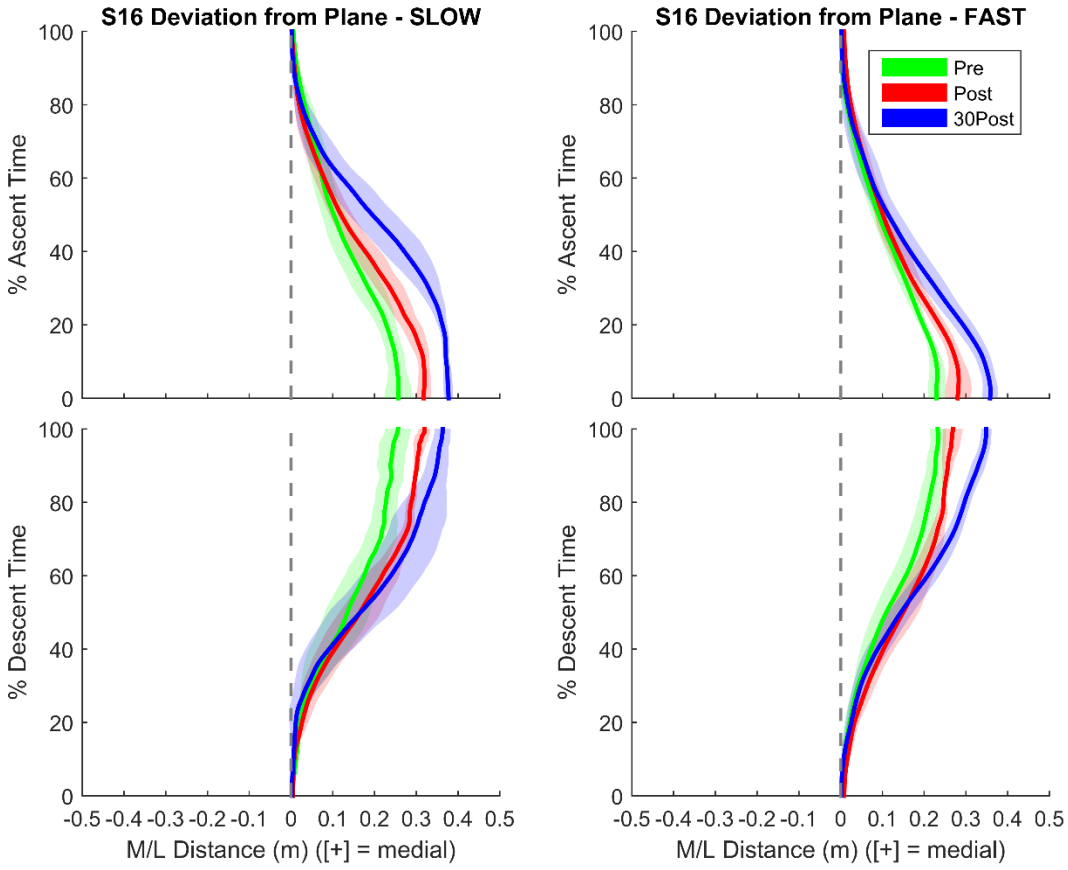


Figure 5.19 Squat deviation from the plane for S16 during fast and slow squats.
Data are time normalized to 101 data points between the start and end of each direction of movement (ascent and descent). Note that the plot is configured with 'time' on the y-axis and 'medial/lateral deviation' on the x-axis to illustrate the direction of deviation in a more anatomically relevant way. Shaded error bars indicate ± 1 SD.

5.4 Knee Proprioception

There was no main effect of the kneeling exposure on the absolute angular error (AAE) during the knee angle reproduction task ($F_{2,76}$ = 0.01, p = .9982) (Figure 5.4). The absolute angular error was 2.7° at baseline (pre), post-kneeling, and 30 minutes post-kneeling time points, (pre: SD 2.2°, post: SD 2.1°, 30post: SD 2.4°). Therefore, contrary to hypotheses 5a and 5b, the kneeling exposure did not change participants' knee proprioceptive acuity based on this method of measurement.

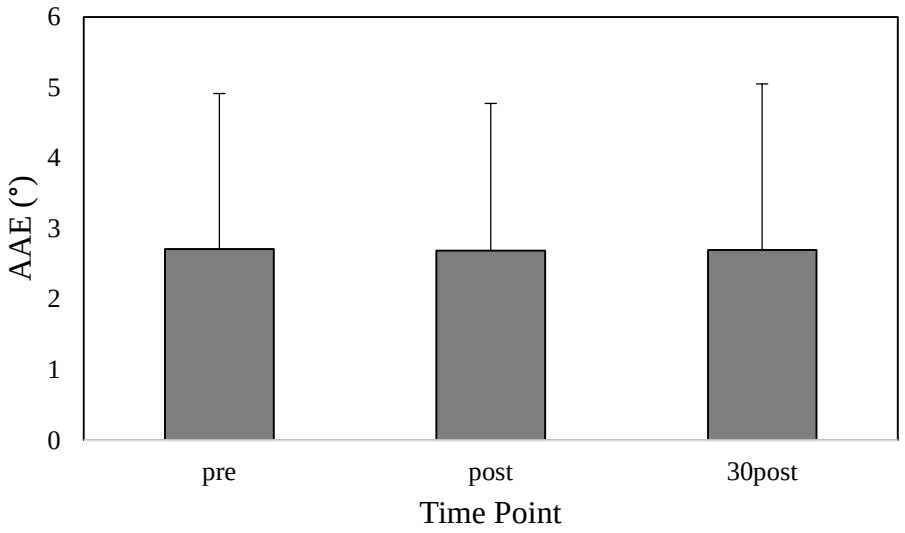


Figure 5.20 The effect of the kneeling exposure on knee proprioception.
The average absolute angular error is plotted at baseline (pre), post-kneeling, and 30 minutes post-kneeling time points. Error bars indicate + 1 SD.

There was a main effect of trial on absolute angular error ($F_{2,76}$ = 8.82, p = .0004). The first trial (mean 3.3° (SD 2.7°)) had significantly greater error than both the second (mean 2.5° (SD 1.9°), $F_{1,38}$ = 10.06, p = .003, r = 0.46) and third (mean 2.3° (SD 1.8°), $F_{1,38}$ = 14.68, p = .0005, r = .53) trials (Figure 5.5).

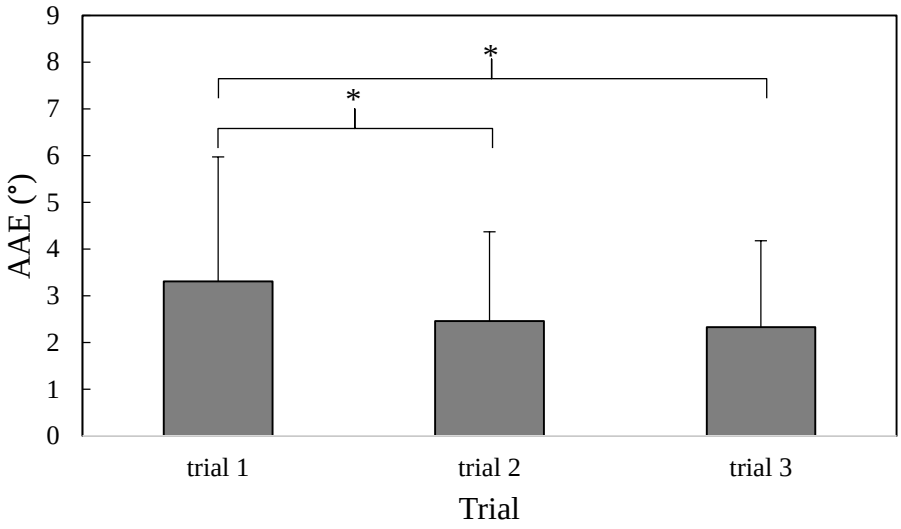


Figure 5.21 The effect of trial on knee proprioception.
The average absolute angular error is plotted for the first, second, and third trials. The asterisk (*) denotes a significant difference at p < .05. Error bars indicate + 1 SD.

There was also a main effect of sex. Across all conditions males exhibited significantly smaller absolute angular error compared to females (male: mean 2.2° (SD 2.0°), female: mean 3.2° (SD 2.3°), $F_{1,38} = 5.47$, p = .0246, $r = .35$). No significant interaction effects were found.

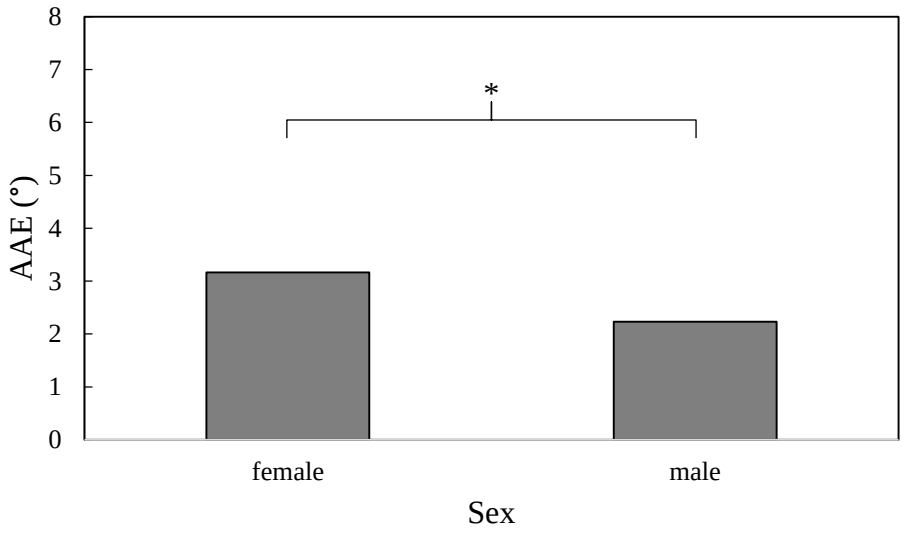


Figure 5.22 The effect of sex on knee proprioception.
The average absolute angular error is plotted for males and females. The asterisk (*) denotes a significant difference at p < .05. Error bars indicate + 1 SD.

Although no main effects of kneeling on knee proprioception were observed, some participants demonstrated trends that might suggest a deficit occurred. Participants S13, S14, S18, S24, S29, S33, and S45 all demonstrated an initial impairment in knee proprioceptive acuity of at least 1° post-kneeling (7/40 participants – 17.5%) (Figure 5.7). S22 did not quite reach the threshold (0.9° difference).

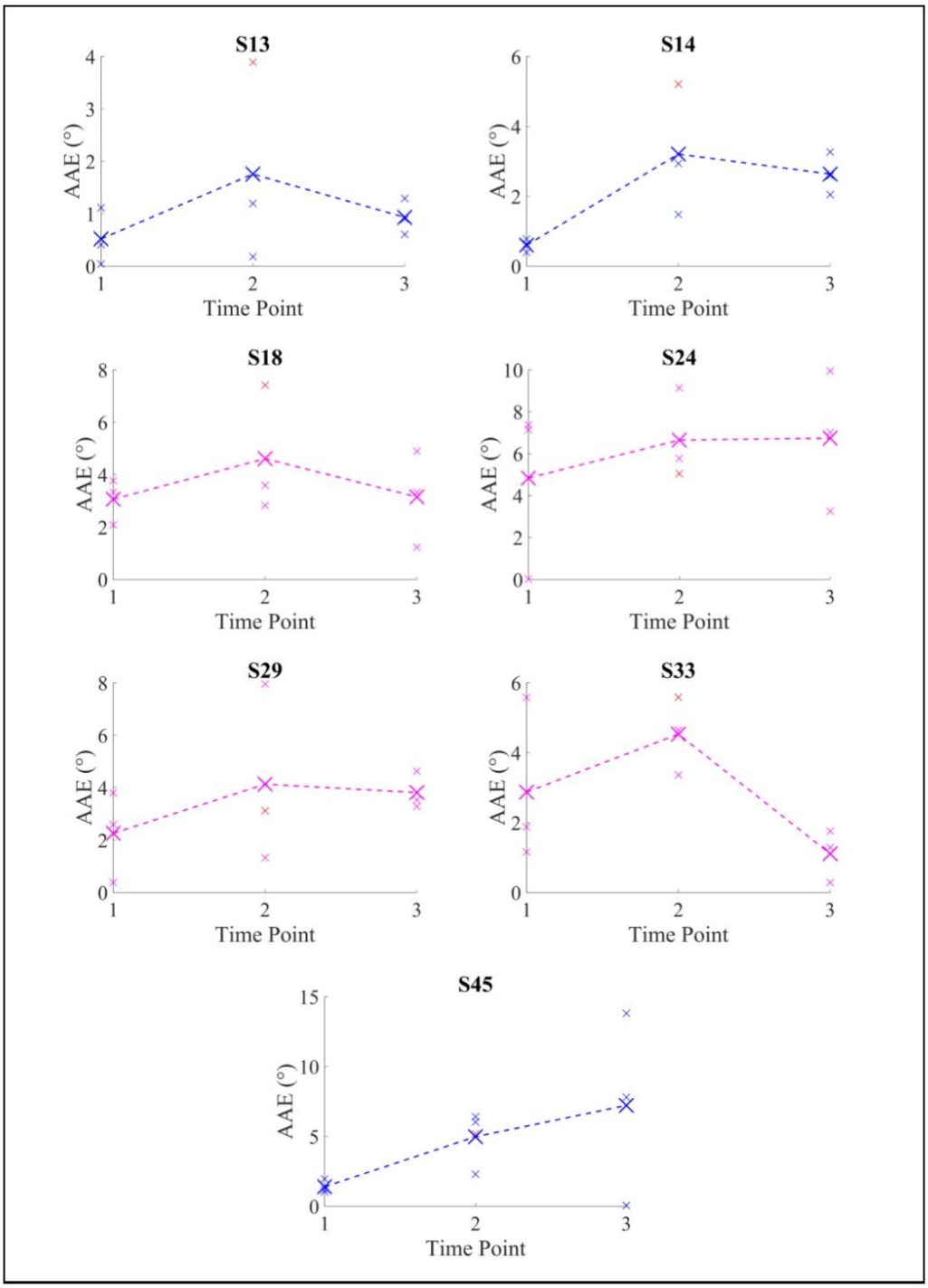


Figure 5.23 Participants who demonstrated an increase in AAE following the kneeling exposure. Male participants are plotted in blue and females in pink. 'Time Point' refers to baseline (1), post-kneeling (2), and 30 minutes post-kneeling (3) measures. Large, bolded 'X's indicate the mean AAE for that time point, smaller 'x's' indicate each individual trial taken. The small, red 'x' at time point 2 indicates the AAE for the first trial measured immediately post-kneeling. Only participants whose average AAE difference between baseline and post-kneeling exceeded 1° are reported.

6.0 Discussion

This project investigated the effects of a simulated occupational kneeling exposure on a number of variables in order to evaluate the hypothesis that a simulated occupational kneeling exposure may induce adaptations in neuromuscular control and movement patterns during gait and squat transitions that could contribute to knee osteoarthritis risk. It was also hypothesized that any negative adaptations may be linked to impairments in joint proprioception, measured as joint position sense. In this way, the results of this study would contribute to improving our understanding of the link between occupational high knee flexion postures and knee osteoarthritis development. The interpretation of the results from the current study was complicated by the fact that, as in all studies on humans, human variability can be quite robust, as indicated by large standard deviation values. Each participant in this study was unique and responded to the kneeling exposure in different ways, and the mean response does not necessarily reflect the response of each participant. In attempt to provide as complete a picture of the results of this study as possible, individual participant curves and values are provided in the Appendices (Appendix I: Heel-Strike Transient Identification, Appendix K: Tri-Axial Knee Joint Angles and Knee Deviation during Squat Transitions).

Knee Adduction Moment

Contrary to the hypothesis, no significant differences were found in the peak KAM during gait, regardless of the load condition, following the kneeling exposure (Figure 5.8). This is contradictory to previous work that showed an increase in the KAM following a 30-minute sustained static kneeling exposure (Kajaks & Costigan, 2015). In the previous study, a within-participant root mean squared difference (RMSD) was calculated for the mean knee adduction moment waveforms for the baseline and post-kneeling conditions in order to examine the

difference in the mean curves between the two conditions. This methodology is different from the analysis used in the current study, where the peak KAM was compared. Based on a visual inspection of the KAM curves from the current study, it is unlikely that a similar analysis method would identify any changes in the mean curves (Appendix L: Gait Kinetics and Kinematics).

The difference in kneeling exposure between the two studies likely accounts for the difference in findings. In the previous study, participants were required to maintain a static full-flexion kneeling posture for 30 minutes, while in the current study, participants completed a more dynamic exposure which involved cyclic shifts from full-flexion kneeling to single-arm supported kneeling. Time spent in full-flexion kneeling totaled only six minutes over the course of the half-hour exposure in the current study. It is possible that the exposure in the current study, while more occupationally relevant based on observation of workers in the floor laying profession, was insufficient to elicit measurable changes in gait.

It should be noted that because the mechanism of change resulting in altered kinetics of gait in the previous study was unknown, it is not possible to state exactly why a more dynamic kneeling exposure would be insufficient to cause a change. For example, although no changes in knee joint proprioception were observed in the current study that is not to say that knee proprioception was not altered in the previous study and contributed to the observed changes, because proprioceptive acuity was not measured. Similarly, the author proposed that time spent in a high-flexion kneeling posture could induce joint laxity. Knee joint laxity was not measured in either study, but it is possible that a dynamic kneeling exposure does not induce, or induces less joint laxity (or ligamentous creep), which may account for the lack of change observed in gait.

As hypothesized, the peak KAM increased when participants carried an external load (Figure 5.9). This finding agrees with previous work that evaluated the external KAM while participants carried a set weight of 13.6 kg in front of the body with two hands (Hall et al., 2013). The authors found a 30% increase in the peak KAM during stance phase of gait for healthy young participants. The results from the current study suggest only an 18% increase when comparing the loaded and unloaded gait trials recorded at baseline; however, this discrepancy can likely be explained by differences in normalization methods for the KAM and the mass of the carried load.

In the current study, females had a significantly higher peak KAM compared to males across all conditions (Figure 5.10). These findings are similar to another study that also found that females had a significantly higher peak KAM compared to males, even after normalizing the results to %BW*H (Barrios & Strotman, 2014). Although the results were not statistically significant, at least one study found the opposite relationship - that the first peak of the KAM tended to be smaller in females compared to males (Kumar et al., 2015). The exact mechanism for the observed difference in peak KAM between males and females in the current study was not readily apparent. Analysis of the knee and ankle joint kinematics and ground reaction force at the moment of peak KAM showed no significant difference between males and females.

For unloaded gait, the baseline peak KAM during the first half of stance phase of gait (Table 5.3) was somewhat smaller than values reported in most previous research that has examined this variable, but within the reported standard deviations (Table 6.3). The somewhat smaller values observed in this study are not likely due to footwear – footwear is actually expected to increase the knee adduction moment (Radzimski, Muendermann, & Sole, 2012). It is possible that the difference is due to dissimilar resolution coordinate systems (e.g. tibial vs.

femoral vs. laboratory), which are not always reported and are known to affect the shape and magnitude of moment waveforms (Brandon & Deluzio, 2011).

Table 6.1

Peak Knee Adduction Moments during Gait Reported in the Literature for Healthy Participants

Author, year	Participants	Footwear	Peak KAM Identification Method	Peak KAM (%BW*H)
Current Study Results (baseline)	20F, 20 M, 21.4 ± 2.48 Y	Participants wore their own running shoes	Peak knee adduction moment during early stance phase	All: 2.46 ± 0.82 M: 2.06 ± 0.72 F: 2.86 ± 0.72
Barrios & Strotman, 2014	10 F, 21.2 ± 2.7 Y 10 M, 23.7 ± 4.5 Y	Participants wore the Nike Air Pegasus running shoe	Peak knee adduction moment during the entire stance phase	*M: 2.83 ± 0.49 *F: 3.06 ± 0.46
Dowling, Fisher, & Andriacchi, 2010	3 F, 6 M, 25.2 ± 5.2 Y	Participants wore their own running shoes	First peak of the knee adduction moment during stance phase	2.48 ± 0.40
Noyes, Schipplein, Andriacchi, Saddemi, & Weise, 1992	7 F, 9 M, 19-45 Y	Not reported	Peak knee adduction moment during the entire stance phase	2.75 ± 0.55
Patterson, Delahunt, & Caulfield, 2014	17 F, 23.7 ± 3.12 Y	Barefoot	Peak knee adduction moment during early stance phase	3.89 ± 1.01
Zabala, Favre, Scanlan, Donahue, & Andriacchi, 2013	19 F, 26 M, 30.2 ± 4.68 Y	Not reported	First peak of the knee adduction moment during stance phase	2.94 ± 0.73

NOTE: when the authors reported peak KAM normalized to body mass and height, instead of body weight, values were divided by 9.81 and multiplied by 100 to convert to %BW*H (denoted (*)).

Peak Rate of Loading and Heel-Strike Transients

No significant effect of the kneeling exposure was found for the peak rate of loading. This finding was contrary to the hypothesis, but supports the null findings also found for the peak KAM, and contributes to the concept that the kneeling exposure in the current study did not

elicit significant changes in gait. Following a thorough search of the literature, it appears that no other authors have analyzed the change in the peak external rate of loading while carrying an external load. However, the finding that the peak vertical rate of loading increased when participants carried a load is not unexpected, as the values were normalized to bodyweight. Thus, an increase in the rate of loading would be expected with an increase in the external load compared to unloaded gait, because the participants were required to maintain the same gait speed.

With the understanding that carrying a load increased the external rate of loading, workers may be advised to walk more slowly when carrying heavy materials to reduce the peak rate of loading, and by extension, the peak forces at the knee. However, while reduced gait speed will decrease the peak rate of loading (as well as the peak knee adduction moment), walking more slowly will increase the KAM impulse due to increased absolute time spent in the stance phase. The KAM impulse has also been associated with medial knee joint loading (Calder et al., 2014; Kean et al., 2009), and it has been shown in the current study that carried external loads already increase the KAM. Therefore, a better suggestion for workers, in the interest of sparing knee joint health, might be to use a dolly or trolley to move materials when the workspace permits it, or, when possible, make more frequent trips with lighter loads.

The peak rate of loading at baseline in the current study (Table 5.4) was similar to the findings of previous work that examined the peak rate of loading using comparable methods. The participants in the previous study were significantly older (113 F, 91 M, 64.7 ± 8.6 Y), but participants also wore running shoes. The authors reported a very similar peak rate of loading as the current study (22.37 ± 8.40 BW/s) (Hunt et al., 2010).

Heel-strike transients were not as common in the tested population as some studies have suggested (Table 6.4). Only two participants (5% of the study sample) demonstrated heel-strike transients at least 50% of the time during baseline gait trials. It is possible that the reduced prevalence of heel-strike transients observed in the current study may be related to the fact that participants wore running shoes. For example, it has been suggested that shoe cushioning, such orthotic inserts, can reduce peak magnitudes of 'transient stress waves' during gait (Collins & Whittle, 1989). In addition, in a case study, an individual who demonstrated heel-strike transients when walking barefoot showed a significant decrease when wearing shoes, although the peak vertical force was higher (Radin et al., 1986). Also, a minimum of only three trials were taken in each of the loading conditions to reduce the burden on the participants and ensure that post-measures could be collected within the 30-minute post-kneeling window. In comparison, previous analyses of heel-strike transients report prevalence based on ten or more trials. Therefore, it is possible that insufficient strides were taken to determine the true presence of heel-strike transients.

Table 6.2

Heel-Strike Transients: Comparison of HST Prevalence Results across Studies

Author, year	Participants	Footwear	HST Identification	Definition of a Heel-Striker	# of Heel-Strikers
Current Study Results (baseline)	20F, 20 M, 21.4 ± 2.5 Y	Participants wore their own running shoes	If, during the upper 50% of the vertical ground reaction force immediately following initial contact, the force peaked, and then decreased by more than 1.2% of the peak vertical ground reaction force.	Participant demonstrated heel-strikes in at least 50% of trials	2/40 (5%)
Hunt et al., 2010	113 F, 91 M, 64.7 ± 8.6 Y	Participants wore their own running shoes	If, during the upper 50% of the vertical ground reaction force immediately following initial contact, the force peaked, and then decreased by more than 0.5% of the peak vertical ground reaction force.	Participant demonstrated heel-strikes in at least 75% of trials	39/204 (19%)
Liikavainio et al., 2007	21 F, 6 M, 66.2 ± 7.6 Y	Participants wore their own gym shoes	When the ratio between the peak and subsequent local minimum force ≥ 1.2.	If the calculated value was ≥ 1.2	3/27 (11%)
Radin et al., 1986	11 F, 10 M, 29.6 (20-45) Y	barefoot	When the ratio between the peak and subsequent local minimum force ≥ 1.2.	An average heel-strike transient ratio of ≥ 1.2	7/21 (33.3%)
Riskowski et al., 2005	38 F, 23.5 ± 2.60 Y	barefoot	When the ratio between the peak and subsequent local minimum force ≥ 1.2, within the 50 ms window following initial contact	An average heel-strike transient ratio of ≥ 1.2	16/38 (42%)

Quadriceps Activation

Following the kneeling exposure, VM onset was delayed (onset occurred significantly closer to initial contact) during gait. Vastus medialis onset occurred about 7 ms later compared to baseline onset values immediately post-kneeling, and 11 ms later when gait was measured 30 minutes post-kneeling (Figure 5.14). These results support the hypothesis that a kneeling exposure would result in a negative adaptation to neuromuscular control. As previously discussed (3.1.1), the anticipation of external loading through quadriceps pre-activation is thought to be a protective mechanism to reduce the rate of loading at initial contact (Jefferson et al., 1990). In this way quadriceps pre-activation acts to distribute forces at impact and decrease the rate of loading at the joint through eccentric loading (Felson, 2004a; Lindstedt et al., 2001). It should be acknowledged, however, that excess activation of antagonistic muscles crossing the knee may actually result in greater axial compression, therefore inducing greater cartilage loading (Bennell, Hunt, Wrigley, Lim, & Hinman, 2008). Muscle activation was only recorded for VM, therefore, the potential compressive loads due to muscle co-activation cannot be addressed within the scope of this project.

Although a significant delay in quadriceps pre-activation was found, no increase was observed in the variables used in the current study to evaluate joint loading (e.g. peak vertical rate of loading and the prevalence of heel-strike transients). The lack of change in the surrogate measures of joint loading suggests that while the delay in VM activation onset was statistically significant, and such a delay would be considered a negative adaptation to the kneeling exposure, the effects did not manifest as biologically significant changes. In addition, there does not appear to be a standard in the literature to indicate the minimum difference in activation onset that would result in clinically relevant changes. Therefore, due to the lack of significant findings

in the external rate of loading and heel-strike transient presence, the difference in VM onset observed as a result of the kneeling exposure does not appear to be biologically relevant. Further investigation of any differences in lower limb joint angles at the time of VM activation onset would be beneficial to help support this stance.

The carried load also elicited a statistically significant change in VM onset such that VM turned on earlier with respect to initial contact compared to unloaded gait (~ 6 ms) (Figure 5.15). Again, this difference is likely too small to be considered biologically relevant. It is however, interesting to note that earlier VM activation is the opposite trend to previous loaded gait studies which found that adding a load resulted in a delay in quadriceps activation onset (Simpson, Munro, & Steele, 2011; Stastny et al., 2014). One could argue that earlier activation of the quadriceps found in the current study would be a positive adaptation to an external load that would be expected to decrease the external rate of loading during loaded gait. This argument would again be based on the concept that quadriceps pre-activation acts to facilitate the attenuation of external loads (Felson, 2004a; Lindstedt et al., 2001). However, even with statistically earlier VM activation onset, the peak rate of loading was still significantly greater for the loaded gait trials (Figure 5.13). Other research groups have suggested that delayed quadriceps activation occurs during loaded gait trials to allow for greater knee flexion at initial contact (Simpson et al., 2011; Simpson, Munro, & Steele, 2012). Presumably, greater knee flexion was thought to be a product of reduced stiffness at the knee joint and therefore greater load attenuation at the knee. In the current study, there was no main effect of load on the knee flexion angle at initial contact (loaded: mean 2.79° SD 3.43°, unloaded: mean 3.02° SD 3.46°, $F_{1,36} = 2.21$, p = .1456, $r = .24$), suggesting that any changes in VM onset did not affect joint kinematics.

The finding of a baseline average VM onset during unloaded gait of -14.5 ± 3.1 %GC walking at a speed of at 1.4 m/s, corresponds well with other work on gait at self-selected walking speeds (Table 5.5). Onset times of -19.6 ± 5.0 %GC in healthy older adults (Kwon, Minor, Maluf, & Mueller, 2003), and -12.2 ± 4.6 %GC in healthy females 26 – 45 years of age (Powers, Landel, & Perry, 1996) have been previously reported (NOTE: for comparison purposes, these values have been converted to %GC *pre*-initial contact by subtracting the values from 100). Very little research has analyzed the effects of load carriage on the timing of lower limb muscle activation. Vastus medialis onset in a study of 15 strength trained men carrying 25% of their body weight had a VM onset time of -28.60 ± 14 %GC when walking at an unknown speed (Stastny et al., 2014). This does not agree with the finding from the current study of an onset time of -15.5 ± 2.9 %GC for male participants when carrying 20 %BW load (Table 5.5). Although the quadriceps group analyzed was vastus lateralis, a study on prolonged backpack load carrying in females found an activation onset of -0.099 s ± 0.028 s, and -0.094 s ± 0.030 s when carrying loads of 20 and 30 %BW (Simpson et al., 2011). These findings are much closer to initial contact than the findings from the current study for VM onset with a 20 %BW front-carried load (Table 5.5). The differences observed between the current study and these load carrying studies are likely due to differences in the methodology (e.g. muscle analyzed, location of the load, load mass) and participant groups.

There was no effect of the kneeling exposure on VM activation magnitude. Although this finding does not support the hypothesis that activation magnitude at initial contact would decrease, it does correspond with the null findings for the surrogate measurements analyzed in the current study for knee loading. Should there have been an increase in the peak rate of loading or in the prevalence of heel-strike transients, a reduction in quadriceps activation

magnitude would also have been expected. Because no changes were observed in the rate of loading or heel-strike transient prevalence, the lack of change in VM activation magnitude seems to agree with these findings. The activation magnitude of VM at initial contact was greater in the loaded condition, which would be expected due to the greater muscular effort required to maintain the set gait speed while carrying 20% of body weight.

Frontal Plane Knee Motion during Squat Transitions

Frontal plane knee motion, quantified as the deviation of the knee joint center from an anatomically defined, body-fixed plane, was used to evaluate changes in neuromuscular control during squat transitions to-and-from the floor. Using this measure, greater absolute deviation was considered greater frontal plane knee motion and therefore a negative adaptation to the kneeling exposure. This interpretation was based on the understanding that an individual's movement patterns reflect that person's own neuromuscular control strategy (Frost, 2013), and that greater frontal plane knee motion may increase injury risk (Hewett et al., 2005). In the current study, both the peak and mean deviation of the knee joint center had greater amplitudes following the kneeling exposure (Figure 5.18, Figure 5.20). This finding of increased frontal plane knee motion does not indicate an increase in knee osteoarthritis risk, necessarily. It does however highlight that the kneeling exposure altered neuromuscular control strategy during a squat transition.

Although it is unknown exactly how increased frontal plane knee motion during a squat transition would affect knee osteoarthritis risk, it could be hypothesized that increased deviation from neutral may increase the risk of traumatic injury to the joint, which would contribute to knee osteoarthritis risk. For example, as mentioned, meniscal tears are a common injury among

workers who frequently kneel (McMillan & Nichols, 2005; Reid et al., 2010; Snoeker et al., 2013), and both the injury itself, as well as the potential subsequent meniscectomy, are associated with an increased risk for the development of knee osteoarthritis (Cicuttini, Forbes, Yuanyuan, Rush, & Stuckey, 2002; Englund et al., 2009). Meniscal tears are usually caused by sudden shear forces between the tibia and the femur when the foot is planted and the femur is internally rotated (Prentice, 2011; Shiraev, Anderson, & Hope, 2012). While a neutral knee alignment does not guarantee that an individual has eliminated the risk of knee injury, based on findings that certain movement patterns during specific tasks can be risk factors for injury (Hewett et al., 2005; Pohl, Mullineaux, Milner, Hamill, & Davis, 2008), injury risk may be greater in more highly deviated postures.

Although not the main focus of this project, when the effects of the kneeling exposure were ignored, the mean knee joint center deviation was greater in slow squatting compared to fast squatting (Figure 5.21). This relationship seems to be somewhat counter-intuitive, and in fact is the opposite of the findings of a similar analysis which found, in general, that increasing movement speed had a negative effect on frontal plane knee motion when analyzing a squat task in male firefighters (Frost et al., 2015). The exact mechanism for the altered movement strategy observed for squatting at different speeds is not known, but differences in movement patterns are thought to be affected by the perception of risk (Dufek, Bates, Stergiou, & James, 1995), as well as fitness, body awareness and coordination, previous task experience, and attention (Frost et al., 2015). It is likely ill advised to recommend that workers move through squat tasks quickly simply to reduce mean knee deviations, especially considering that there was no main effect of speed on peak knee deviation. In addition, although the current study did not address this concern, workers may squat to the floor to transfer loads. In such cases, moving quickly may not

be possible (and would likely be further ill advised). It is clear that the speed of movement affected the neuromuscular control strategy, and workers should be aware of neutral movement patterns when squatting.

It was also found that the first squat in each set showed a smaller peak deviation than each of the subsequent trials, regardless of squat speed or the kneeling exposure. Participants in the current study performed the squats in succession (i.e. one after the other without significant re-adjustment of foot placement). Based on observation of floor installers in the workplace, workers do not typically perform squats in succession, so practically speaking, squat performance may be more similar to the first squat, depending on the length of time that has passed since the previous squat. However, there was still an increase in peak deviation following the kneeling exposure. Therefore, while frontal plane knee motion increased as the trials went on, the first squat still showed an overall increase in peak deviation and thus indicates an immediate change in kinematics, and by extension neuromuscular control during a squat transition.

A lack of association was observed between knee joint angles and frontal plane knee motion during the squat transition task. This finding suggests that the values of knee joint angle and deviation from the plane should not be used to evaluate neuromuscular control in isolation, as the measures clearly evaluate different aspects of the transition. The lack of association between knee joint angles and frontal plane knee motion is likely due to the fact that the deviation measure takes into account the positioning of the entire lower body, including the ankle and hip, whereas knee joint angle is a simple joint-specific evaluation of the movement.

Some of the differences observed between measures may be due in part to the way the thigh segment was tracked and re-created in Visual3D. The thigh cluster was firmly affixed to the lateral aspect of the thigh using double-sided tape, Hypafix, and a Velcro strap, and efforts were made to place the cluster distally on the thigh to minimize soft-tissue volume under the cluster. However, particularly for participants who were able to achieve highly flexed squat postures, the bulk of the thigh may deform and compress. Anecdotally, this compression appears to cause some rotation and a lateral shift of the cluster with respect to the thigh. The potential for cluster movement generated some concern that the pre- to post-kneeling differences in frontal plane knee motion may have been caused simply because the participant was able to achieve a more highly flexed knee posture following the kneeling exposure. Greater knee flexion would theoretically result in greater deformation of soft-tissue and subsequent erroneous interpretation of an increase in the deviation of the knee joint center from the plane, which was not caused by a change in movement pattern but simply an increase in the deformation of the thigh soft tissue.

Using participant S16 as an example, it is clear that while not impossible, this is not likely always the case. Participant S16 achieved a nearly identical peak knee flexion angle at all three measurement times (Figure 6.2), yet the peak and mean deviation of the knee joint center from the body-fixed plane increased following the kneeling exposure (Figure 5.23). This would imply that the increase in the knee joint center deviation was truly due to a change in movement pattern and not a change in soft-tissue deformation. Similarly, participant S11 demonstrated an ~10° increase in peak knee flexion when squatting slowly between pre- and post-kneeling measurements; however, little to no change was found between those time points when analyzing the frontal plane knee motion curve (see S11 in Appendix K: Tri-Axial Knee Joint Angles and Knee Deviation during Squat Transitions). Therefore, while the limitations of motion capture

markers attached to soft-tissue is a concern, it does not appear to have masked the effects of the kneeling exposure on frontal plane knee motion.

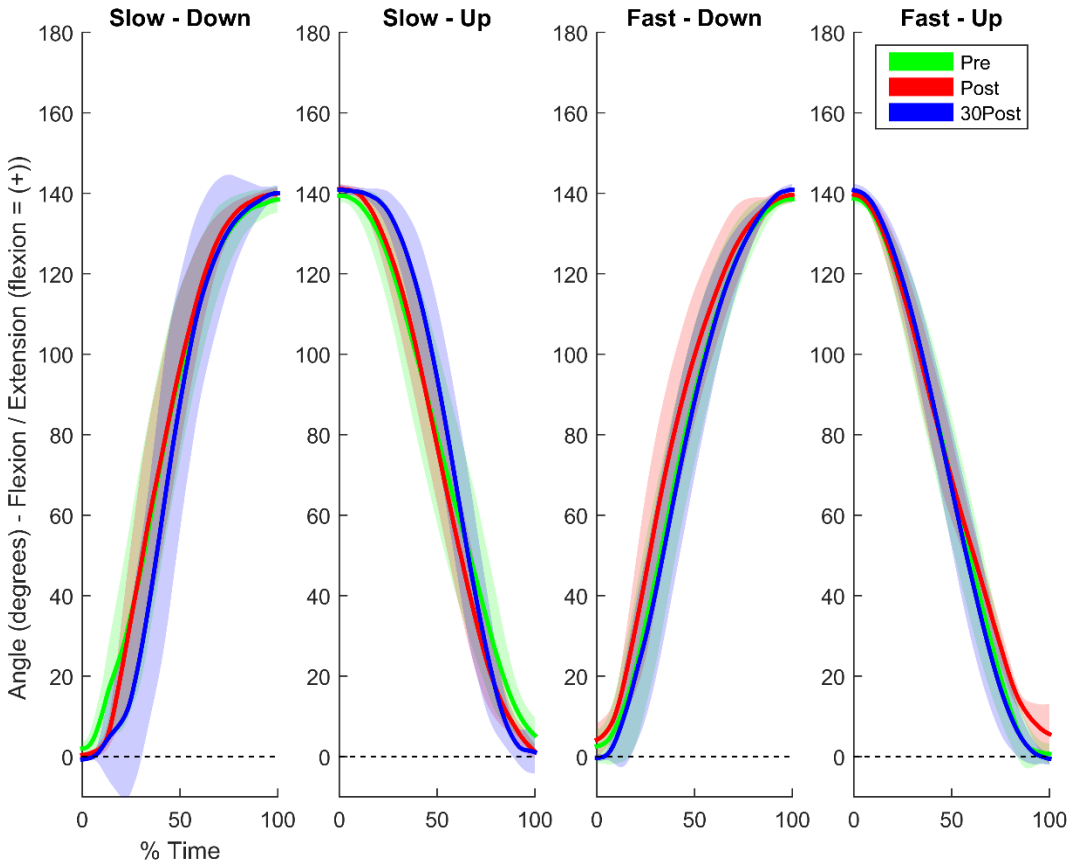


Figure 6.1 Knee flexion angle for S16 during fast and slow squats.
Data are normalized to 101 data points between the start and end of each direction of movement (ascent and descent). Shaded error bars indicate ± 1 SD.

Knee Proprioception

It was hypothesized that the kneeling exposure would cause a deficit in proprioceptive acuity at the knee; however, no significant effects of the kneeling exposure were found for the absolute angular error during the knee angle reproduction task. This lack of difference suggests that proprioceptive deficits may not contribute to the mechanism of knee osteoarthritis

development in occupational kneelers. However, as noted previously, some participants did show a decrease in proprioceptive acuity following kneeling (Figure 5.7). This finding might imply that although the majority of participants were not affected by the kneeling exposure, some individuals may have experienced muscle stretch or ligamentous creep sufficient to alter knee joint proprioception. It should be noted that for participants who showed proprioceptive deficits post-kneeling, no clear association was evident between the deficit and any demographic variables, or negative adaptations in other outcome measures. In fact, five out of the seven participants who showed a deficit in knee joint position sense actually showed less frontal plane knee motion during slow squat transitions. This lack of association between deficits in knee joint proprioception and negative changes in motor control suggests that proprioception is not likely the main contributing mechanism for any negative adaptations observed in the current study (Table 6.1).

Based on the lack of change in knee joint position sense, this study does not support a link between impaired proprioception and neuromuscular control changes. However, that is not to say that a proprioceptive deficit is not the pathway for the observed changes found in the previous study (Kajaks & Costigan, 2015). First, since Kajaks' project did not quantify knee joint proprioception, it is not possible to rule out the possibility that proprioceptive deficits contributed to the changes in neuromuscular control during gait seen in that particular study. Second, in the current study, the exposure did not elicit a biologically significant change in the kinematics and kinetics of gait. Corresponding to this finding, no changes in proprioception were found when testing the knee at a flexion angle of 20°, which was selected to evaluate joint position sense at a knee flexion angle that approaches those seen at initial contact of gait (Riskowski et al., 2005; Winter, 1991), and therefore should be most sensitive to changes in knee

proprioception relevant to altered loading profiles during the impact phase of gait (i.e. the peak rate of loading, peak knee adduction moment). At the same time, the exposure *was* sufficient to elicit a significant increase in both the peak and mean knee joint deviation during the squat task (Figure 5.18, Figure 5.20). However, knee proprioception was not tested in a weight-bearing posture, nor at positions of greater knee joint flexion required in the squat task. Although not all authors agree (Torres, Vasques, Duarte, & Cabri, 2010), studies have found that knee joint position sense may differ, or may be differentially affected by interventions, depending on the knee angle evaluated (Bennell et al., 2005; Erden, 2009; Ghaffarinejad et al., 2007). Different findings at different knee angles may be a reasonable outcome due to the different proprioceptive inputs available at the end- and mid-joint ranges of motion (Hogervorst & Brand, 1998; Rothwell, 1994). Therefore, it is possible that proprioceptive acuity was impaired in the current study, but within the untested range, and coincidentally, within the range of motion that is required when performing a high-flexion squat where effects of kneeling were observed.

A main effect of trial was found when measuring knee proprioception (Figure 5.5). The first trial was less accurate than both of the following trials. Although participants were given thorough instructions about the task and at least three practice trials were performed, participants were asked to replicate the same posture three times in succession. Therefore, it is possible that a practice effect was involved when knee proprioception was evaluated at each time point. Only three trials were collected due to time constraints and the interest in being able to capture any effects of the kneeling exposure in either gait or squat performance that may disappear with time. In future studies, it may be advisable to take additional trials (e.g. 20 – 30, or more) at baseline to ensure the measured value is a true representation of the participant's baseline proprioceptive acuity, thus improving the ability to detect changes.

A difference in proprioception was also found between sexes such that females had a higher absolute angular error (AAE) compared to males, ignoring the effects of the kneeling exposure (Figure 5.6). In a study of proprioception in collegiate athletes, researchers also found a significant difference between males and females, where males had greater reduced thresholds to the detection of motion, when measuring proprioception as a threshold to detection of passive motion during knee extension (Rozzi et al., 1999). Two separate studies on healthy participants (athleticism not specified), one which analyzed proprioception in the upper limb, and a second which analyzed a knee angle reproduction task, found that women demonstrated greater absolute errors; however, the differences were not statistically significant (Birmingham et al., 1998; Björklund, Crenshaw, Djupsjöbacka, & Johansson, 2003). In another study, the authors did not report statistics for the sex comparison, but males tended to have a lower AAE compared to females when evaluating knee joint position sense (males: 1.90° (0.94°) vs. females: 3.13° (1.46°)) (Gear, 2011).

The values in the current study for AAE were within the ranges of values reported in the literature in studies that used similar methods and similar participant groups (Table 6.2). Although the mean AAE in the current study was within previously recorded ranges, the standard deviation was large (2.1°), with a very high maximum AAE value (9.1°). It is possible that the values for AAE found in the current study were on the higher end of the reported range due to the fact that participant activity levels varied widely, from inactive (1 day/week), to very active (7 days/week). In contrast, many studies evaluate only athletes/non-athletes exclusively. It has been suggested that athletes' comparatively enhanced motor performance compared to non-athletes may contribute to more accurate repositioning of the joint (Muaidi, Nicholson, & Refshauge, 2009; Naseri & Pourkazemi, 2012). Although the difference did not achieve

statistical significance (1-tail T-test assuming unequal variances, p = .08, (Excel 2013, Microsoft, Redmond, WA, USA), the results from the current study suggest a trend towards greater proprioceptive acuity in more active individuals. When participants were grouped based on self-reported activity, those who were active more than three days per week tended to have a smaller AAE at baseline compared to participants who were only active three or fewer days per week (Figure 6.1).

Table 6.3

Participants who demonstrated a Deficit in Knee Proprioception of ≥ 1° Post-Kneeling, contrasted with Outcome Variables in Gait and Squat Transitions Measured Post-Kneeling.

		Participant						
		S13	**S14**	**S18**	**S24**	**S29**	**S33**	**S45**
Sex		male	male	female	female	female	female	Male
Activity (self-reported days/week)		3	3	6	4	3	4	6
Ely's Angle (°)		52	45	76	40	57	68	40
Average AAE (°)		1.2	2.6	1.5	1.8	1.9	1.7	3.6
Peak KAM (% BW*H)	Unloaded	0.27	-0.36	0.32	0.08	0.08	0.13	-0.29
	Loaded	0.09	0.11	0.61	0.13	-0.26	0.32	-0.16
Peak ROL (BW/s)	Unloaded	-0.085	-0.581	0.367	-2.277	0.287	-1.618	1.726
	Loaded	1.434	0.115	-0.163	-0.431	-1.932	0.936	-0.762
VM Onset (ms)	Loaded	-19	2	17	19	2	12	23
	Unloaded	30	17	-4	23	12	-5	-24
VM Magnitude	Loaded	- 490	138	2003	-30	112	168	104
	Unloaded	2518	-363	256	112	285	-220	83
Peak Knee Deviation (m)	Slow – descent	0.001	0.000	-0.027	-0.091	-0.084	-0.010	-0.010
	Slow – ascent	0.000	0.001	-0.028	-0.135	-0.093	-0.005	-0.016
	Fast – descent	0.001	0.001	-0.018	0.005	-0.049	0.024	0.060
	Fast – ascent	0.001	0.000	-0.019	0.041	-0.091	0.032	0.065
Mean Knee Deviation (m)	Slow – descent	-0.041	0.025	-0.057	-0.014	-0.047	-0.003	0.003
	Slow – ascent	-0.022	-0.010	0.006	-0.025	-0.039	0.000	-0.004
	Fast – descent	-0.015	-0.010	-0.030	-0.005	-0.016	0.012	0.047
	Fast – ascent	-0.034	0.005	-0.026	0.006	-0.030	0.016	0.030

Underlined values indicate what might be considered a biologically significant change (≥ 0.5 %BW*H, ≥ 1 cm, ≥ 15 ms, ≥ 1 BW/s). No significance estimated for VM magnitude changes. NOTE: (-) values are considered a positive adaptation, whereas (+) values indicate a negative adaptation (changes in performance that are less desirable), which the exception of VM magnitude where a decrease in activation magnitude (+ change between pre and post) would be considered a negative adaptation.

Table 6.4

Knee Proprioception: Comparison of Average Angular Error Results across Studies

Author, year	Participant Demographics	Testing Angle (°)	AAE (°) ± SD (range)
Current Study Results (baseline)	20F, 20 M, 21.4 ± 2.48 Y	20	2.7 ± 2.1 (0.014 - 9.1)
Baker et al., 2002	15 F, 5 M, 25.5 ± 8.6 Y - Estimated daily physical activity 3158 ± 1516 kcal	20	1.3 ± 0.5 (0.6 - 2.6)
Daneshjoo, Mokhtar, Rahnama, & Yusof, 2012	36 M, 17-20 - Professional soccer players	30	5.4 ± 3.5
Gear, 2011	10 F, 8 M, 19.5 ± 1.2 Y - All were NCAA Division III basketball or soccer athletes	15	2.58 ± 1.38
Ghaffarinejad et al., 2007	21 F, 18 M, 25.6 ± 1.2 Y - All performed ≥ moderate physical activity ≥ 3x/week	20	1.1 ± 1.2
Han & Lee, 2014	15 F, 21.47 ± 0.52 Y 15 M, 21.87 ± 0.83 Y	30, 40, 60	3.41 ± 1.42
Hosp et al., 2015	12 F, 23.6 ± 2 Y - Non-professional athletes	20 - 70	4.4 ± 1.4
Naseri & Pourkazemi, 2012	10 F, 10 M, 24.9 ± 5.6 - 'vigorously' active	20	4.9 ± 1.9

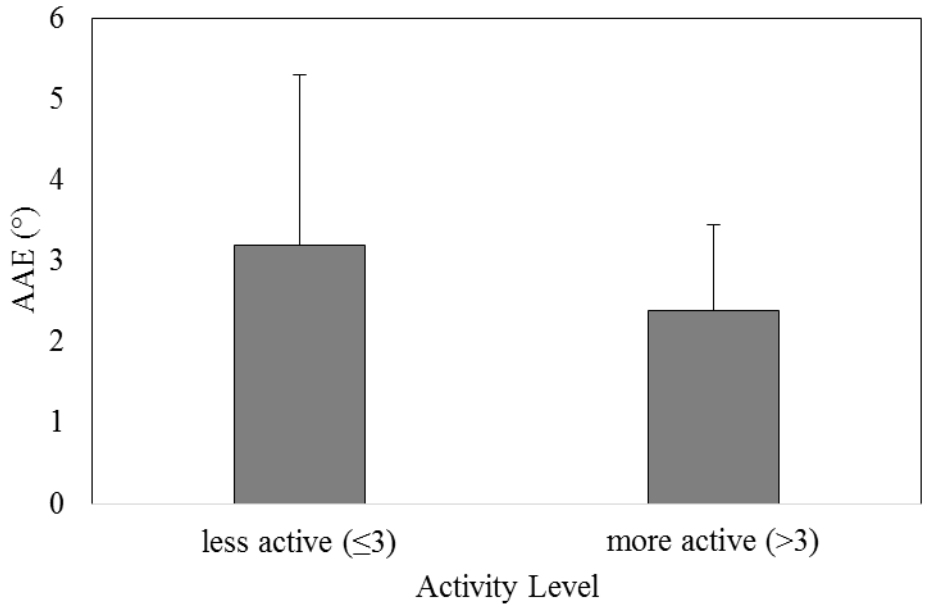


Figure 6.2 Comparing the average angular error during the knee angle replication task between participants based on self-reported activity levels.
Note: n = 17 for 'less active' and n = 23 for 'more active.' Error bars indicate + 1 SD.

Summary

The kneeling exposure in the current study did not induce biologically meaningful changes when group mean effects were analyzed for measures of knee joint proprioception and gait mechanics. However, when performing squat transitions, the kneeling exposure caused an increase in frontal plane knee motion. This increase in knee joint deviation suggests a negative adaptation in neuromuscular control that may or may not directly influence knee osteoarthritis risk. The lack of change in gait mechanics is in contrast to previous work, which examined the effects of a static kneeling exposure on gait (Kajaks, 2008; Kajaks & Costigan, 2015). It is likely that the difference in findings is at least partially due to the fact that in the current study, the kneeling exposure was designed to be better representative of an occupational exposure and hence included periods of both single-arm supported and full-flexion kneeling. At the same time, select participants did in fact demonstrate negative adaptations to the kneeling exposure for measures of knee proprioception and loading in gait. Because the more dynamic exposure in the

current study did not elicit changes in gait, if time spent in occupational kneeling postures truly alters gait kinetics and kinematics as some evidence suggests (Gaudreault et al., 2013; Kajaks & Costigan, 2015), then perhaps the changes occur specifically in correlation with time spent in full-flexion kneeling. Further research is necessary before making specific recommendations, either on workplace exposures or on movement techniques. It may be recommended that workers try to incorporate posture cycling (i.e. full-flexion and single-arm supported kneeling) when possible, as it appears that this type of exposure does not, for most individuals, alter gait – at least in the variables measured in the current study that have been associated with an increased risk of the development of knee osteoarthritis.

When performing squat transitions, the kneeling exposure caused an increase in frontal plane knee motion. This increase suggests a negative adaptation in neuromuscular control that may or may not directly influence knee osteoarthritis risk. These changes persisted for 30 minutes following the exposure. To help mitigate any increase in injury risk resulting from changes in squat control, workers should avoid transferring heavy loads to or from the floor using a squat technique, as external loads will increase the forces placed on the knee. For example, when the worksite permits, workers should consider transferring heavy loads to a cart or trolley to move materials, and lighter loads (e.g. individual tiles) can be brought down to the floor as needed. In addition, although squat transitions performed at a fast pace reduced the mean deviation of the knee, moving more quickly, generally, can place higher loads on the body and increase risk of injury, and thus such an approach should not be encouraged. The findings of this study cannot establish whether proprioception is a potential mechanism for knee osteoarthritis risk among occupational kneelers; however, it has highlighted potential additional risk in squat transitions that had not been previously identified.

7.0 Limitations

This project has a number of limitations. As previously discussed in detail, the efficacy of measuring proprioception using the currently available methods is limited (3.2.2). In addition, there are not only differences between joint position sense and movement sense, but also between weight-bearing and non-weight-bearing tests, as well as within different ranges of joint motion. The validity of measuring proprioception based on conscious perception has also been questioned. In the current study, proprioception was measured as joint position sense at a single knee joint angle. Therefore, the ability to comment on knee proprioception as a potential mechanism causing changes in the neuromuscular control of a squat transition is limited, as this task involves a large knee joint range of motion and is a weight-bearing activity.

In addition, although not all occupational kneelers wear work boots, some of the most commonly studied populations, including miners, floor layers, and tile setters, may wear safety footwear while kneeling. Work boots have been shown to alter the location of center of pressure at the knee compared to barefoot kneeling (Tennant, Kingston, Chong, & Acker, 2015), as well as the muscle demand while walking (Dobson, Riddiford-Harland, & Steele, 2014). Participants did not wear work boots in this study; therefore, the effects of safety footwear on the measured outcomes cannot be addressed. Other concerns related to footwear include the fact that although all participants wore comfortable, traditional running shoes, the style and wear condition (e.g. new vs. old shoes, frequently vs. infrequently used) of the participants' footwear was not controlled for. Therefore, the effects of different shoe construction on the vertical loading profile and heel-strike transient prevalence may be confounding factors. At the same time, it was important that participants were shod for data collections because loading at initial contact is

different in barefoot compared to shod gait (Fong Yan, Sinclair, Hiller, Wegener, & Smith, 2013), and workers will always wear some form of footwear.

Although not all kneeling occupations require lifting while in a kneeling position, many do. The card sorting simulation task did not incorporate a load component and therefore, while likely more occupationally relevant than a static kneeling exposure, it is still not likely a true a representation of the demands of heavy kneeling work. In addition, as briefly addressed in the discussion, the current motion capture methodology is susceptible to the effects of soft tissue deformation, which can be problematic in postures requiring high knee flexion, such as a full-flexion squat. Along a similar line of thought, although this finding has been contested (Teichtahl, Wluka, Morris, Davis, & Cicuttini, 2009), joint moment asymmetry has been found among healthy young individuals, including in the external knee adduction moment (Lathrop-Lambach et al., 2014). In the current study, data was only collected from the dominant leg. Therefore, although it seems unlikely that one leg would have been affected by the kneeling exposure differently based on the efforts made to ensure an equal exposure, it is possible that by not collecting data from both lower limbs, changes were missed.

Furthermore, it was not possible to directly measure the forces inside the knee. All of the measures used in this project were an estimation of load inside the knee that relied on force data collected from the force plates. This data does not measure the force generated in the joint or at the articular surface. Therefore, even though an individual demonstrates high rates of loading, heel-strike transients, or high external knee adduction moments, it is possible that the individual utilizes cushioning mechanisms or exhibits natural dampening that protects the knee joint from excessive loads. In an attempt to give some insight into these control strategies, vastus medialis activation was measured, with delayed or decreased magnitude of pre-activation potentially

indicating that neuromuscular control and force attenuation mechanisms were not optimal. However, although research suggests that the quadriceps group is the main active mechanism of force attenuation, many muscles of the lower limb contribute, whether actively or passively (Wright, Neptune, Van Den Bogert, & Nigg, 1998). Therefore, these measures, while informative, are still unable to address the inability to directly measure loading inside the knee joint.

In terms of study design, limitations of this study include the fact that it is not longitudinal in nature. Therefore, it is possible that a single, simulated occupational kneeling exposure is inadequate to effect changes in neuromuscular control. Although one author observed differences after a half-hour exposure (Kajaks & Costigan, 2015), the results of the current study suggest that an occupational kneeling exposure over the course of a number of days, weeks, or even months is required to observe a change.

8.0 Contributions and Future Directions

This project has made significant contributions to the understanding of occupational kneeling and the effects of a high knee flexion exposure on gait, squat transitions, and knee joint proprioception.

1. **A 30-minute dynamic kneeling exposure elicits no biologically significant effects on the gait measures of neuromuscular control (and thus knee OA risk) evaluated in the current study.** This finding is in contrast to earlier work that did measure significant effects of a static full-flexion kneeling exposure on gait kinetics (Kajaks & Costigan, 2015); albeit, the kneeling exposure examined was likely less occupationally relevant than the dynamic exposure in the current study. Therefore, to reduce the effects of prolonged kneeling on maladaptation in gait, cyclic movement should be encouraged, and spending prolonged time in a static, full-flexion kneeling posture should be avoided. Future research should investigate how long workers can safely kneel while avoiding inducing alterations in gait kinetics found in previous work. Based on the findings that a full-flexion kneeling exposure induces changes, but a dynamic one does not, this line of inquiry will likely require investigation into the creep-recovery response of knee joint ligaments based on physiological loads on these structures in high-flexion.
2. **Knee joint deviation during a squat transition to-and-from the floor increases following a 30-minute dynamic kneeling exposure.** This is the first study to investigate neuromuscular control during squat transitions following a simulated occupational kneeling exposure. The observed increase in frontal plane knee motion is considered a negative adaptation that could increase the risk of injury due to non-neutral positioning in a weight-bearing posture. The current study highlights the analysis of squat transitions as a more

sensitive measure to change following a kneeling exposure, compared to gait, and should be considered in future research on the effects of kneeling. In the future, researchers may consider investigating the effects of the frequency of different transitions to-and-from the floor, and the impact of such transitions on knee joint health and knee OA risk.

3. **There are no changes in knee joint position sense after a 30-minute dynamic kneeling exposure.** This was the first study to investigate proprioception at the knee following a simulated occupational kneeling exposure. It was found that knee joint proprioception (measured as joint position sense) at 20° of knee flexion was not affected by the kneeling exposure. This does not mean that changes in knee joint proprioception do not mediate occupation-related knee OA. Although the current study only evaluated healthy young people following a short kneeling exposure, a small number of participants did exhibit a deficit. Therefore, the findings suggest that if changes in knee proprioception contribute to knee OA risk, the changes may not occur without longer exposures to a full-flexion kneeling posture. Because knee joint laxity has also been proposed as a potential mechanism linking kneeling exposures to altered gait, a better understanding of the viscoelastic properties of the cruciate ligaments, including an evaluation of ligamentous creep in high knee flexion loading conditions, is needed in order to determine whether kneeling or other high knee flexion postures truly induce ligamentous laxity as expected. Future studies might also consider examining knee joint proprioception in individuals who adopt high knee flexion postures such as kneeling and squatting occupationally, both early in their careers, and later as workers age, to determine whether joint proprioception is impaired compared to healthy controls who do not kneel or assume high knee flexion postures in their occupation. In addition, other methods of measuring neuromuscular response in a fundamental way may be

beneficial to better identify potential pathways of change. For example, an evaluation of the quadriceps muscle reflex response to measure the effect of cruciate ligamentous creep on neuromuscular control following a kneeling exposure could be investigated.

It should also be noted that, anecdotally, participants sometimes complained of shoulder, wrist, and lower back discomfort during the simulated occupational kneeling exposure. While disorders of the back have been explored with respect to kneeling postures similar to those investigated in the current study, less data is available on the its effects on the shoulder and wrist, so this may be an area for future work.

References

Alexander, R. M. (1991). Energy-saving mechanisms in walking and running. *Journal of Experimental Biology, 160,* 55-69.

Almosnino, S., Kingston, D., & Graham, R. B. (2013). Three-dimensional knee joint moments during performance of the bodyweight squat: Effects of stance width and foot rotation. *Journal of Applied Biomechanics, 29*(1), 33-43.

Amin, S., Goggins, J., Niu, J., Guermazi, A., Grigoryan, M., Hunter, D. J., . . . Felson, D. T. (2008). Occupation-related squatting, kneeling, and heavy lifting and the knee joint: A magnetic resonance imaging-based study in men. *Journal of Rheumatology, 35*(8), 1645-1649.

Anderson, D. D., Brown, T. D., & Radin, E. L. (1993). The influence of basal cartilage calcification on dynamic juxtaarticular stress transmission. *Clinical Orthopaedics and Related Research,* (286), 298-307.

Anderson, J. J., & Felson, D. T. (1988). Factors associated with osteoarthritis of the knee in the first national health and nutrition examination survey (HANES I). *American Journal of Epidemiology, 128*(1), 179-189.

Andrews, S., Shrive, N., & Ronsky, J. (2011). The shocking truth about meniscus. *Journal of Biomechanics, 44*(16), 2737-2740.

Andriacchi, T. P., Ogle, J. A., & Galante, J. O. (1977). Walking speed as a basis for normal and abnormal gait measurements. *Journal of Biomechanics, 10*(4), 261-268.

Arden, N., & Nevitt, M. C. (2006). Osteoarthritis: Epidemiology. *Best Practice and Research: Clinical Rheumatology, 20*(1), 3-25.

Badlani, J., Borrero, C., Golla, S., Harner, C., & Irrgang, J. (2013). The effects of meniscus injury on the development of knee osteoarthritis: Data from the osteoarthritis initiative. *The American Journal of Sports Medicine, 41*(6), 1238-1244. doi:10.1177/0363546513490276

Baert, I. A. C., Mahmoudian, A., Nieuwenhuys, A., Jonkers, I., Staes, F., Luyten, F. P., . . . Verschueren, S. M. P. (2013). Proprioceptive accuracy in women with early and established knee osteoarthritis and its relation to functional ability, postural control, and muscle strength. *Clinical Rheumatology, 32*(9), 1365-1374.

Baker, V., Bennell, K., Stillman, B., Cowan, S., & Crossley, K. (2002). Abnormal knee joint position sense in individuals with patellofemoral pain syndrome. *Journal of Orthopaedic Research, 20*(2), 208-214.

Baliunas, A. J., Hurwitz, D. E., Ryals, A. B., Karrar, A., Case, J. P., Block, J. A., & Andriacchi, T. P. (2002). Increased knee joint loads during walking are present in subjects with knee osteoarthritis. *Osteoarthritis and Cartilage, 10*(7), 573-579.

Barrack, R. L., Skinner, H. B., Brunet, M. E., & Cook, S. D. (1984). Joint kinesthesia in the highly trained knee. *Journal of Sports Medicine and Physical Fitness, 24*(1), 18-20.

Barrack, R. L., Skinner, H. B., Cook, S. D., & Haddad Jr., R. H. (1983). Effect of articular disease and total knee arthroplasty on knee joint-position sense. *Journal of Neurophysiology, 50*(3), 684-687.

Barrios, J. A., & Strotman, D. E. (2014). A sex comparison of ambulatory mechanics relevant to osteoarthritis in individuals with and without asymptomatic varus knee alignment. *Journal of Applied Biomechanics, 30*(5), 632-636. doi:10.1123/JAB.2014-0039

Baumgart, C., Gokeler, A., Donath, L., Hoppe, M. W., & Freiwald, J. (2015). Effects of static stretching and playing soccer on knee laxity. *Clinical Journal of Sport Medicine, 25*(6), 541-545.

Bayramoglu, M., Toprak, R., & Sozay, S. (2007). Effects of osteoarthritis and fatigue on proprioception of the knee joint. *Archives of Physical Medicine and Rehabilitation, 88*(3), 346-350.

Begon, M., Monnet, T., & Lacouture, P. (2007). Effects of movement for estimating the hip joint centre. *Gait and Posture, 25*(3), 353-359.

Belvedere, C., Ensini, A., Feliciangeli, A., Cenni, F., D'Angeli, V., Giannini, S., & Leardini, A. (2012). Geometrical changes of knee ligaments and patellar tendon during passive flexion. *Journal of Biomechanics, 45*(11), 1886-1892. doi:10.1016/j.jbiomech.2012.05.029

Ben, M., & Harvey, L. A. (2010). Regular stretch does not increase muscle extensibility: A randomized controlled trial. *Scandinavian Journal of Medicine and Science in Sports, 20*(1), 136-144.

Bennell, K. L., Hunt, M. A., Wrigley, T. V., Lim, B., & Hinman, R. S. (2008). Role of muscle in the genesis and management of knee osteoarthritis. *Rheumatic Diseases Clinics of North America, 34*(3), 731-754. doi:10.1016/j.rdc.2008.05.005

Bennell, K., Hinman, R. S., Metcalf, B. R., Crossley, K. M., Buchbinder, R., Smith, M., & McColl, G. (2003). Relationship of knee joint proprioception to pain and disability in individuals with knee osteoarthritis. *Journal of Orthopaedic Research, 21*(5), 792-797.

Bennell, K., Hinman, R. S., Wrigley, T. V., Creaby, M. W., & Hodges, P. (2011). Exercise and osteoarthritis: Cause and effects. *Comprehensive Physiology, 1*(4), 1943-2008.

Bennell, K., Wee, E., Crossley, K., Stillman, B., & Hodges, P. (2005). Effects of experimentally-induced anterior knee pain on knee joint position sense in healthy individuals. *Journal of Orthopaedic Research, 23*(1), 46-53.

Bennell, K., Wrigley, T. V., Hunt, M. A., Lim, B. -., & Hinman, R. S. (2013). Update on the role of muscle in the genesis and management of knee osteoarthritis. *Rheumatic Disease Clinics of North America, 39*(1), 145-176.

Beynnon, B. D., Renstrom, P. A., Konradsen, L., Elmqvist, L., Gottlieb, D., & Dirks, M. (2000). Chapter 12: Validation of techniques to measure knee proprioception. In S. M. Lephart, & F. H. Fu (Eds.), *Proprioception and neuromuscular control in joint stability* (pp. 127-144) Human Kinetics.

Birmingham, T. B., Hunt, M. A., Jones, I. C., Jenkyn, T. R., & Giffin, J. R. (2007). Test-retest reliability of the peak knee adduction moment during walking in patients with medial compartment knee osteoarthritis. *Arthritis Care and Research, 57*(6), 1012-1017.

Birmingham, T. B., Kramer, J. F., Kirkley, A., Inglis, J. T., Spaulding, S. J., & Vandervoort, A. A. (2001). Association among neuromuscular and anatomic measures for patients with knee osteoarthritis. *Archives of Physical Medicine and Rehabilitation, 82*(8), 1115-1118.

Birmingham, T., Kramer, J., Inglis, J., Mooney, C., Murray, L., Fowler, P., & Kirkley, S. (1998). Effect of a neoprene sleeve on knee joint position sense during sitting open kinetic chain and supine closed kinetic chain tests. *The American Journal of Sports Medicine, 26*(4), 562-566. doi:10.1177/03635465980260041601

Björklund, M., Crenshaw, A. G., Djupsjöbacka, M., & Johansson, H. (2000). Position sense acuity is diminished following repetitive low-intensity work to fatigue in a simulated occupational setting. *European Journal of Applied Physiology, 81*(5), 361-367.

Björklund, M., Crenshaw, A. G., Djupsjöbacka, M., & Johansson, H. (2003). Position sense acuity is diminished following repetitive low-intensity work to fatigue in a simulated occupational setting. A critical comment. *European Journal of Applied Physiology, 88*(4-5), 485-486.

Björklund, M., Hamberg, J., & Crenshaw, A. G. (2001). Sensory adaptation after a 2-week stretching regimen of the rectus femoris muscle. *Archives of Physical Medicine and Rehabilitation, 82*(9), 1245-1250.

Bohannon, R. W. (1997). Comfortable and maximum walking speed of adults aged 20-79 years: Reference values and determinants. *Age and Ageing, 26*(1), 15-19.

Brandon, S. C. E., & Deluzio, K. J. (2011). Robust features of knee osteoarthritis in joint moments are independent of reference frame selection. *Clinical Biomechanics, 26*(1), 65-70. doi:10.1016/j.clinbiomech.2010.08.010

Brandt, K. D., Dieppe, P., & Radin, E. (2009). Etiopathogenesis of osteoarthritis. *Medical Clinics of North America, 93*(1), 1-24.

Brandt, K. D., Dieppe, P., & Radin, E. L. (2008). Etiopathogenesis of osteoarthritis. *Rheumatic Disease Clinics of North America, 34*(3), 531-559.

Brandt, K. D., & Fife, R. S. (1986). Ageing in relation to the pathogenesis of osteoarthritis. *Clinics in Rheumatic Diseases, 12*(1), 117-130.

Buckwalter, J. A., & Mankin, H. J. (1997). Articular cartilage. part II: Degeneration and osteoarthrosis, repair, regeneration, and transplantation. *Journal of Bone and Joint Surgery - Series A, 79*(4), 612-632.

Bullock-Saxton, J. E., Wong, W. J., & Hogan, N. (2001). The influence of age on weight-bearing joint reposition sense of the knee. *Experimental Brain Research, 136*(3), 400-406.

Calder, K. M., Acker, S. M., Arora, N., Beattie, K. A., Callaghan, J. P., Adachi, J. D., & Maly, M. R. (2014). Knee power is an important parameter in understanding medial knee joint load in knee osteoarthritis. *Arthritis Care and Research, 66*(5), 687-694.

Carter N.D, Jenkinson, T. R., Wilson, D., Jones, D. W., & Torode, A. S. (1997). Joint position sense and rehabilitation in the anterior cruciate ligament deficient knee. *British Journal of Sports Medicine, 31*(3), 209-212.

Chaganti, R. K., Tolstykh, I., Javaid, M. K., Neogi, T., Torner, J., Curtis, J., et al. (2014). High plasma levels of vitamin C and E are associated with incident radiographic knee osteoarthritis. *Osteoarthritis and Cartilage, 22*(2), 190-196.

Chang, A. H., Lee, S. J., Zhao, H., Ren, Y., & Zhang, L. -. (2014). Impaired varus-valgus proprioception and neuromuscular stabilization in medial knee osteoarthritis. *Journal of Biomechanics, 47*(2), 360-366.

Chang, C. F., Shih, K. S., Wang, T. M., Huang, C. H., Huang, S. C., & Lu, T. W. (2012). Correlations between heelstrike impulsive loading and joint kinematics of the lower extremities during normal level walking. *Journal of Mechanics, 28*(2), 337-344.

Cheng, X., Zhang, T., Shan, X., & Wang, J. (2014). Effect of posterior cruciate ligament creep on muscular co-activation around knee: A pilot study. *Journal of Electromyography and Kinesiology, 24*(2), 2710-2276. doi:10.1016/j.jelekin.2014.01.005

Cho, Y. J., & Kim, J. Y. (2012). The effects of load, flexion, twisting and window size on the stationarity of trunk muscle EMG signals. *International Journal of Industrial Ergonomics, 42*(3), 287-292. doi:10.1016/j.ergon.2012.02.004

Chokkhanchitchai, S., Tangarunsanti, T., Jaovisidha, S., Nantiruj, K., & Janwityanujit, S. (2010). The effect of religious practice on the prevalence of knee osteoarthritis. *Clinical Rheumatology, 29*(1), 39-44.

Cholewicki, J., & VanVliet Iv, J. J. (2002). Relative contribution of trunk muscles to the stability of the lumbar spine during isometric exertions. *Clinical Biomechanics, 17*(2), 99-105. doi:10.1016/S0268-0033(01)00118-8

Chu, D., LeBlanc, R., D'Ambrosia, P., D'Ambrosia, R., Baratta, R. V., & Solomonow, M. (2003). Neuromuscular disorder in response to anterior cruciate ligament creep. *Clinical Biomechanics, 18*(3), 222-230.

Cicuttini, F. M., Forbes, A., Yuanyuan, W., Rush, G., & Stuckey, S. L. (2002). Rate of knee cartilage loss after partial meniscectomy. *Journal of Rheumatology, 29*(9), 1954-1956.

Claes, S., Vereecke, E., Maes, M., Victor, J., Verdonk, P., & Bellemans, J. (2013). Anatomy of the anterolateral ligament of the knee. *Journal of Anatomy, 223*(4), 321-328.

Coggon, D., Croft, P., Kellingray, S., Barrett, D., McLaren, M., & Cooper, C. (2000). Occupational physical activities and osteoarthritis of the knee. *Arthritis & Rheumatism, 43*(7), 1443-1449. doi:10.1002/1529-0131(200007)43:73.0.CO;2-1

Collins, J. J., & Whittle, M. W. (1989). Impulsive forces during walking and their clinical implications. *Clinical Biomechanics, 4*(3), 179-187.

Cook, T. M., & Neumann, D. A. (1987). The effects of load placement on the EMG activity of the low back muscles during load carrying by men and women. *Ergonomics, 30*(10), 1413-1423.

Cooper, C., McAlindon, T., Coggon, D., Egger, P., & Dieppe, P. (1994). Occupational activity and osteoarthritis of the knee. *Annals of the Rheumatic Diseases, 53*(2), 90-93. doi:10.1136/ard.53.2.90

Courville, A., Sbriccoli, P., Zhou, B. -., Solomonow, M., Lu, Y., & Burger, E. L. (2005). Short rest periods after static lumbar flexion are a risk factor for cumulative low back disorder. *Journal of Electromyography and Kinesiology, 15*(1), 37-52. doi:10.1016/j.jelekin.2004.06.005

Canadian Standards Association (2012). *Z1004-12 - Workplace ergonomics: A management and implementation standard* (1st ed.). PDF: CSA.

Dahlkvist, N. J., Mayo, P., & Seedhom, B. B. (1982). Forces during squatting and rising from a deep squat. *Engineering in Medicine, 11*(2), 69-76.

Daneshjoo, A., Mokhtar, A. H., Rahnama, N., & Yusof, A. (2012). The effects of comprehensive warm-up programs on proprioception, static and dynamic balance on male soccer players. *PLoS ONE, 7*(12) doi:10.1371/journal.pone.0051568

De Luca, C. J. (1997). The use of surface electromyography in biomechanics. *Journal of Applied Biomechanics, 13*(2), 135-163.

Dieppe, P. A., & Kirwan, J. (1994). The localization of osteoarthritis. *British Journal of Rheumatology, 33*(3), 201-203.

Dieppe, P. A., & Lohmander, L. S. (2005). Pathogenesis and management of pain in osteoarthritis. *Lancet, 365*(9463), 965-973.

Diracoglu, D., Baskent, A., Celik, A., Issever, H., & Aydin, R. (2008). Long-term effects of kinesthesia/balance and strengthening exercises on patients with knee osteoarthritis: A one-year follow-up study. *Journal of Back and Musculoskeletal Rehabilitation, 21*(4), 253-262.

Dobson, J. A., Riddiford-Harland, D. L., & Steele, J. R. (2014). Effects of wearing gumboots and leather lace-up boots on gait and perceived comfort when walking on simulated underground coal mine surfaces. World Congress of Biomechanics, Boston 2014.

Dowling, A. V., Fisher, D. S., & Andriacchi, T. P. (2010). Gait modification via verbal instruction and an active feedback system to reduce peak knee adduction moment. *Journal of Biomechanical Engineering-Transactions of the Asme, 132*(7), 071007. doi:10.1115/1.4001584

Dufek, J. S., Bates, B. T., Stergiou, N., & James, C. R. (1995). Interactive effects between group and single-subject response patterns. *Human Movement Science, 14*(3), 301-323. doi:10.1016/0167-9457(95)00013-I

Englund, M., Guermazi, A., & Lohmander, S. L. (2009). The role of the meniscus in knee osteoarthritis: A cause or consequence? *Radiologic Clinics of North America, 47*(4), 703-712.

Englund, M., Guermazi, A., Roemer, F. W., Aliabadi, P., Yang, M., Lewis, C. E., . . . Felson, D. T. (2009). Meniscal tear in knees without surgery and the development of radiographic osteoarthritis among middle-aged and elderly persons: The multicenter osteoarthritis study. *Arthritis and Rheumatism, 60*(3), 831-839.

Erden, Z. (2009). Is there any difference in joint position sense among different knee angles? [Dizin farkli{dotless} açi{dotless}lari{dotless}nda eklem pozisyon hissi farkli{dotless} mi{dotless}di{dotless}r?] *Eklem Hastaliklari Ve Cerrahisi, 20*(1), 47-51.

Erden, Z., Otman, S., Atilla, B., & Tunay, V. B. (2003). Relationship between pain intensity and knee joint position sense in patients with severe osteoarthritis. *Pain Clinic, 15*(3), 293-297.

Ezzat, A. M., & Li, L. C. (2014). Occupational physical loading tasks and knee osteoarthritis: A review of the evidence. *Physiotherapy Canada, 66*(1), 91-107.

Felson, D. T. (1994). Do occupation-related physical factors contribute to arthritis? *Bailliere's Clinical Rheumatology, 8*(1), 63-77.

Felson, D. T. (2004a). Risk factors for osteoarthritis: Understanding joint vulnerability. *Clinical Orthopaedics and Related Research,* (427 SUPPL.), S16-S21.

Felson, D. T. (2004b). An update on the pathogenesis and epidemiology of osteoarthritis. *Radiologic Clinics of North America, 42*(1), 1-9.

Felson, D. T., Gross, K. D., Nevitt, M. C., Yang, M., Lane, N. E., Torner, J. C., . . . Hurley, M. V. (2009). The effects of impaired joint position sense on the development and progression of pain and structural damage in knee osteoarthritis. *Arthritis Care and Research, 61*(8), 1070-1076.

Felson, D. T., Lawrence, R. C., Dieppe, P. A., Hirsch, R., Helmick, C. G., Jordan, J. M., . . . Fries, J. F. (2000). Osteoarthritis: New insights - part 1: The disease and its risk factors. *Annals of Internal Medicine, 133*(8), 635-646.

Field, A., & Miles, J. (2010). Mixed design ANOVA (GLM 5). *Discovering statistics using SAS* (pp. 436-464) Sage.

Fitzgerald, G. K., Piva, S. R., & Irrgang, J. J. (2004). Reports of joint instability in knee osteoarthritis: Its prevalence and relationship to physical function. *Arthritis Care and Research, 51*(6), 941-946.

Fong Yan, A., Sinclair, P. J., Hiller, C., Wegener, C., & Smith, R. M. (2013). Impact attenuation during weight bearing activities in barefoot vs. shod conditions: A systematic review. *Gait and Posture, 38*(2), 175-186.

Frost, D. M. (2013). *Towards the establishment of a worker-centered framework to physically prepare firefighters: The evaluation of movement and the transfer of training* (Doctoral). Available from UWSpace.

Frost, D. M., Beach, T. A. C., Callaghan, J. P., & McGill, S. M. (2015). The influence of load and speed on individuals' movement behavior. *Journal of Strength and Conditioning Research, 29*(9), 2417-2425. doi:10.1519/JSC.0000000000000264

Frost, D. M., Beach, T. A. C., McGill, S. M., & Callaghan, J. P. (2015). A proposed method to detect kinematic differences between and within individuals. *Journal of Electromyography and Kinesiology, 25*(3), 479-487. doi:10.1016/j.jelekin.2015.02.012

Fuss, F. K. (1989). Anatomy of the cruciate ligaments and their function in extension and flexion of the human knee joint. *American Journal of Anatomy, 184*(2), 165-176.

Garsden, L. R., & Bullock-Saxton, J. E. (1999). Joint reposition sense in subjects with unilateral osteoarthritis of the knee. *Clinical Rehabilitation, 13*(2), 148-155.

Garstang, S. V., & Stitik, T. P. (2006). Osteoarthritis: Epidemiology, risk factors, and pathophysiology. *American Journal of Physical Medicine and Rehabilitation, 85*(11 SUPPL.), S2-S12.

Gaudreault, N., Hagemeister, N., Poitras, S., & de Guise, J. A. (2013). Comparison of knee gait kinematics of workers exposed to knee-straining posture to those of non-knee-straining workers. *Gait and Posture, 38*(2), 187-191.

Gear, W. S. (2011). Effect of different levels of localized muscle fatigue on knee position sense. *Journal of Sports Science and Medicine, 10*(4), 725-730.

Ghaffarinejad, F., Taghizadeh, S., & Mohammadi, F. (2007). Effect of static stretching of muscles surrounding the knee on knee joint position sense. *British Journal of Sports Medicine, 41*(10), 684-687.

Ghori, G. M. U., & Luckwill, R. G. (1985). Responses of the lower limb to load carrying in walking man. *European Journal of Applied Physiology and Occupational Physiology, 54*(2), 145-150.

Gibson, T., Hameed, K., Kadir, M., Sultana, S., Fatima, Z., & Syed, A. (1996). Knee pain amongst the poor and affluent in Pakistan. *British Journal of Rheumatology, 35*(2), 146-149.

Goble, D. J., Mousigian, M. A., & Brown, S. H. (2012). Compromised encoding of proprioceptively determined joint angles in older adults: The role of working memory and attentional load. *Experimental Brain Research, 216*(1), 35-40.

Gregory, J. E., Mark, R. F., Morgan, D. L., Patak, A., Polus, B., & Proske, U. (1990). Effects of muscle history on the stretch reflex in cat and man. *Journal of Physiology, 424*, 93-107.

Griffin, T. M., & Guilak, F. (2005). The role of mechanical loading in the onset and progression of osteoarthritis. *Exercise and Sport Sciences Reviews, 33*(4), 195-200.

Grob, K. R., Kuster, M. S., Higgins, S. A., Lloyd, D. G., & Yata, H. (2002). Lack of correlation between different measurements of proprioception in the knee. *Journal of Bone and Joint Surgery - Series B, 84*(4), 614-618.

Guccione, A. A., Felson, D. T., Anderson, J. J., Anthony, J. M., Zhang, Y., Wilson, P. W. F., . . . Kannel, W. B. (1994). The effects of specific medical conditions on the functional limitations of elders in the Framingham study. *American Journal of Public Health, 84*(3), 351-358.

Guilak, F. (2011). Biomechanical factors in osteoarthritis. *Best Practice and Research: Clinical Rheumatology, 25*(6), 815-823.

Hall, M., Boyer, E. R., Gillette, J. C., & Mirka, G. A. (2013). Medial knee joint loading during stair ambulation and walking while carrying loads. *Gait and Posture, 37*(3), 460-462. doi:10.1016/j.gaitpost.2012.08.008

Hall, M. C., Mockett, S. P., & Doherty, M. (2006). Relative impact of radiographic osteoarthritis and pain on quadriceps strength, proprioception, static postural sway and lower limb function. *Annals of the Rheumatic Diseases, 65*(7), 865-870.

Han, J. T., & Lee, J. -. (2014). Effects of kinesiology taping on repositioning error of the knee joint after quadriceps muscle fatigue. *Journal of Physical Therapy Science, 26*(6), 921-923. doi:10.1589/jpts.26.921

Hannan, M. T., Felson, D. T., & Pincus, T. (2000). Analysis of the discordance between radiographic changes and knee pain in osteoarthritis of the knee. *Journal of Rheumatology, 27*(6), 1513-1517.

Hassan, B. S., Doherty, S. A., Mockett, S., & Doherty, M. (2002). Effect of pain reduction on postural sway, proprioception, and quadriceps strength in subjects with knee osteoarthritis. *Annals of the Rheumatic Diseases, 61*(5), 422-428.

Hassan, B. S., Mockett, S., & Doherty, M. (2001). Static postural sway, proprioception, and maximal voluntary quadriceps contraction in patients with knee osteoarthritis and normal control subjects. *Annals of the Rheumatic Diseases, 60*(6), 612-618.

Henriksen, M., Simonsen, E. B., Graven-Nielsen, T., Lund, H., Danneskiold-Samsøe, B., & Bliddal, H. (2006). Impulse-forces during walking are not increased in patients with knee osteoarthritis. *Acta Orthopaedica, 77*(4), 650-656.

Herzog, W., Longino, D., & Clark, A. (2003). The role of muscles in joint adaptation and degeneration. *Langenbeck's Archives of Surgery, 388*(5), 305-315. doi:10.1007/s00423-003-0402-6

Hewett, T. E., Myer, G. D., Ford, K. R., Heidt Jr., R. S., Colosimo, A. J., McLean, S. G., . . . Succop, P. (2005). Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: A prospective study. *American Journal of Sports Medicine, 33*(4), 492-501. doi:10.1177/0363546504269591

Hewett, T. E., Paterno, M. V., & Myer, G. D. (2002). Strategies for enhancing proprioception and neuromuscular control of the knee. *Clinical Orthopaedics and Related Research,* (402), 76-94.

Hewitt, B. A., Refshauge, K. M., & Kilbreath, S. L. (2002). Kinesthesia at the knee: The effect of osteoarthritis and bandage application. *Arthritis Care and Research, 47*(5), 479-483.

Hiemstra, L. A., Lo, I. K. Y., & Fowler, P. J. (2001). Effect of fatigue on knee proprioception: Implications for dynamic stabilization. *Journal of Orthopaedic and Sports Physical Therapy, 31*(10), 598-605.

Hodges, P. W., & Bui, B. H. (1996). A comparison of computer-based methods for the determination of onset of muscle contraction using electromyography. *Electroencephalography and Clinical Neurophysiology - Electromyography and Motor Control, 101*(6), 511-519.

Hofer, J. K., Gejo, R., McGarry, M. H., & Lee, T. Q. (2011). Effects on tibiofemoral biomechanics from kneeling. *Clinical Biomechanics, 26*(6), 605-611.

Hogervorst, T., & Brand, R. A. (1998). Mechanoreceptors in joint function. *Journal of Bone and Joint Surgery - Series A, 80*(9), 1365-1378.

Hootman, J. M., Fitzgerald, S., Macera, C. A., & Blair, S. N. (2004). Lower extremity muscle strength and risk of self-reported hip or knee osteoarthritis. *Journal of Physical Activity and Health, 1*(4), 321-330.

Hortobágyi, T., Garry, J., Holbert, D., & Devita, P. (2004). Aberrations in the control of quadriceps muscle force in patients with knee osteoarthritis. *Arthritis Care and Research, 51*(4), 562-569.

Hoshino, A., & Wallace, W. A. (1987). Impact-absorbing properties of the human knee. *Journal of Bone and Joint Surgery - Series B, 69*(5), 807-811.

Hosp, S., Bottoni, G., Heinrich, D., Kofler, P., Hasler, M., & Nachbauer, W. (2015). A pilot study of the effect of kinesiology tape on knee proprioception after physical activity in healthy women. *Journal of Science and Medicine in Sport, 18*(6), 709-713. doi:10.1016/j.jsams.2014.09.004

Hosseini, A., Qi, W., Tsai, T. -., Liu, Y., Rubash, H., & Li, G. (2015). In vivo length change patterns of the medial and lateral collateral ligaments along the flexion path of the knee. *Knee Surgery, Sports Traumatology, Arthroscopy, 23*(10), 3055-3061. doi:10.1007/s00167-014-3306-9

Howarth, S. J., & Callaghan, J. P. (2009). The rule of 1 s for padding kinematic data prior to digital filtering: Influence of sampling and filter cutoff frequencies. *Journal of Electromyography and Kinesiology, 19*(5), 875-881. doi:10.1016/j.jelekin.2008.03.010

Hunt, M. A., Hinman, R. S., Metcalf, B. R., Lim, B. -., Wrigley, T. V., Bowles, K. -., . . . Bennell, K. L. (2010). Quadriceps strength is not related to gait impact loading in knee osteoarthritis. *Knee, 17*(4), 296-302.

Hunter, D. J. (Ed.). (2009). *Osteoarthritis - medical clinics of North America, v. 93, no. 1*. Philadelphia, PA.: Suanders.

Hunter, D. J., Schofield, D., & Callander, E. (2014). The individual and socioeconomic impact of osteoarthritis. *Nature Reviews Rheumatology, 10*(7), 437-441.

Hunter, D. J., Zhang, Y. Q., Tu, X., LaValley, M., Niu, J. B., Amin, S., . . . Felson, D. T. (2006). Change in joint space width: Hyaline articular cartilage loss or alteration in meniscus? *Arthritis and Rheumatism, 54*(8), 2488-2495.

Hurley, M. V. (1999). The role of muscle weakness in the pathogenesis of osteoarthritis. *Rheumatic Disease Clinics of North America, 25*(2), 283-298.

Hurley, M. V., & Scott, D. L. (1998). Improvements in quadriceps sensorimotor function and disability of patients with knee osteoarthritis following a clinically practicable exercise regime. *British Journal of Rheumatology, 37*(11), 1181-1187.

Hurley, M. V., Scott, D. L., Rees, J., & Newham, D. J. (1997). Sensorimotor changes and functional performance in patients with knee osteoarthritis. *Annals of the Rheumatic Diseases, 56*(11), 641-648.

Imhof, H., Sulzbacher, I., Grampp, S., Czerny, C., Youssefzadeh, S., & Kainberger, F. (2000). Subchondral bone and cartilage disease: A rediscovered functional unit. *Investigative Radiology, 35*(10), 581-588.

Iosifidis, M. I., Tsarouhas, A., & Fylaktou, A. (2014). Lower limb clinical and radiographic osteoarthritis in former elite male athletes. *Knee Surgery, Sports Traumatology, Arthroscopy,*

Jefferson, R. J., Collins, J. J., Whittle, M. W., Radin, E. L., & O'Connor, J. J. (1990). Role of the quadriceps in controlling impulsive forces around heel strike. *Proceedings of the Institution of Mechanical Engineers, Part H: Journal of Engineering in Medicine, 204*(1), 21-28.

Jensen, L. K., Rytter, S., & Bonde, J. P. (2010). Exposure assessment of kneeling work activities among floor layers. *Applied Ergonomics, 41*(2), 319-325.

Jensen, L. K., Rytter, S., Marott, J. L., & Bonde, J. P. (2012). Relationship between years in the trade and the development of radiographic knee osteoarthritis and MRI-detected meniscal tears and bursitis in floor layers. A cross-sectional study of a historical cohort. *BMJ Open, 2*(3)

Johansson, H., Sjolander, P., & Sojka, P. (1991a). Receptors in the knee joint ligaments and their role in the biomechanics of the joint. *Critical Reviews in Biomedical Engineering, 18*(5), 341-368.

Johansson, H., Sjolander, P., & Sojka, P. (1991b). A sensory role for the cruciate ligaments. *Clinical Orthopaedics and Related Research,* (268), 161-178.

Johansson, H., Sjölander, P., & Sojka, P. (1990). Activity in receptor afferents from the anterior cruciate ligament evokes reflex effects on fusimotor neurones. *Neuroscience Research, 8*(1), 54-59. doi:10.1016/0168-0102(90)90057-L

Kajaks, T. (2008). *The effect of sustained static kneeling on knee joint gait parameters.* (Unpublished Master's). Queen's University, Kingston, ON, CA.

Kajaks, T., & Costigan, P. (2015). The effect of sustained static kneeling on kinetic and kinematic knee joint gait parameters. *Applied Ergonomics, 46*(PA), 224-230.

Kaprio, J., Kujala, U. M., Peltonen, L., & Koskenvuo, M. (1996). Genetic liability to osteoarthritis may be greater in women than men [7]. *British Medical Journal, 313*(7051), 232.

Kean, C. O., Birmingham, T. B., Garland, J. S., Jenkyn, T. R., Ivanova, T. D., Jones, I. C., & Giffin, R. J. (2009). Moments and muscle activity after high tibial osteotomy and anterior cruciate ligament reconstruction. *Medicine and Science in Sports and Exercise, 41*(3), 612-619.

Kellgren, J. H., & Lawrence, J. S. (1952). Rheumatism in miners. II. X-ray study. *British Journal of Industrial Medicine, 9*(3), 197-207.

Kellgren, J. H., & Lawrence, J. S. (1957). Radiological assessment of osteo-arthrosis. *Annals of the Rheumatic Diseases, 16*(4), 494-502.

Kim, S. -., Bae, J. -., & Lim, H. -. (2013). Does torn discoid meniscus have effects on limb alignment and arthritic change in middle-aged patients? *Journal of Bone and Joint Surgery - Series A, 95*(22), 2008-2014.

Knoop, J., Steultjens, M. P. M., van der Leeden, M., van der Esch, M., Thorstensson, C. A., Roorda, L. D., . . . Dekker, J. (2011). Proprioception in knee osteoarthritis: A narrative review. *Osteoarthritis and Cartilage, 19*(4), 381-388.

Kopec, J. A., Sayre, E. C., Schwartz, T. A., Renner, J. B., Helmick, C. G., Badley, E. M., . . . Jordan, J. M. (2013). Occurrence of radiographic osteoarthritis of the knee and hip among African Americans and whites: A population-based prospective cohort study. *Arthritis Care and Research, 65*(6), 928-935.

Koralewicz, L. M., & Engh, G. A. (2000). Comparison of proprioception in arthritic and age-matched normal knees. *Journal of Bone and Joint Surgery - Series A, 82*(11), 1582-1588.

Kuettner, K. E., & Goldberg, V. M. (Eds.). (1995). *Osteoarthritic disorders* (1st ed.). Rosemont, IL: The Academy.

Kuijt, M. T. K., Inklaar, H., Gouttebarge, V., & Frings-Dresen, M. H. W. (2012). Knee and ankle osteoarthritis in former elite soccer players: A systematic review of the recent literature. *Journal of Science and Medicine in Sport, 15*(6), 480-487.

Kujala, U. M., Kaprio, J., & Sarna, S. (1994). Osteoarthritis of weight bearing joints of lower limbs in former elite male athletes. *British Medical Journal, 308*(6923), 231-234.

Kumar, D., Souza, R., Subburaj, K., MacLeod, T., Singh, J., Calixto, N., . . . Majumdar, S. (2015). Are there sex differences in knee cartilage composition and walking mechanics in healthy and osteoarthritis populations? *Clinical Orthopaedics and Related Research, 473*(8), 2548-2558. doi:10.1007/s11999-015-4212-2

Kurosawa, H., Fukubayashi, T., & Nakajima, H. (1980). Load-bearing mode of the knee joint: Physical behavior of the knee joint with or without menisci. *Clinical Orthopaedics and Related Research, 149*, 283-290.

Kwon, O. -., Minor, S. D., Maluf, K. S., & Mueller, M. J. (2003). Comparison of muscle activity during walking in subjects with and without diabetic neuropathy. *Gait and Posture, 18*(1), 105-113. doi:10.1016/S0966-6362(02)00166-2

Lane, N. E. (1995). Exercise: A cause of osteoarthritis. *The Journal of Rheumatology.Supplement, 43*, 3-6.

Larsen, R., Lund, H., Christensen, R., Røgind, H., Donneskiold-Samsøe, B., & Bliddal, H. (2005). Effect of static stretching of quadriceps and hamstring muscles on knee joint position sense. *British Journal of Sports Medicine, 39*(1), 43-46.

Laslett, L. L., Quinn, S., Burgess, J. R., Parameswaran, V., Winzenberg, T. M., Jones, G., et al. (2014). Moderate vitamin D deficiency is associated with changes in knee and hip pain in older adults: A 5-year longitudinal study. *Annals of the Rheumatic Diseases, 73*(4), 697-703.

Lathrop-Lambach, R. L., Asay, J. L., Jamison, S. T., Pan, X., Schmitt, L. C., Blazek, K., . . . Chaudhari, A. M. W. (2014). Evidence for joint moment asymmetry in healthy populations during gait. *Gait & Posture, 40*(4), 526-531. doi:10.1016/j.gaitpost.2014.06.010

Lattanzio, P. -., & Petrella, R. J. (1998). Knee proprioception: A review of mechanisms measurements, and implications of muscular fatigue. *Orthopedics, 21*(4), 463-471.

Lawrence, R. C., Felson, D. T., Helmick, C. G., Arnold, L. M., Choi, H., Deyo, R. A., . . . Wolfe, F. (2008). Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II. *Arthritis and Rheumatism, 58*(1), 26-35.

Lelas, J. L., Merriman, G. J., Riley, P. O., & Kerrigan, D. C. (2003). Predicting peak kinematic and kinetic parameters from gait speed. *Gait and Posture, 17*(2), 106-112.

Lephart, S. M., & Fu, F. H. (Eds.). (2000). *Proprioception and neuromuscular control in joint stability* (2nd ed.). Champaign, Ill.: Human Kinetics.

Lewek, M. D., Rudolph, K. S., & Snyder-Mackler, L. (2004). Quadriceps femoris muscle weakness and activation failure in patients with symptomatic knee osteoarthritis. *Journal of Orthopaedic Research, 22*(1), 110-115.

Li, G., Zayontz, S., Most, E., DeFrate, L. E., Suggs, J. F., & Rubash, H. E. (2004). In situ forces of the anterior and posterior cruciate ligaments in high knee flexion: An in vitro investigation. *Journal of Orthopaedic Research, 22*(2), 293-297.

Liikavainio, T., Isolehto, J., Helminen, H. J., Perttunen, J., Lepola, V., Kiviranta, I., . . . Komi, P. V. (2007). Loading and gait symmetry during level and stair walking in asymptomatic subjects with knee osteoarthritis: Importance of quadriceps femoris in reducing impact force during heel strike? *Knee, 14*(3), 231-238.

Lin, D. H., Lin, Y. F., Chai, H. M., Han, Y. C., & Jan, M. (2007). Comparison of proprioceptive functions between computerized proprioception facilitation exercise and closed kinetic chain exercise in patients with knee osteoarthritis. *Clinical Rheumatology, 26*(4), 520-528.

Lin, H., Hsu, A., Chang, J., Chien, C., & Chang, G. (2008). Comparison of EMG activity between maximal manual muscle testing and cybex maximal isometric testing of the quadriceps femoris. *Journal of the Formosan Medical Association, 107*(2), 175-180. doi:10.1016/S0929-6646(08)60131-X

Lin, Z., Willers, C., Xu, J., & Zheng, M. -. (2006). The chondrocyte: Biology and clinical application. *Tissue Engineering, 12*(7), 1971-1984.

Lindstedt, S. L., LaStayo, P. C., & Reich, T. E. (2001). When active muscles lengthen: Properties and consequences of eccentric contractions. *News in Physiological Sciences, 16*(6), 256-261.

Liu, H., Zhao, Z., Clarke, R. B., Gao, J., Garrett, I. R., & Margerrison, E. E. C. (2013). Enhanced tissue regeneration potential of juvenile articular cartilage. *American Journal of Sports Medicine, 41*(11), 2658-2667.

Lund, H., Juul-Kristensen, B., Hansen, K., Christensen, R., Christensen, H., Danneskiold-Samsoe, B., & Bliddal, H. (2008). Movement detection impaired in patients with knee osteoarthritis compared to healthy controls: A cross-sectional case-control study. *Journal of Musculoskeletal Neuronal Interactions, 8*(4), 391-400.

MacIsaac, D., Parker, P. A., & Scott, R. N. (2001). The short-time Fourier transform and muscle fatigue assessment in dynamic contractions. *Journal of Electromyography and Kinesiology, 11*(6), 439-449. doi:10.1016/S1050-6411(01)00021-9

Madding, S. W., Wong, J. G., Hallum, A., & Medeiros, J. M. (1987). Effect of duration of passive stretch on hip abduction range of motion. *Journal of Orthopaedic and Sports Physical Therapy, 8*(8), 409-416.

Manninen, P., Heliövaara, M., Riihimäki, H., & Suomalainen, O. (2002). Physical workload and the risk of severe knee osteoarthritis. *Scandinavian Journal of Work, Environment and Health, 28*(1), 25-32.

Marks, R., Quinney, H. A., & Wessel, J. (1993). Proprioceptive sensibility in women with normal and osteoarthritic knee joints. *Clinical Rheumatology, 12*(2), 170-175.

Mastalerz, A., Gwarek, L., Sadowski, J., & Szczepanski, T. (2012). The influence of the run intensity on bioelectrical activity of selected human leg muscles. *Acta of Bioengineering and Biomechanics, 14*(2), 101-107. doi:10.5277/abb120213

Mazzuca, S. A., Brandt, K. D., Katz, B. P., Ding, Y., Lane, K. A., & Buckwalter, K. A. (2007). Risk factors for early radiographic changes of tibiofemoral osteoarthritis. *Annals of the Rheumatic Diseases, 66*(3), 394-399.

McAlindon, T. E., Felson, D. T., Zhang, Y., Hannan, M. T., Aliabadi, P., Weissman, B., et al. (1996). Relation of dietary intake and serum levels of vitamin D to progression of osteoarthritis of the knee among participants in the Framingham study. *Annals of Internal Medicine,125*(5), 353-359.

McDermott, I. D., Masouros, S. D., & Amis, A. A. (2008). Biomechanics of the menisci of the knee. *Current Orthopaedics, 22*(3), 193-201.

McGill, S. M., & Brown, S. (1992). Creep response of the lumbar spine to prolonged full flexion. *Clinical Biomechanics, 7*(1), 43-46. doi:10.1016/0268-0033(92)90007-Q

McMillan, G., & Nichols, L. (2005). Osteoarthritis and meniscus disorders of the knee as occupational diseases of miners. *Occupational and Environmental Medicine, 62*(8), 567-575.

McWilliams, D. F., Leeb, B. F., Muthuri, S. G., Doherty, M., & Zhang, W. (2011). Occupational risk factors for osteoarthritis of the knee: A meta-analysis. *Osteoarthritis and Cartilage, 19*(7), 829-839. doi:10.1016/j.joca.2011.02.016

Mikesky, A. E., Meyer, A., & Thompson, K. L. (2000). Relationship between quadriceps strength and rate of loading during gait in women. *Journal of Orthopaedic Research, 18*(2), 171-175.

Minshull, C., Gleeson, N., Walters-Edwards, M., Eston, R., & Rees, D. (2007). Effects of acute fatigue on the volitional and magnetically-evoked electromechanical delay of the knee flexors in males and females. *European Journal of Applied Physiology, 100*(4), 469-478. doi:10.1007/s00421-007-0448-1

Miura, K., Ishibashi, Y., Tsuda, E., Okamura, Y., Otsuka, H., & Toh, S. (2004). The effect of local and general fatigue on knee proprioception. *Arthroscopy - Journal of Arthroscopic and Related Surgery, 20*(4), 414-418. doi:10.1016/j.arthro.2004.01.007

Miyazaki, T., Wada, M., Kawahara, H., Sato, M., Baba, H., & Shimada, S. (2002). Dynamic load at baseline can predict radiographic disease progression in medial compartment knee osteoarthritis. *Annals of the Rheumatic Diseases, 61*(7), 617-622.

Moisio, K. C., Sumner, D. R., Shott, S., & Hurwitz, D. E. (2003). Normalization of joint moments during gait: A comparison of two techniques. *Journal of Biomechanics, 36*(4), 599-603.

Moore, K. L., & Dalley, A. F. (2006). *Clinically oriented anatomy* (5th ed.). Baltimore, MD: Lippincott Williams & Wilkins.

Moore, B. D., Drouin, J., Gansneder, B. M., & Shultz, S. J. (2002). The differential effects of fatigue on reflex response timing and amplitude in males and females. *Journal of Electromyography and Kinesiology, 12*(5), 351-360. doi:http://dx.doi.org/10.1016/S1050-6411(02)00032-9

Muaidi, Q. I., Nicholson, L. L., & Refshauge, K. M. (2009). Do elite athletes exhibit enhanced proprioceptive acuity, range and strength of knee rotation compared with non-athletes? *Scandinavian Journal of Medicine and Science in Sports, 19*(1), 103-112. doi:10.1111/j.1600-0838.2008.00783.x

Mündermann, A., Dyrby, C. O., Hurwitz, D. E., Sharma, L., & Andriacchi, T. P. (2004). Potential strategies to reduce medial compartment loading in patients with knee osteoarthritis of varying severity: Reduced walking speed. *Arthritis and Rheumatism, 50*(4), 1172-1178.

Muthuri, S. G., McWilliams, D. F., Doherty, M., & Zhang, W. (2011). History of knee injuries and knee osteoarthritis: A meta-analysis of observational studies. *Osteoarthritis and Cartilage, 19*(11), 1286-1293.

Nagura, T., Dyrby, C. O., Alexander, E. J., & Andriacchi, T. P. (2002). Mechanical loads at the knee joint during deep flexion. *Journal of Orthopaedic Research, 20*(4), 881-886.

Naseri, N., & Pourkazemi, F. (2012). Difference in knee joint position sense in athletes with and without patellofemoral pain syndrome. *Knee Surgery, Sports Traumatology, Arthroscopy, 20*(10), 2071-2076.

Neame, R. L., Muir, K., Doherty, S., & Doherty, M. (2004). Genetic risk of knee osteoarthritis: A sibling study. *Annals of the Rheumatic Diseases, 63*(9), 1022-1027.

Nelson-Wong, E., & Callaghan, J. P. (2014). Transient low back pain development during standing predicts future clinical low back pain in previously asymptomatic individuals. *Spine, 39*(6), E379-E383.

Nicolella, D. P., O'Connor, M. I., Enoka, R. M., Boyan, B. D., Hart, D. A., Resnick, E., . . . Kohrt, W. M. (2012). Mechanical contributors to sex differences in idiopathic knee osteoarthritis. *Biology of Sex Differences, 3*(28)

Nigg, B. M., Cole, G. K., & Bruggemann, G. -. (1995). Impact forces during heel-toe running. *Journal of Applied Biomechanics, 11*(4), 407-432.

Noyes, F. R., Schipplein, O. D., Andriacchi, T. P., Saddemi, S. R., & Weise, M. (1992). The anterior cruciate ligament-deficient knee with varus alignment. an analysis of gait adaptations and dynamic joint loadings. *American Journal of Sports Medicine, 20*(6), 707-716.

Nuki, G. (1999). Osteoarthritis: A problem of joint failure. *Zeitschrift Fur Rheumatologie, 58*(3), 142-147.

O'Hara, B. P., Urban, J. P. G., & Maroudas, A. (1990). Influence of cyclic loading on the nutrition of articular cartilage. *Annals of the Rheumatic Diseases, 49*(7), 536-539.

O'Reilly, S., Jones, A., & Doherty, M. (1997). Muscle weakness in osteoarthritis. *Current Opinion in Rheumatology, 9*(3), 259-262.

Pai, Y. I. -., Rymer, W. Z., Chang, R. W., & Sharma, L. (1997). Effect of age and osteoarthritis on knee proprioception. *Arthritis and Rheumatism, 40*(12), 2260-2265.

Park, K. K., Hosseini, A., Tsai, T. -., Kwon, Y. -., & Li, G. (2015). Elongation of the collateral ligaments after cruciate retaining total knee arthroplasty and the maximum flexion of the knee. *Journal of Biomechanics, 48*(3), 418-424. doi:10.1016/j.jbiomech.2014.12.036

Park, S. E., DeFrate, L. E., Suggs, J. F., Gill, T. J., Rubash, H. E., & Li, G. (2006). Erratum to "the change in length of the medial and lateral collateral ligaments during in vivo knee flexion". *The Knee, 13*(1), 77-82. doi:10.1016/j.knee.2004.12.012

Partridge, R. E., & Duthie, J. J. (1968). Rheumatism in dockers and civil servants. A comparison of heavy manual and sedentary workers. *Annals of the Rheumatic Diseases, 27*(6), 559-568.

Patterson, M. R., Delahunt, E., & Caulfield, B. (2014). Peak knee adduction moment during gait in anterior cruciate ligament reconstructed females. *Clinical Biomechanics, 29*(2), 138-142. doi:10.1016/j.clinbiomech.2013.11.021

Pearle, A. D., Warren, R. F., & Rodeo, S. A. (2005). Basic science of articular cartilage and osteoarthritis. *Clinics in Sports Medicine, 24*(1), 1-12.

Peeler, J., & Anderson, J. E. (2008). Reliability of the Ely's test for assessing rectus femoris muscle flexibility and joint range of motion. *Journal of Orthopaedic Research, 26*(6), 793-799.

Pohl, M. B., Mullineaux, D. R., Milner, C. E., Hamill, J., & Davis, I. S. (2008). Biomechanical predictors of retrospective tibial stress fractures in runners. *Journal of Biomechanics, 41*(6), 1160-1165. doi:10.1016/j.jbiomech.2008.02.001

Potvin J.R., & Bent L.R. (1997). A validation of techniques using surface EMG signals from dynamic contractions to quantify muscle fatigue during repetitive tasks. *Journal of Electromyography and Kinesiology, 7*(2), 131-139.

Potvin, J. R., & Brown, S. H. M. (2004). Less is more: High pass filtering, to remove up to 99% of the surface EMG signal power, improves EMG-based biceps brachii muscle force estimates. *Journal of Electromyography and Kinesiology, 14*(3), 389-399. doi:10.1016/j.jelekin.2003.10.005

Powers, C. M., Landel, R., & Perry, J. (1996). Timing and intensity of vastus muscle activity during functional activities in subjects with and without patellofemoral pain. *Physical Therapy, 76*(9), 946-967.

Prentice, W., E. (2011). In Arnheim D., D. (Ed.), *Principles of athletic training: A competency-based approach* (14th ed.). New York: McGraw-Hill Higher Education, c2011.

Proske, U., Wise, A. K., & Gregory, J. E. (2000). The role of muscle receptors in the detection of movements. *Progress in Neurobiology, 60*(1), 85-96.

Radin, E. L., & Rose, R. M. (1986). Role of subchondral bone in the initiation and progression of cartilage damage. *Clinical Orthopaedics and Related Research, 213*, 34-40.

Radin, E. L. (2004). Who gets osteoarthritis and why? *Journal of Rheumatology, 31*(SUPPL. 70), 10-15.

Radin, E. L., Martin, R. B., Burr, D. B., Caterson, B., Boyd, R. D., & Goodwin, C. (1984). Effects of mechanical loading on the tissues of the rabbit knee. *Journal of Orthopaedic Research, 2*(3), 221-234.

Radin, E. L., Whittle, M. W., Yang, K. H., Jefferson, R. J., Rodgers, M. M., Kish, V. L., & O'Connor, J. J. (1986). Heelstrike transient, its relationship with the angular velocity of the shank and the effects of quadriceps paralysis. 121-123.

Radin, E. L., Yang, K. H., Riegger, C., Kish, V. L., & O'Connor, J. J. (1991). Relationship between lower limb dynamics and knee joint pain. *Journal of Orthopaedic Research, 9*(3), 398-405.

Radzimski, A. O., Muendermann, A., & Sole, G. (2012). Effect of footwear on the external knee adduction moment - A systematic review. *Knee, 19*(3), 163-175. doi:10.1016/j.knee.2011.05.013

Reid, C., Bush, P., Cummings, N., McMullin, D., & Durrani, S. (2010). A review of occupational knee disorders. *Journal of Occupational Rehabilitation, 20*(4), 489-501. doi:10.1007/s10926-010-9242-8

Riskowski, J. L., Mikesky, A. E., Bahamonde, R. E., Alvey III, T. V., & Burr, D. B. (2005). Proprioception, gait kinematics, and rate of loading during walking: Are they related? *Journal of Musculoskeletal Neuronal Interactions, 5*(4), 379-387.

Robertson, G. E., Caldwell, G. E., Hamill, J., Kamen, G., & Whittlesey, S. N. (2004). *Research methods in biomechanics* (1st ed.). Champaign, IL, US: Human Kinetics.

Roos, E. M., Herzog, W., Block, J. A., & Bennell, K. L. (2011). Muscle weakness, afferent sensory dysfunction and exercise in knee osteoarthritis. *Nature Reviews Rheumatology, 7*(1), 57-63.

Rosenbaum, D., & Hennig, E. M. (1995). The influence of stretching and warm-up exercises on Achilles tendon reflex activity. *Journal of Sports Sciences, 13*(6), 481-490.

Rothwell, J. (1994). *Control of human voluntary movement* (2nd ed.). London; New York: Chapman & Hall.

Rozzi, S. L., Lephart, S. M., & Fu, F. H. (1999). Effects of muscular fatigue on knee joint laxity and neuromuscular characteristics of male and female athletes. *Journal of Athletic Training, 34*(2), 106-114.

Rutherford, O. M., Jones, D. A., & Newham, D. J. (1986). Clinical and experimental application of the percutaneous twitch superimposition technique for the study of human muscle activation. *Journal of Neurology Neurosurgery and Psychiatry, 49*(11), 1288-1291.

Sandmark, H., Hogstedt, C., & Vingård, E. (2000). Primary osteoarthrosis of the knee in men and women as a result of lifelong physical load from work. *Scandinavian Journal of Work, Environment and Health, 26*(1), 20-25.

Sbriccoli, P., Solomonow, M., Zhou, B. -., Lu, Y., & Sellards, R. (2005). Neuromuscular response to cyclic loading of the anterior cruciate ligament. *American Journal of Sports Medicine, 33*(4), 543-551.

Schiphof, D., Boers, M., & Bierma-Zeinstra, S. M. A. (2008). Differences in descriptions of Kellgren and Lawrence grades of knee osteoarthritis. *Annals of the Rheumatic Diseases, 67*(7), 1034-1036.

Schipplein, O. D., & Andriacchi, T. P. (1991). Interaction between active and passive knee stabilizers during level walking. *Journal of Orthopaedic Research, 9*(1), 113-119.

Schmitz, A., Silder, A., Heiderscheit, B., Mahoney, J., & Thelen, D. G. (2009). Differences in lower-extremity muscular activation during walking between healthy older and young adults. *Journal of Electromyography and Kinesiology, 19*(6), 1085-1091.

Schneiders, A. G., Sullivan, S. J., O'Malley, K. J., Clarke, S. V., Knappstein, S. A., & Taylor, L. J. (2010). A valid and reliable clinical determination of footedness. *PM and R, 2*(9), 835-841.

Schwartz, M. H., & Rozumalski, A. (2005). A new method for estimating joint parameters from motion data. *Journal of Biomechanics, 38*(1), 107-116.

Seedhom, B. B. (1979). Transmission of the load in the knee joint with special reference to the role of the menisci. Part I. Anatomy, analysis and apparatus. *Engineering in Medicine, 8*(4), 207-219.

Seedhom, B. B., & Hargreaves, D. J. (1979). Transmission of the load in the knee joint with special reference to the role of the menisci. Part II: Experimental results, discussion and conclusions. *Engineering in Medicine, 8*(4), 220-228.

Segal, N. A., Findlay, C., Wang, K., Torner, J. C., & Nevitt, M. C. (2012). The longitudinal relationship between thigh muscle mass and the development of knee osteoarthritis. *Osteoarthritis and Cartilage, 20*(12), 1534-1540.

Segal, N. A., & Glass, N. A. (2011). Is quadriceps muscle weakness a risk factor for incident or progressive knee osteoarthritis? *Physician and Sportsmedicine, 39*(4), 44-50.

Segal, N. A., Glass, N. A., Felson, D. T., Hurley, M., Yang, M., Nevitt, M., . . . Torner, J. C. (2010). Effect of quadriceps strength and proprioception on risk for knee osteoarthritis. *Medicine and Science in Sports and Exercise, 42*(11), 2081-2088.

Segal, N. A., Glass, N. A., Torner, J., Yang, M., Felson, D. T., Sharma, L., . . . Lewis, C. E. (2010). Quadriceps weakness predicts risk for knee joint space narrowing in women in the MOST cohort. *Osteoarthritis and Cartilage, 18*(6), 769-775.

Segal, N. A., Torner, J. C., Felson, D., Niu, J., Sharma, L., Lewis, C. E., & Nevitt, M. (2009). Effect of thigh strength on incident radiographic and symptomatic knee osteoarthritis in a longitudinal cohort. *Arthritis Care and Research, 61*(9), 1210-1217.

Sharma, L., Kapoor, D., & Issa, S. (2006). Epidemiology of osteoarthritis: An update. *Current Opinion in Rheumatology, 18*(2), 147-156.

Sharma, L., Pai, Y. I. -., Holtkamp, K., & Rymer, W. Z. (1997). Is knee joint proprioception worse in the arthritic knee versus the unaffected knee in unilateral knee osteoarthritis? *Arthritis and Rheumatism, 40*(8), 1518-1525.

Shiraev, T., Anderson, S. E., & Hope, N. (2012). Meniscal tear: Presentation, diagnosis and management. *Australian Family Physician, 41*(4), 182-186.

Shrier, I. (2004). Muscle dysfunction versus wear and tear as a cause of exercise related osteoarthritis: An epidemiological update. *British Journal of Sports Medicine, 38*(5), 526-535.

Simpson, K. M., Munro, B. J., & Steele, J. R. (2011). Backpack load affects lower limb muscle activity patterns of female hikers during prolonged load carriage. *Journal of Electromyography and Kinesiology, 21*(5), 782-788. doi:10.1016/j.jelekin.2011.05.012

Simpson, K. M., Munro, B. J., & Steele, J. R. (2012). Effects of prolonged load carriage on ground reaction forces, lower limb kinematics and spatio-temporal parameters in female recreational hikers. *Ergonomics, 55*(3), 316-326. doi:10.1080/00140139.2011.642004

Skinner, H. B., Barrack, R. L., & Cook, S. D. (1984). Age related decline in proprioception. *Clinical Orthopaedics and Related Research, 184*, 208-211.

Slemenda, C., Brandt, K. D., Heilman, D. K., Mazzuca, S., Braunstein, E. M., Katz, B. P., & Wolinsky, F. D. (1997). Quadriceps weakness and osteoarthritis of the knee. *Annals of Internal Medicine, 127*(2), 97-104.

Slemenda, C., Heilman, D. K., Brandt, K. D., Katz, B. P., Mazzuca, S. A., Braunstein, E. M., & Byrd, D. (1998). Reduced quadriceps strength relative to body weight: A risk factor for knee osteoarthritis in women? *Arthritis and Rheumatism, 41*(11), 1951-1959.

Snoeker, B. A. M., Bakker, E. W. P., Kegel, C. A. T., & Lucas, C. (2013). Risk factors for meniscal tears: A systematic review including meta-analysis. *Journal of Orthopaedic and Sports Physical Therapy, 43*(6), 352-367.

Solomon, L., Beighton, P., & Lawrence, J. S. (1975). Rheumatic disorders in the South African Negro. Part II. Osteoarthrosis. *South African Medical Journal, 49*(42), 1737-1740.

Solomonow, M. (2006). Sensory - motor control of ligaments and associated neuromuscular disorders. *Journal of Electromyography and Kinesiology, 16*(6), 549-567.

Solomonow, M., Baratta, R., Zhou, B. H., Shoji, H., Bose, W., Beck, C., & D'Ambrosia, R. (1987). The synergistic action of the anterior cruciate ligament and thigh muscles in maintaining joint stability. *American Journal of Sports Medicine, 15*(3), 207-214.

Solomonow, M. (2009). Ligaments: A source of musculoskeletal disorders. *Journal of Bodywork & Movement Therapies, 13*(2), 136-154. doi:10.1016/j.jbmt.2008.02.001

Sowers, M., Lachance, L., Hochberg, M., & Jamadar, D. (2000). Radiographically defined osteoarthritis of the hand and knee in young and middle-aged African American and Caucasian women. *Osteoarthritis and Cartilage, 8*(2), 69-77.

Stastny, P., Lehnert, M., Zaatar, A., Svoboda, Z., Xaverova, Z., & Jelen, K. (2014). Knee joint muscles neuromuscular activity during load-carrying walking. *Neuroendocrinology Letters, 35*(7), 633-639.

Stillman, B. C., & McMeeken, J. M. (2001). The role of weightbearing in the clinical assessment of knee joint position sense. *Australian Journal of Physiotherapy, 47*(4), 247-253.

Stokes, M., & Young, A. (1984). The contribution of reflex inhibition to arthrogenous muscle weakness. *Clinical Science, 67*(1), 7-14.

Streepey, J. W., Mock, M. J., Riskowski, J. L., Vanwye, W. R., Vitvitskiy, B. M., & Mikesky, A. E. (2010). Effects of quadriceps and hamstrings proprioceptive neuromuscular facilitation stretching on knee movement sensation. *Journal of Strength and Conditioning Research, 24*(4), 1037-1042.

Tanaka, S., Smith, A. B., Halperin, W., & Jensen, R. (1982). Carpet-layer's knee. *New England Journal of Medicine, 307*(20), 1276-1277.

Teichtahl, A. J., Wluka, A. E., Morris, M. E., Davis, S. R., & Cicuttini, F. M. (2009). The associations between the dominant and nondominant peak external knee adductor moments during gait in healthy subjects: Evidence for symmetry. *Archives of Physical Medicine and Rehabilitation, 90*(2), 320-324. doi:10.1016/j.apmr.2008.07.030

Tennant, L., Kingston, D., Chong, H., & Acker, S. (2015). The effect of work boots on knee mechanics and the center of pressure at the knee during static kneeling. *Journal of Applied Biomechanics, 31*(5), 363-369.

Thambyah, A., Goh, J. C. H., & De, S. D. (2005). Contact stresses in the knee joint in deep flexion. *Medical Engineering and Physics, 27*(4), 329-335. doi:10.1016/j.medengphy.2004.09.002

Thun, M., Tanaka, S., Smith, A. B., Halperin, W. E., Lee, S. T., Luggen, M. E., & Hess, E. V. (1987). Morbidity from repetitive knee trauma in carpet and floor layers. *British Journal of Industrial Medicine, 44*(9), 611-620.

Toft, E., Espersen, G. T., Kalund, S., Sinkjaer, T., & Hornemann, B. C. (1989). Passive tension of the ankle before and after stretching. *American Journal of Sports Medicine, 17*(4), 489-494.

Torres, R., Vasques, J., Duarte, J. A., & Cabri, J. M. H. (2010). Knee proprioception after exercise-induced muscle damage. *International Journal of Sports Medicine, 31*(6), 410-415.

Tveit, M., Rosengren, B. E., Nilsson, J. -., & Karlsson, M. K. (2012). Former male elite athletes have a higher prevalence of osteoarthritis and arthroplasty in the hip and knee than expected. *American Journal of Sports Medicine, 40*(3), 527-533.

Uhlig, T., Slatkowsky-Christensen, B., Moe, R. H., & Kvien, T. K. (2010). The burden of osteoarthritis:The societal and the patient perspective. *Therapy, 7*(6), 605-619.

Van Dommelen, B. A., & Fowler, P. J. (1989). Anatomy of the posterior cruciate ligament. A review. *American Journal of Sports Medicine, 17*(1), 24-29.

Verdini, F., Marcucci, M., Benedetti, M. G., & Leo, T. (2006). Identification and characterisation of heel strike transient. *Gait and Posture, 24*(1), 77-84.

Vigorita, J. V., Ghelman, B., & Mintz, D. (2008). *Orthopaedic pathology* (2nd ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Vincent, J. -., Magnussen, R. A., Gezmez, F., Uguen, A., Jacobi, M., Weppe, F., . . . Neyret, P. (2012). The anterolateral ligament of the human knee: An anatomic and histologic study. *Knee Surgery, Sports Traumatology, Arthroscopy, 20*(1), 147-152.

Walker, P. S., & Erkman, M. J. (1975). The role of the menisci in force transmission across the knee. *Clinical Orthopaedics and Related Research, 109*, 184-192.

Watt, I. (2009). Osteoarthritis revisited - again! *Skeletal Radiology, 38*(5), 419-423.

Watt, I., & Dieppe, P. (1990). Osteoarthritis revisited. *Skeletal Radiology, 19*(1), 1-3.

Whittle, M. W. (1999). Generation and attenuation of transient impulsive forces beneath the foot: A review. *Gait and Posture, 10*(3), 264-275.

Winter, D. A. (1991). *The biomechanics and motor control of human gait: Normal, elderly, and pathological* (2nd ed.). Waterloo, ON, CA: Waterloo Biomechanics.

Winter, D. A., Patla, A. E., & Frank, J. S. (1990). Assessment of balance control in humans. *Medical Progress through Technology, 16*(1-2), 31-51.

Wright, I. C., Neptune, R. R., Van Den Bogert, A. J., & Nigg, B. M. (1998). Passive regulation of impact forces in heel-toe running. *Clinical Biomechanics, 13*(7), 521-531.

Wu, G., & Cavanagh, P. R. (1995). ISB recommendations for standardization in the reporting of kinematic data. *Journal of Biomechanics, 28*(10), 1257-1261.

Xie, F., Thumboo, J., Fong, K. -., Lo, N. -., Yeo, S. -., Yang, K. -., & Li, S. -. (2008). A study on indirect and intangible costs for patients with knee osteoarthritis in Singapore. *Value in Health, 11*(SUPPL. 1), S84-S90.

Yang, Z., Wickwire, A. C., & Debski, R. E. (2010). Development of a subject-specific model to predict the forces in the knee ligaments at high flexion angles. *Medical and Biological Engineering and Computing, 48*(11), 1077-1085.

Yeung, S. S., Au, A. L., & Chow, C. C. (1999). Effects of fatigue on the temporal neuromuscular control of vastus medialis muscle in humans. *European Journal of Applied Physiology and Occupational Physiology, 80*(4), 379-385. doi:10.1007/s004210050607

Yoshida, S., Aoyagi, K., Felson, D. T., Aliabadi, P., Shindo, H., & Takemoto, T. -. (2002). Comparison of the prevalence of radiographic osteoarthritis of the knee and hand between Japan and the United States. *Journal of Rheumatology, 29*(7), 1454-1458.

Zabala, M. E., Favre, J., Scanlan, S. F., Donahue, J., & Andriacchi, T. P. (2013). Three-dimensional knee moments of ACL reconstructed and control subjects during gait, stair ascent, and stair descent. *Journal of Biomechanics, 46*(3), 515-520. doi:10.1016/j.jbiomech.2012.10.010

Zeni Jr. J.A., Richards J.G., & Higginson J.S. (2008). Two simple methods for determining gait events during treadmill and overground walking using kinematic data. *Gait and Posture, 27*(4), 710-714.

Zhang, Y., Hunter, D. J., Nevitt, M. C., Xu, L., Niu, J., Lui, L. -., . . . Felson, D. T. (2004). Association of squatting with increased prevalence of radiographic tibiofemoral knee osteoarthritis: The Beijing osteoarthritis study. *Arthritis and Rheumatism, 50*(4), 1187-1192.

Appendix A: Biological Contributors to Knee Osteoarthritis

A number of biological or systemic factors are understood to contribute to individual susceptibility to osteoarthritis development that were not addressed in this project, but should be acknowledged as they are understood to play an important role in knee osteoarthritis initiation and/or development.

Factor	Contribution to Knee Osteoarthritis Initiation and/or Development
Age	It is believed that it is the chemical and physical changes associated with increasing age (regardless of biomechanical loading history) that may contribute to knee osteoarthritis development. For example, cartilage volume and proteoglycan content, and bone mass all decrease with age. Therefore, age alone causes radiographic joint space narrowing. (Brandt & Fife, 1986; Kopec et al., 2013; Watt & Dieppe, 1990)
Sex	Women are at higher risk of developing knee osteoarthritis than men (Felson et al., 2000; Garstang & Stitik, 2006). The role of estrogen in bone mineral density may be important, although studies results are conflicting (Garstang and Stitik, 2006). In addition, pelvic dimensions, knee morphology, Q-angle, and neuromuscular strength have been suggested as possible risk factors for knee osteoarthritis specific to women (Zhang et al., 2004).
Race	Chinese women in Beijing demonstrated an increased prevalence of tibiofemoral osteoarthritis compared to Caucasian women (9.5%), despite having lower BMIs. However, Chinese men demonstrated a decreased prevalence of tibiofemoral osteoarthritis (7%) (Zhang et al., 2004). African-Americans may also show increased risk for knee osteoarthritis compared to Caucasians (Kopec et al., 2013; Sowers, Lachance, Hochberg, & Jamadar, 2000)
Genetics	A number of genetic factors have been linked to increased risk for the development of osteoarthritis which are thought to increase susceptibility to osteoarthritis development from mechanical factors (Felson et al., 1998; Kaprio, Kujala, Peltonen, & Koskenvuo, 1996; Neame, Muir, Doherty, & Doherty, 2004)

Factor	Contribution to Knee Osteoarthritis Initiation and/or Development
Nutrition	Diet may play a role in osteoarthritis development, although the results of studies on nutrition and osteoarthritis are highly conflicting. *Antioxidants* Early work has demonstrated that diets high in antioxidants are associated with a decreased risk for the progression of knee osteoarthritis and reduce risk of developing knee pain (Chaganti et al., 2014; McAlindon et al., 1996). However, more recently it has been suggested that high levels of antioxidant intake has no effect on knee osteoarthritis development, and may actually be associated with an increased risk (Chaganti et al., 2014). *Vitamin D* Low intake of vitamin D has been shown to be associated with increased risk for the development of knee osteoarthritis, but studies are conflicting as to vitamin D's preventative role (McAlindon et al., 1996; Laslett et al., 2014).
Bone Density	High bone density, as associated with obesity, is correlated with the radiographic knee osteoarthritis, especially when osteoarthritis is identified as the presence of osteophytes. However, the relationship between bone density and osteoarthritis may change between initial and late stages of the disease. (Arden & Nevitt, 2006)

Appendix B: Participant Exclusion Factors

Exclusion Factor	Examples
Current or previous lower extremity injury or surgery	Ligamentous tears, meniscal injury, surgical interventions, etc …
Current lower limb pathology	Ankle sprains, leg muscle strains, etc …
History of fainting or falling	
Taking medication that may affect balance	
Exposed to knee-straining postures (kneeling/squatting) during sport, leisure, or work activities	Sport – baseball catchers, curlers (frequent participation), etc ... Leisure – gardening/landscaping, etc … Occupation – landscaper, childcare worker, floor layer
Confirmed diagnosis of knee osteoarthritis	
Inability to kneel for 30 minutes	
Allergy or sensitivity to alcohol	

Appendix C: Participant Screening Questionnaire

This questionnaire asks some questions about your health status. This information is used to guide us with your entry into the study.

Contradictions to participation in this study include:

1. Any previous history of knee pain that required medical intervention or time off from work longer than three days
2. Previous knee surgery
3. Employment that required prolonged knee-straining postures such as kneeling or squatting in the past 12 months
4. Inability to kneel for 30 minutes at one time
5. Experienced bouts of dizziness and/or fainting
6. Allergy or sensitivity to alcohol

Past Relevant Health History (Check all that apply):

Musculoskeletal pain/disorders

☐ Hip Injury, please specify: _____
☐ Knee Injury, please specify: _____
☐ Ankle Injury, please specify: _____

Cardiovascular Disorders

☐ Heart Murmur
☐ Disease of the Arteries
☐ Heart Attack
☐ High Blood Pressure
☐ Congenital Heart Disease
☐ High Cholesterol
☐ Heart Disease

Respiratory disorders/disease

☐ Emphysema
☐ Asthma
☐ Pneumonia
☐ Bronchitis

Other

☐ Neoplasm
☐ Fainting
☐ Arthritis
☐ Dizziness

Current Relevant Health History (Check all that apply):

- ☐ Irregular Heartbeat
- ☐ Chest Pain
- ☐ Wheezing (Asthma)
- ☐ Back pain/injury
- ☐ Leg Pain/injury
- ☐ Fatigue
- ☐ Persistent Coughing
- ☐ Dizziness
- ☐ Shoulder pain/injury

Allergies

- ☐ Rubbing Alcohol
- ☐ Adhesives

Appendix D: Participant Information Questionnaire

Follow the directions carefully and answer to the best of your ability. To indicate a response, simply write the answer on the line provided, or, completely fill in the box beside the option you wish to select. You may omit any question you prefer not to answer. If you have any questions, please ask the research assistant.

1. What is your sex?	☐ Male ☐ Female
2. How old are you?	_____
3. How much do you weigh?	_____
4. How tall are you?	_____
5. Which hand do you write with?	☐ Right ☐ Left
6. Would you consider yourself to be in a 'good' mood today?	☐ Yes ☐ No
7. What brand of running shoes are you wearing today? (e.g. Saucony)	_____
8. What model of running shoe are you wearing today? (if known) (e.g. Saucony *Ride*, Asics *Cumulus*)	_____
9. How old are the running shoes you are wearing today?	_____years _____ months
10. On average, how many days a week to you participate in physical activities? Participation must be at least 30 minutes long. (e.g. team sports, exercise class, yoga, hiking, cycling, etc …)	☐ Less than 1 day/week ☐ 1 day/week ☐ 2 days/week ☐ 3 days/week ☐ 4 days/week ☐ 5 days/week ☐ 6 days/week ☐ every day

Thank you for taking the time to complete this survey.

Appendix E: Example Kneeling Exposure Protocol

VAS – visual analog pain scale reading

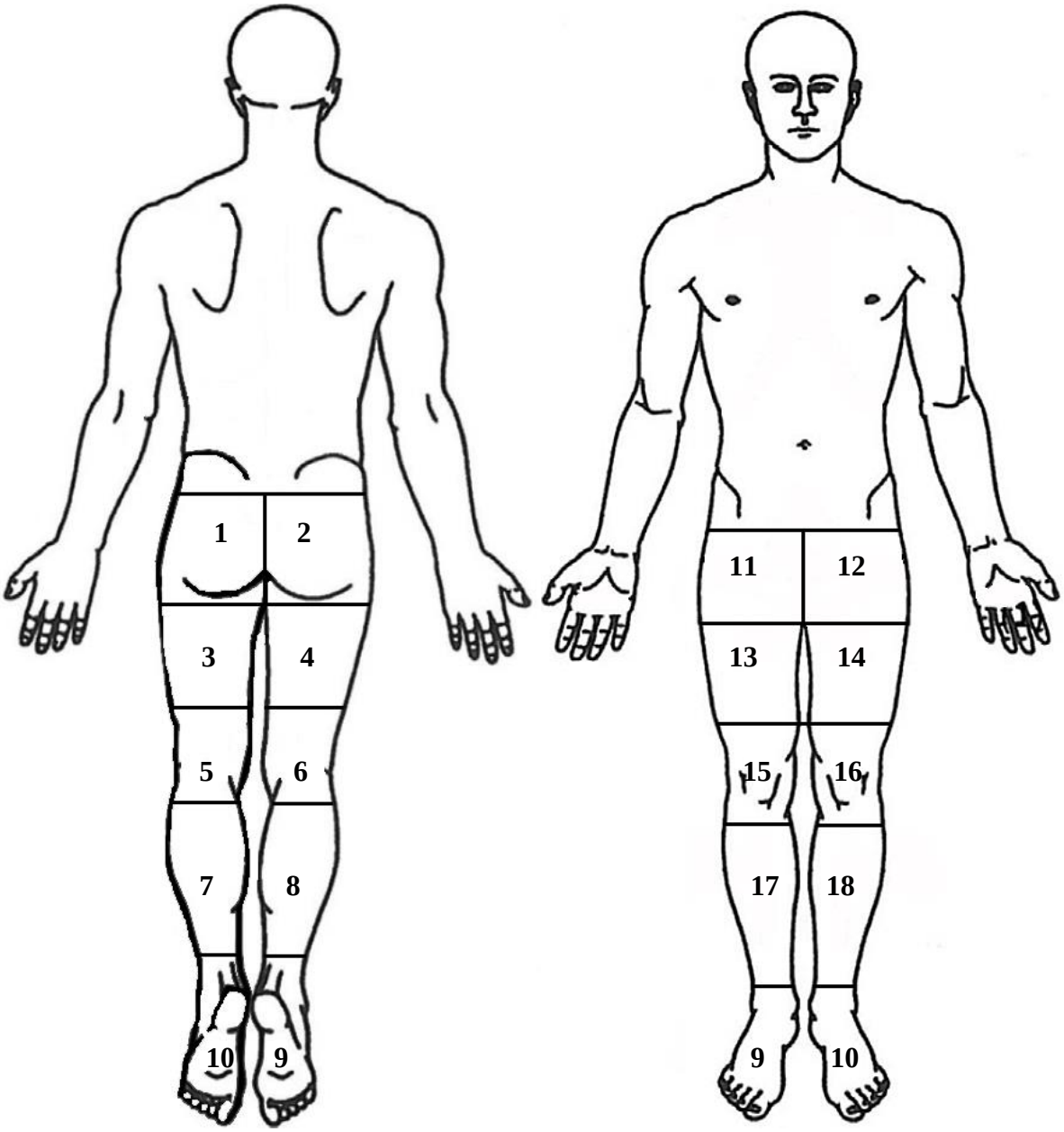
TASK – card sorting task

FFK – full-flexion kneeling

The thick, black horizontal line indicates a 30-second standing break.

Time (min)	Task	Quadrants	VAS
00:00.0	**PRE**		VAS
02:00.0	TASK	1, 2	
02:30.0	FFK		
04:30.0	TASK	3, 4	
05:00.0	FFK		VAS
07:00.0	TASK	2, 4	
07:30.0	FFK		
09:30.0	TASK	1, 3	
10:00.0	FFK		VAS
12:00.0	TASK	4, 1	
12:30.0	FFK		
14:30.0	TASK	3, 2	
15:00.0	FFK		VAS
17:00.0	TASK	2, 3	
17:30.0	FFK		
19:30.0	TASK	1, 4	
20:00.0	FFK		VAS
22:00.0	TASK	2, 1	
22:30.0	FFK		
24:30.0	TASK	4, 2	
25:00.0	FFK		VAS
27:00.0	TASK	4, 3	
27:30.0	FFK		
29:30.0	TASK	3, 1	
30:00.0	FFK		VAS

Appendix F: Pain Diagram



Appendix G: Proprioception Participant Instructions

This is a test of how well you can replicate a target knee angle. A blindfold will be placed over your eyes so that you cannot use vision to assist you. When I say 'start,' I will slowly move your leg to a specific posture. It is very important that you relax all of the muscles of your leg while it is positioned. Once your leg is positioned at the target posture, I will ask if you are 'ready.' When you indicate that you are 'ready,' I will remove my hand and you will hold the posture as still as possible for a count of 5 seconds. During this time, try to focus all of your attention on the knee angle and limit any extraneous movements. After the 5 seconds has passed, I will hold your leg, ask you to relax your muscles, and slowly return your leg to the starting position. After a period of 5 seconds you will be asked to replicate the target knee angle you just held. Once you have reached what you believe to be the target angle, you will indicate to the investigator that you are 'ready' and you will hold the posture as still as possible. Once 5 seconds has passed the investigator will say 'done,' and you may relax your leg and return your leg to the starting position. This procedure will be repeated 3 times.

Appendix H: Kneeling Exposure Marker Visibility for Calculation of APDFs

Table H.1

Percent Joint Angle Visibility during the Kneeling Exposure

	Joint Angle Visibility (%)		
Participant Code	**Hip**	**Knee**	**Ankle**
S02	99.83	100.00	100.00
S03	97.34	96.97	98.06
S06	100.00	99.96	99.96
S08	100.00	100.00	100.00
S09	99.90	100.00	98.43
S10	100.00	100.00	100.00
S11	99.84	98.68	98.68
S12	99.84	100.00	100.00
S13	100.00	100.00	92.78
S14	96.54	99.16	100.00
S16	99.97	99.98	99.98
S17	100.00	97.73	97.67
S18	98.49	98.49	98.49
S19	100.00	100.00	100.00
S20	99.84	100.00	99.84
S21	99.97	99.45	99.45
S22	100.00	99.98	99.98
S24	98.74	99.92	99.92
S25	100.00	100.00	99.96
S26	100.00	100.00	100.00
S27	100.00	99.98	99.98
S28	99.21	99.45	99.41
S29	100.00	98.33	98.33
S30	100.00	100.00	100.00
S31	100.00	100.00	100.00
S32	95.37	95.52	99.76
S33	100.00	100.00	99.99
*S34	72.21	100.00	99.99
S35	98.76	98.76	99.37
S36	99.93	100.00	99.98
S37	99.86	100.00	100.00
S38	98.09	99.93	100.00
S39	99.18	99.93	99.92
S40	99.99	99.99	99.99
S41	99.40	99.98	99.99
S42	100.00	100.00	100.00
S43	100.00	100.00	93.11
S44	100.00	100.00	100.00
S45	99.97	99.97	99.99
S46	99.21	100.00	100.00

Participants with at least one joint that had less than 90% visibility are marked with an asterisk (*).

Appendix I: Heel-Strike Transient Identification

Table I.1

Heel-Strike Transient Prevalence

Participant Code	Shoe Age (mos)	Unloaded			Loaded		
		Pre	Post	30Post	Pre	Post	30Post
S02	20	0	0	0	0	0	0
S03	1	0	0	33.333	20	0	33.333
S06	3	20	0	0	0	0	0
S08	9	16.667	25	0	0	33.333	0
*S09	48	0	50	25	20	100	60
S10	12	0	0	0	0	0	0
S11	20	0	0	0	0	0	0
S12	20	20	0	0	0	0	0
S13	7	0	0	0	0	0	0
S14	4	0	0	0	20	0	0
*S16	12	40	100	66.667	0	25	20
S17	6	0	0	0	0	0	0
S18	12	0	0	0	0	0	0
S19	6	0	0	0	0	0	0
S20	6	0	0	25	16.667	0	0
S21	7	0	0	0	0	0	0
S22	16	0	0	0	0	0	0
◊S24	24	60	0	75	0	0	0
S25	72	0	0	0	0	0	0
S26	2	0	0	0	0	0	0
S27	48	20	0	0	0	0	0
S28	2	0	0	0	0	0	0
S29	0	0	0	0	0	0	0
S30	8	0	0	0	0	0	0
S31	18	0	0	0	0	0	0
S32	24	0	0	0	0	0	0
S33	12	0	0	0	0	0	0
S34	-	0	0	0	0	0	0
◊S35	24	50	50	25	20	0	0
S36	9	0	0	0	0	0	0
S37	24	0	0	0	20	0	20
S38	8	0	0	0	0	0	0
S39	36	0	0	0	0	0	0
S40	36	0	0	0	0	0	40
S41	12	0	0	50	0	0	0
S42	24	0	0	16.667	0	0	0
S43	8	40	0	0	0	0	0
S44	18	0	0	0	0	0	0
S45	12	0	0	0	0	0	0
S46	11	0	0	0	0	0	0

Prevalence is reported as a % of the total trials in each condition that an HST occurred.

* Participants who demonstrated a change from < 50% to ≥50% HST prevalence between pre- and post-measures in at least one of the loading conditions

◊ Participants who were classified as heel-strikers based on ≥ 50% prevalence of HSTs at baseline in the unloaded condition

Appendix J: Full ANOVA Results

Below are the full ANOVA results. Only significant main effects that showed significance for the kneeling exposure, sex, or squat speed were explored in the document. When differences are significant based on a threshold of $p < .05$, values are underlined. Greenhouse-Geisser (G-G) corrected p-values are reported when Mauchly's test indicated that sphericity was violated.

Variable Legend:

SEX: male or female
TIME: pre, post, and 30 minutes post
TRIAL: 1, 2, and 3
LOAD: loaded and unloaded
DIRECTION: ascending and descending
SPEED: slow and fast

Gait

Table J.1

Results for Peak Knee Adduction Moment

VARIABLE	F VALUE	PR > F	PR > F (G-G)
SEX	12.27	.0012	-
TIME	3.07	.0523	-
LOAD	113.56	< .0001	-
TRIAL	0.42	.6569	-
TIME*SEX	0.44	.6483	-
LOAD*SEX	0.13	.7185	-
TRIAL*SEX	0.41	.6628	-
TIME*LOAD	0.79	-	.4396
TIME*TRIAL	0.90	.4628	-
LOAD*TRIAL	0.50	.6057	-
TIME*LOAD*SEX	3.17	.0474	-
TIME*TRIAL*SEX	2.45	.0482	-
LOAD*TRIAL*SEX	0.37	.6932	-
TIME*LOAD*TRIAL	0.98	.4229	-
TIME*LOAD*TRIAL*SEX	0.77	.4229	-

Table J.2

Results for Peak Vertical Rate of Loading

VARIABLE	F VALUE	PR > F	PR > F (G-G)
SEX	1.72	.1970	-
TIME	1.96	-	.1600
LOAD	111.40	< .0001	-
TRIAL	0.88	-	.4026
TIME*SEX	1.03	.3610	-
LOAD*SEX	0.23	.6359	-
TRIAL*SEX	0.00	-	.9926
TIME*LOAD	0.47	.6260	-
TIME*TRIAL	0.92	.4522	-
LOAD*TRIAL	0.53	.5922	-
TIME*LOAD*SEX	0.19	.8253	-
TIME*TRIAL*SEX	0.14	.9651	-
LOAD*TRIAL*SEX	0.77	.4649	-
TIME*LOAD*TRIAL	1.57	-	.1939
TIME*LOAD*TRIAL*SEX	0.88	-	.4609

Table J.3

Results for Vastus Medialis Onset

VARIABLE	F VALUE	PR > F	PR > F (G-G)
SEX	0.04	.8353	-
TIME	10.33	-	.0003
LOAD	5.38	.0258	-
TRIAL	6.74	.0020	-
TIME*SEX	0.52	-	.5726
LOAD*SEX	0.82	.3716	-
TRIAL*SEX	0.3	.7403	-
TIME*LOAD	4.61	.0129	-
TIME*TRIAL	0.3	.8783	-
LOAD*TRIAL	0.11	.8992	-
TIME*LOAD*SEX	0.34	.7126	-
TIME*TRIAL*SEX	1.61	.1121	-
LOAD*TRIAL*SEX	0.95	.3914	-
TIME*LOAD*TRIAL	2.77	.0293	-
TIME*LOAD*TRIAL*SEX	0.87	.4810	-

Table J.4

Results for Vastus Medialis Magnitude

VARIABLE	F VALUE	PR > F	PR > F (G-G)
SEX	3.32	.0766	-
TIME	2.61	.0803	-
LOAD	41.02	< .0001	-
TRIAL	1.10	.3371	-
TIME*SEX	1.82	.1696	-
LOAD*SEX	0.18	.6760	-
TRIAL*SEX	0.57	.5671	-
TIME*LOAD	0.32	-	.6850
TIME*TRIAL	0.55	-	.6487
LOAD*TRIAL	0.36	.7014	-
TIME*LOAD*SEX	2.14	-	.1351
TIME*TRIAL*SEX	0.34	-	.7943
LOAD*TRIAL*SEX	0.42	.6602	-
TIME*LOAD*TRIAL	1.39	-	.2491
TIME*LOAD*TRIAL*SEX	0.73	-	.5447

Squat Transitions

Table J.5

Results for Peak Deviation from the Plane

VARIABLE	F VALUE	PR > F	PR > F (G-G)
SEX	0.23	.6353	-
TIME	5.44	-	.0096
SPEED	0.09	.7685	-
DIRECTION	3.88	.0562	-
TRIAL	9.09	-	.0010
TIME*SEX	0.29	-	.7119
SPEED*SEX	5.79	.0210	-
DIRECTION*SEX	0.28	.6009	-
TRIAL*SEX	1.54	-	.2237
TIME*SPEED	0.31	.7362	-
TIME*DIRECTION	0.39	-	.6307
SPEED*DIRECTION	2.01	.1644	-
TIME*TRIAL	0.71	-	.5446
SPEED*TRIAL	1.98	0.1458	-
DIRECTION*TRIAL	0.88	0.4177	-
TIME*SPEED*SEX	1.34	.2679	-
TIME*DIRECTION*SEX	1.38	-	.2559
SPEED*DIRECTION*SEX	0.19	.6620	-
TIME*TRIAL*SEX	1.09	-	.3534
SPEED*TRIAL*SEX	0.02	.9818	-
DIRECTION*TRIAL*SEX	2.13	.1253	-

VARIABLE	F VALUE	PR > F	PR > F (G-G)
TIME*SPEED*DIRECTION	0.30	.7438	-
TIME*SPEED*TRIAL	2.68	-	.0449
TIME*DIRECTION*TRIAL	1.10	-	.3485
SPEED*DIRECTION*TRIAL	1.57	.2147	-
TIME*SPEED*DIRECTION*SEX	2.83	.0654	-
TIME*SPEED*TRIAL*SEX	2.17	-	.0884
TIME*DIRECTION*TRIAL*SEX	1.2	-	.3117
SPEED*DIRECTION*TRIAL*SEX	0.75	0.4773	-
TIME*SPEED*DIRECTION*TRIAL	0.49	-	.6885
TIME*SPEED*DIRECTION*TRIAL*SEX	0.38	-	.7634

Table J.6

Results for Mean Deviation from the Plane

VARIABLE	F VALUE	PR > F	PR > F (G-G)
SEX	0.17	.6853	-
TIME	5.82	.0044	-
SPEED	9.85	.0033	-
DIRECTION	1.12	.2969	-
TRIAL	0.72	.4885	-
TIME*SEX	0.12	.8850	-
SPEED*SEX	1.76	.1923	-
DIRECTION*SEX	0.17	.6849	-
TRIAL*SEX	1.43	.2462	-
TIME*SPEED	0.21	-	.7697
TIME*DIRECTION	3.00	.0558	-
SPEED*DIRECTION	7.06	.0114	-
TIME*TRIAL	0.49	.7465	-
SPEED*TRIAL	0.21	.8076	-
DIRECTION*TRIAL	5.82	.0045	-
TIME*SPEED*SEX	1.62	-	.2086
TIME*DIRECTION*SEX	0.24	.7854	-
SPEED*DIRECTION*SEX	1.72	.1971	-
TIME*TRIAL*SEX	0.41	.8027	-
SPEED*TRIAL*SEX	1.00	.3730	-
DIRECTION*TRIAL*SEX	0.90	.4096	-
TIME*SPEED*DIRECTION	0.18	.8344	-
TIME*SPEED*TRIAL	0.58	.6794	-
TIME*DIRECTION*TRIAL	1.37	.2456	-
SPEED*DIRECTION*TRIAL	3.03	.0544	-
TIME*SPEED*DIRECTION*SEX	1.36	.2638	-
TIME*SPEED*TRIAL*SEX	1.64	.1682	-
TIME*DIRECTION*TRIAL*SEX	1.35	.2557	-
SPEED*DIRECTION*TRIAL*SEX	2.12	.1274	-
TIME*SPEED*DIRECTION*TRIAL	2.90	.0237	-
TIME*SPEED*DIRECTION*TRIAL*SEX	1.64	.1661	-

Proprioception

Table J.7

Results for Average Absolute Angular Error

VARIABLE	F VALUE	PR > F	PR > F (G-G)
SEX	5.47	.0246	-
TIME	0.01	-	.9882
TRIAL	8.82	.0004	-
TIME*SEX	0.86	.4268	-
TRIAL*SEX	1.99	.1436	-
TIME*TRIAL	1.17	-	.3266
TIME*TRIAL*SEX	0.15	.9638	-

Appendix K: Tri-Axial Knee Joint Angles and Knee Deviation during Squat Transitions

Individual results for frontal plane knee motion (measured as the deviation of the knee joint center with respect to a body-fixed plane) were not necessarily associated with knee joint kinematics for all participants. For example, an increase in knee abduction angle following the kneeling exposure did not necessarily result in an increase in medial frontal plane knee motion. Based on the understanding that knee kinematics, including the knee abduction angle, may be a risk factor for knee injury (Hewett et al., 2005), but was not necessarily captured by the measure of frontal plane deviation, individual participant curves for both frontal plane motion and the knee joint angles in three-dimensions are provided in this appendix (graphs commence on the following page).

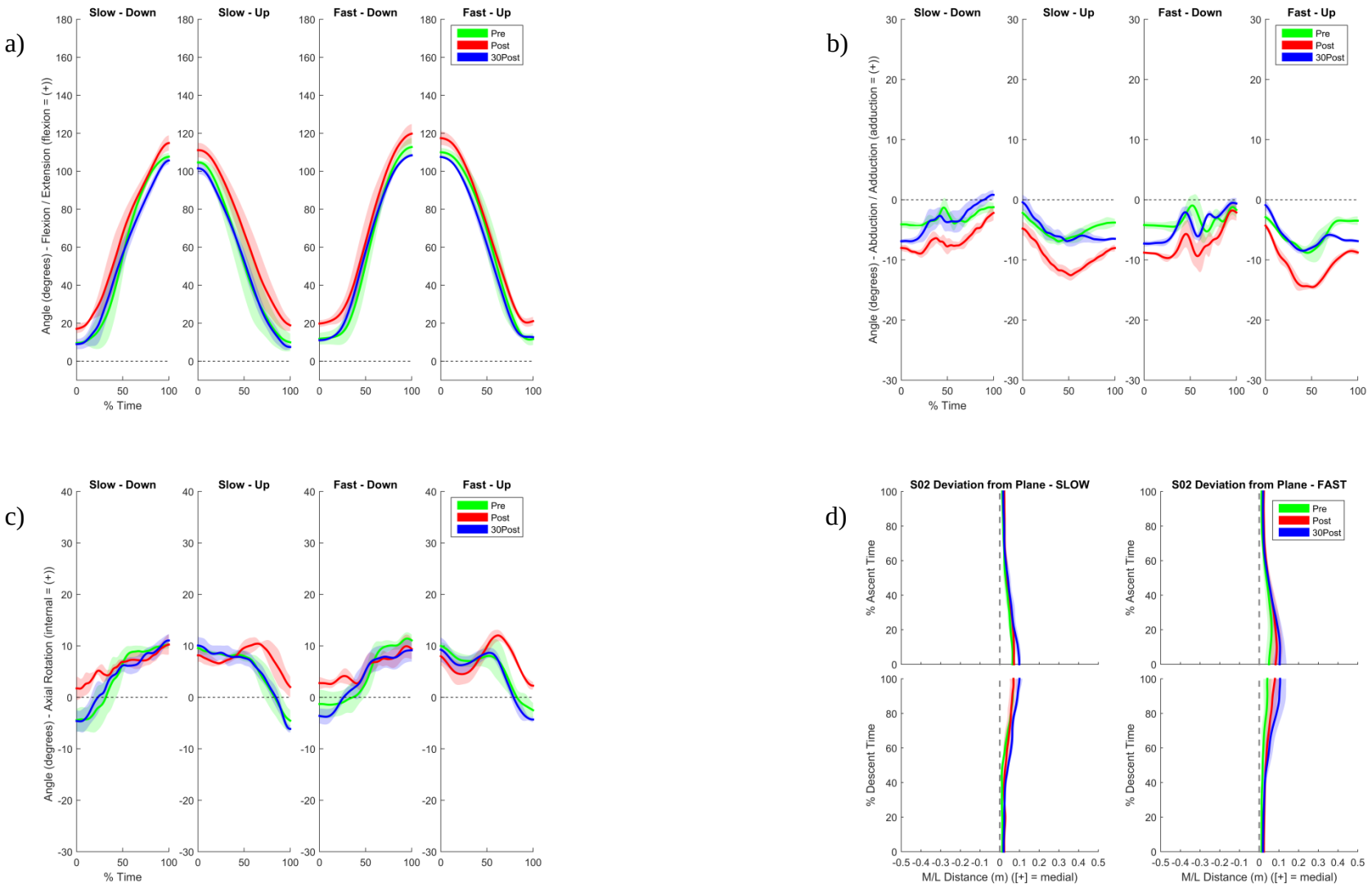


Figure K.1 S02 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

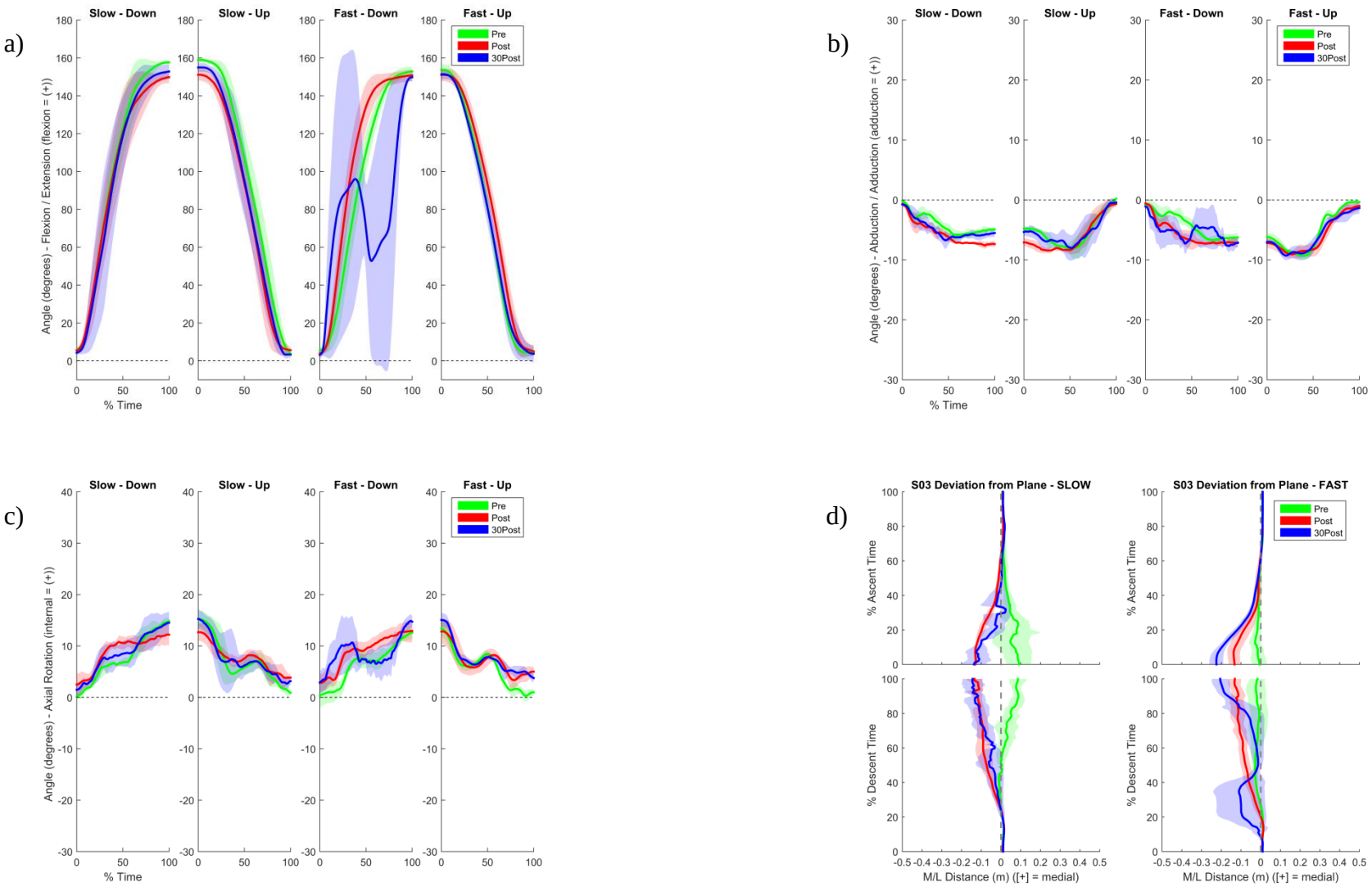


Figure K.2 S03 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

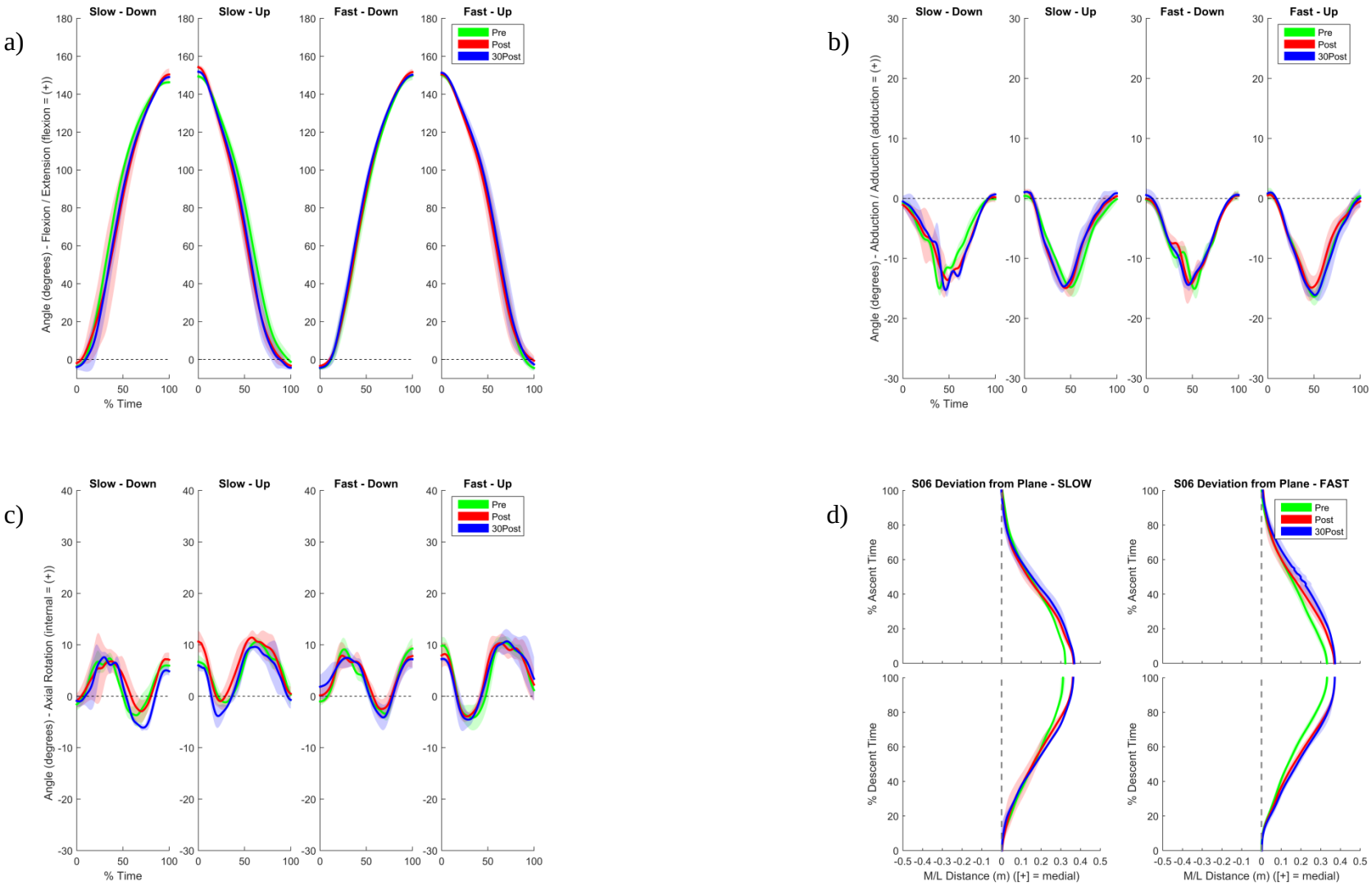


Figure K.3 S06 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

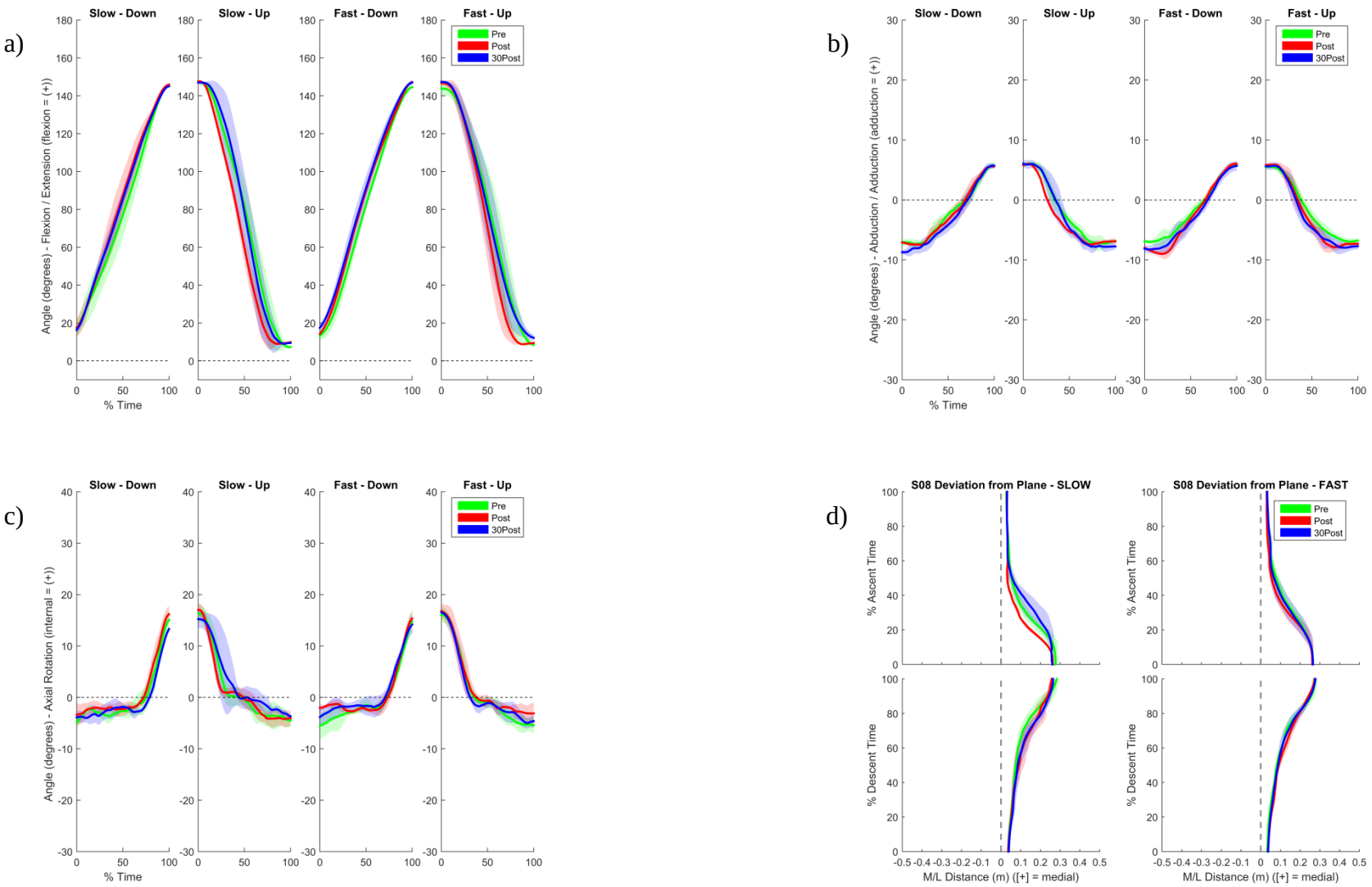


Figure K.4 S08 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

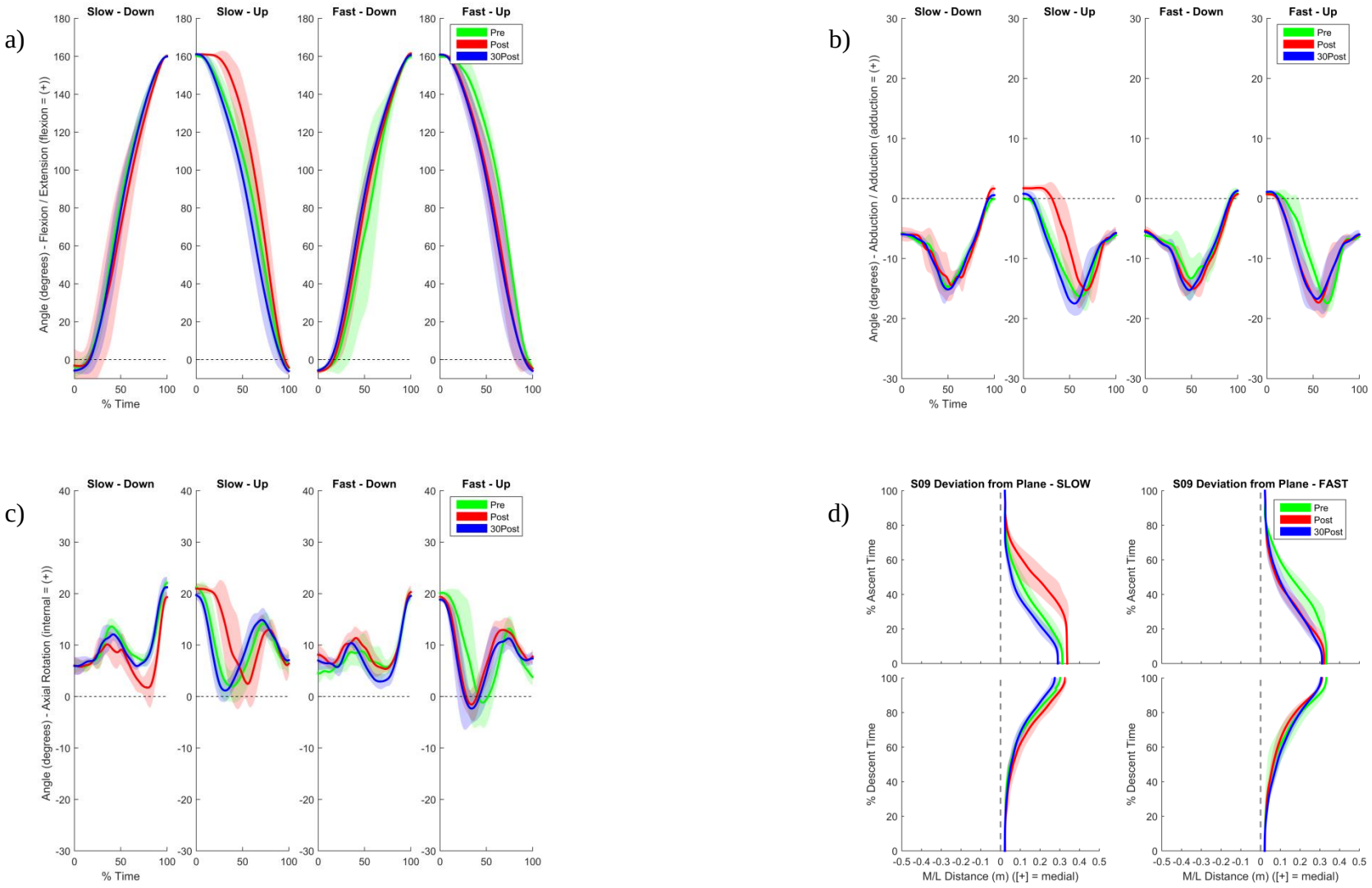


Figure K.5 S09 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

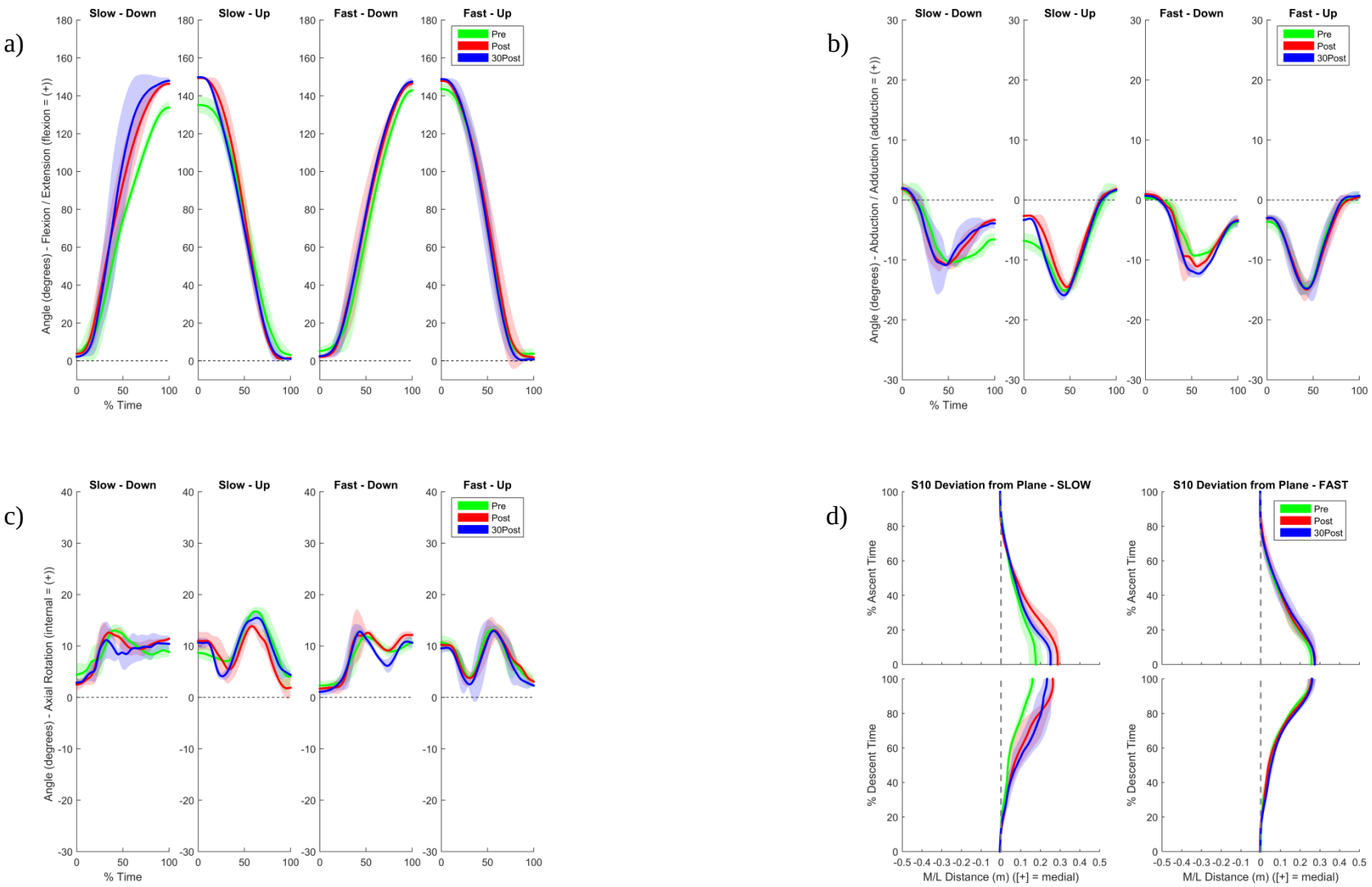


Figure K.6 S10 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

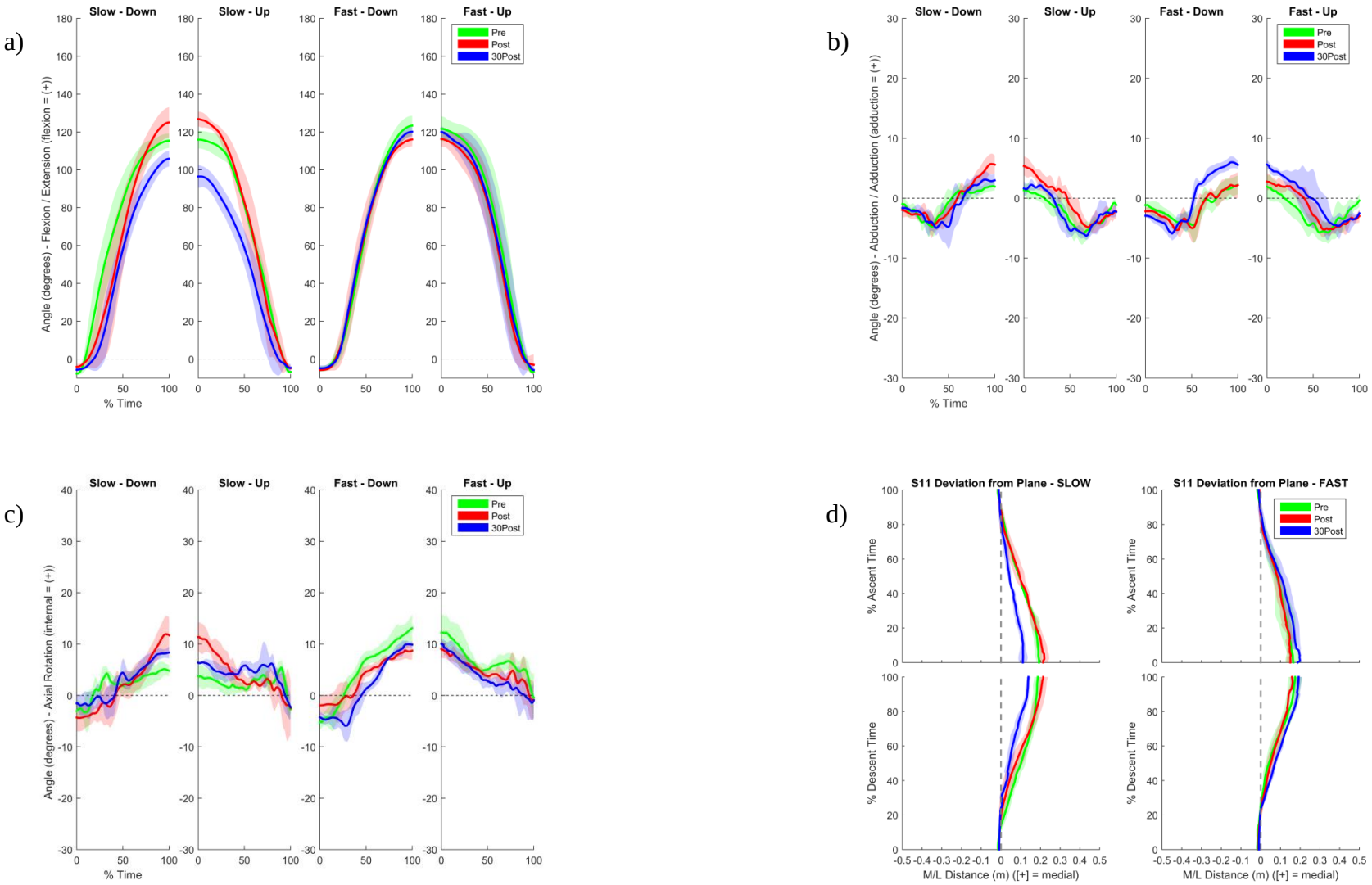


Figure K.7 S11 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

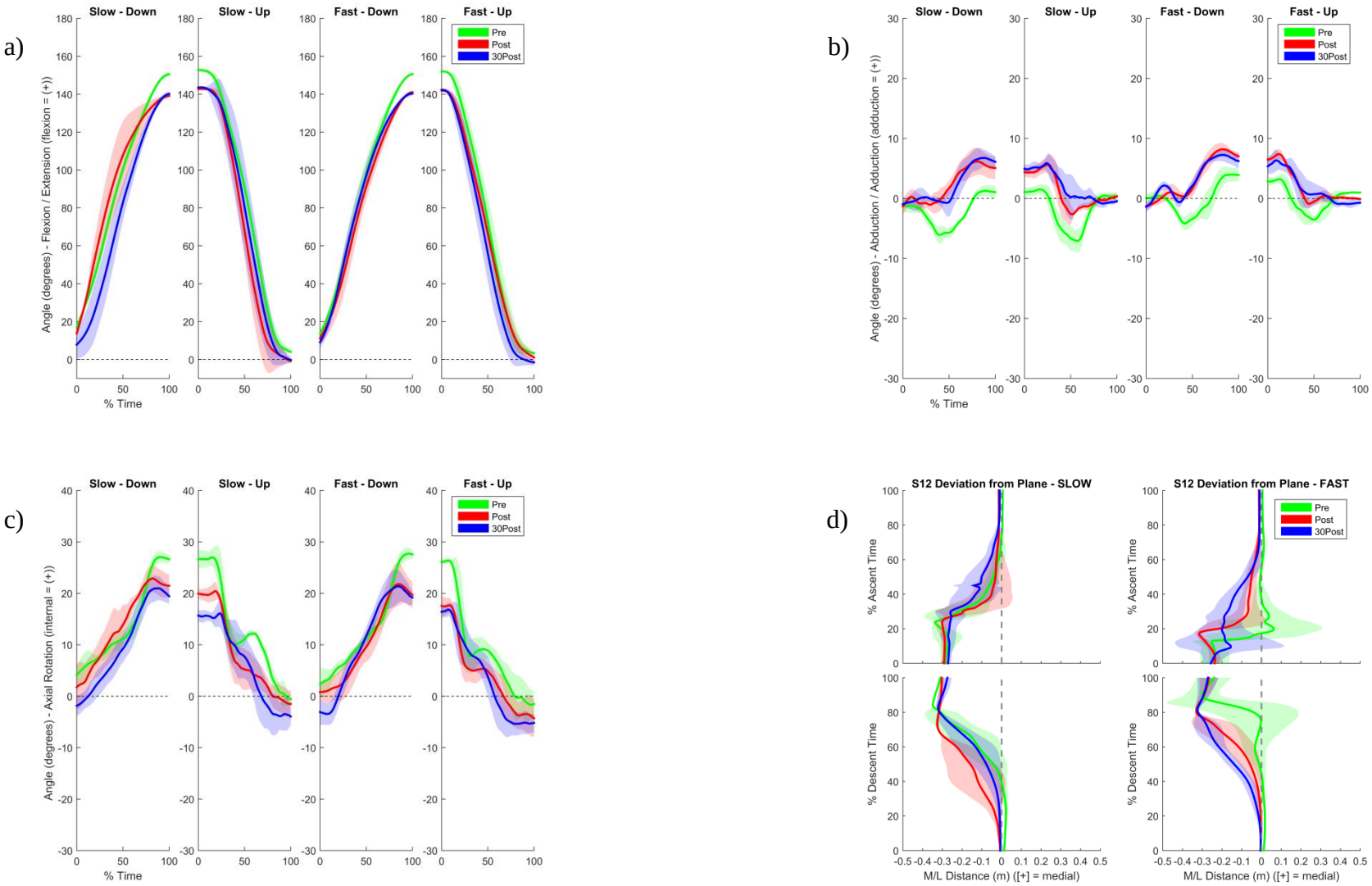


Figure K.8 S12 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

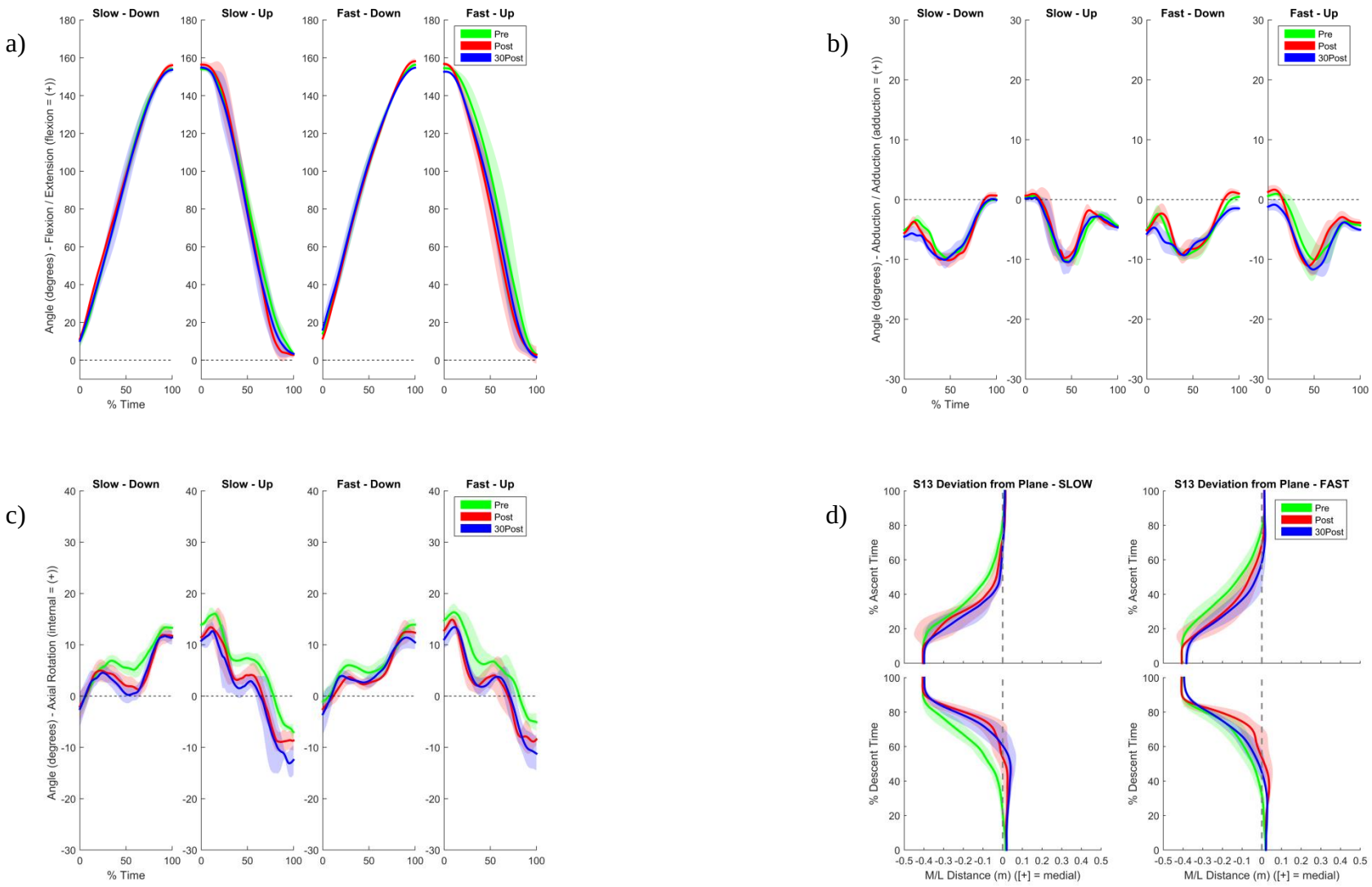


Figure K.9 S13 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

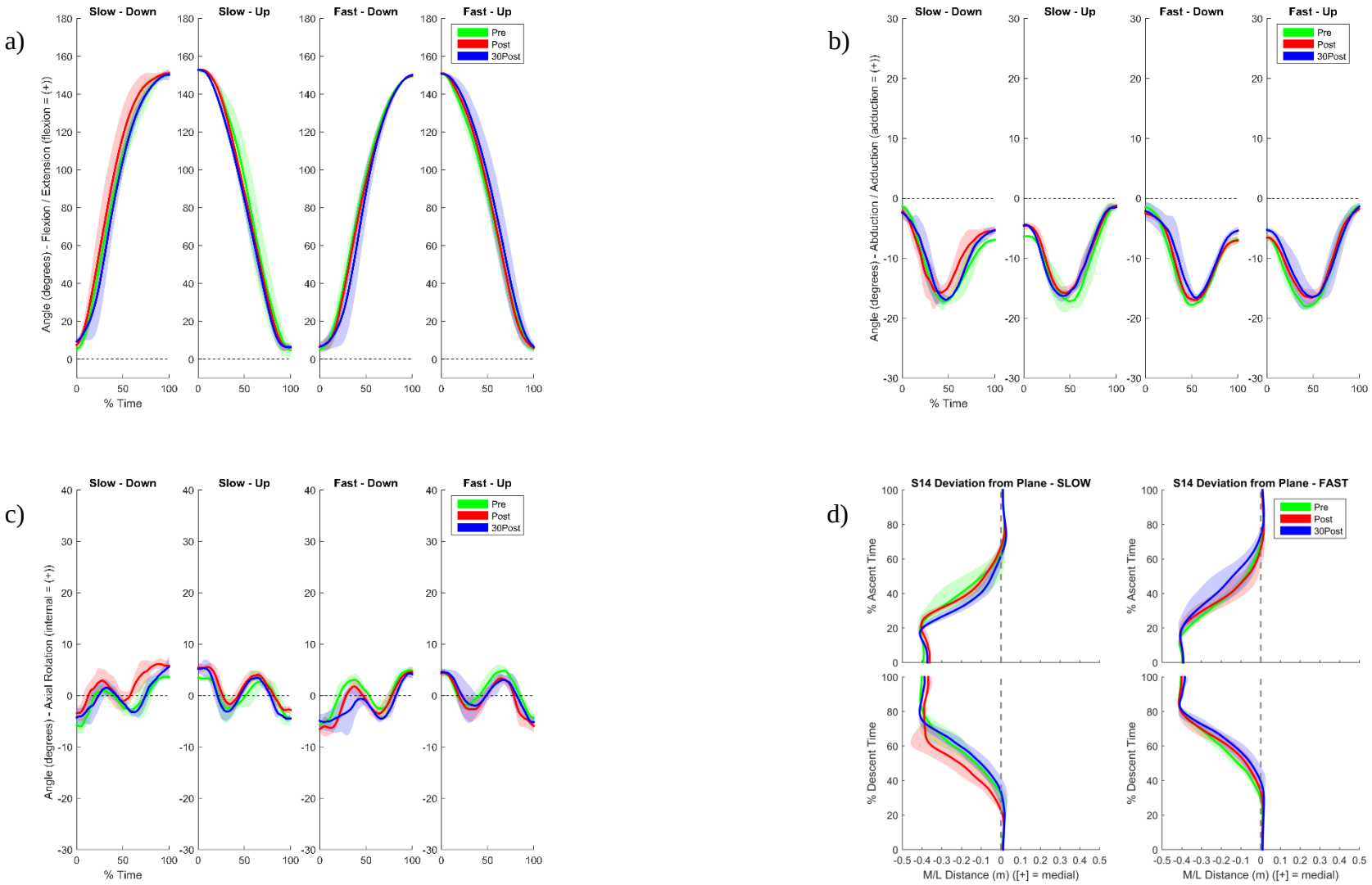


Figure K.10 S14 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

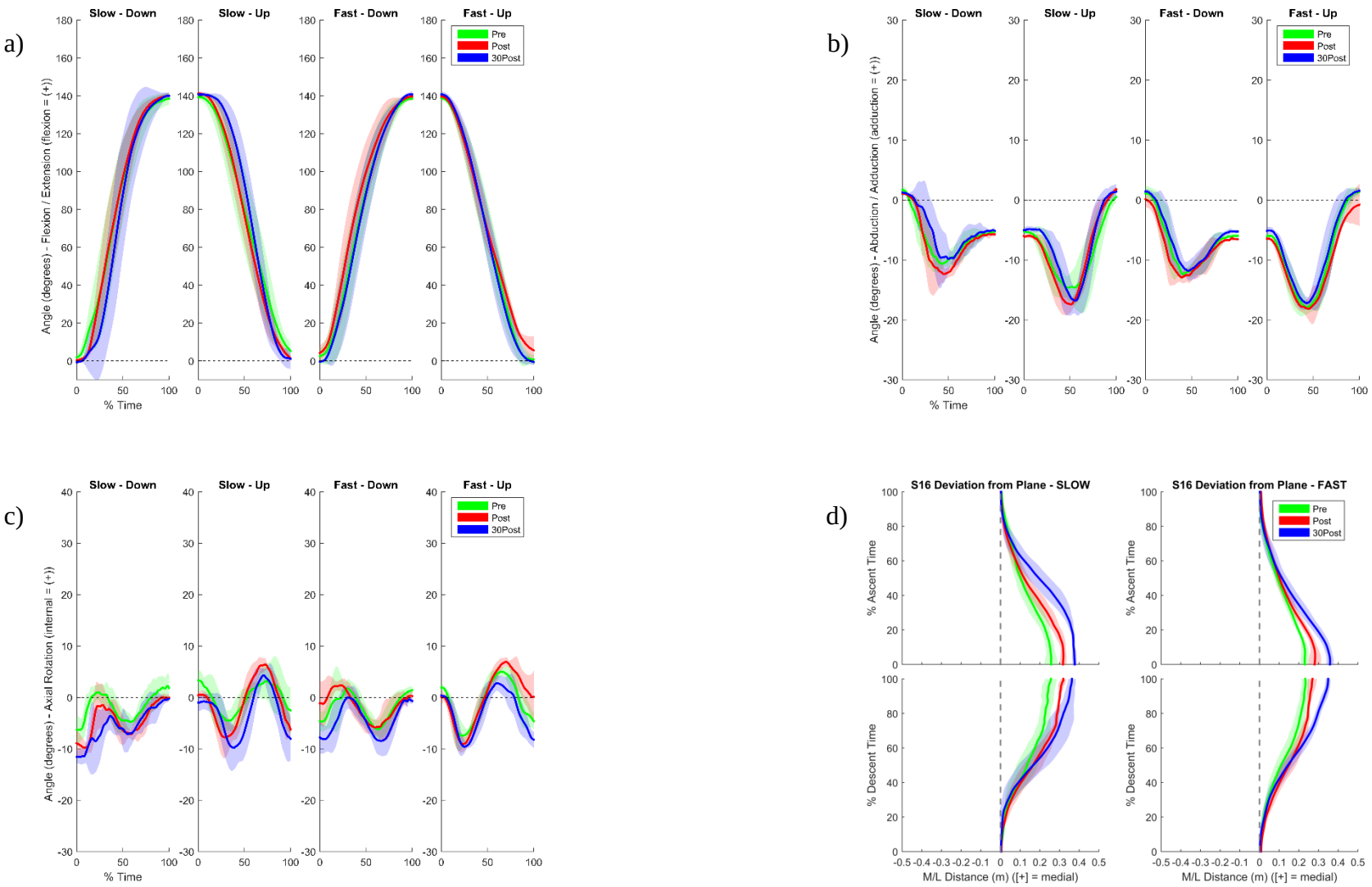


Figure K.11 S16 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

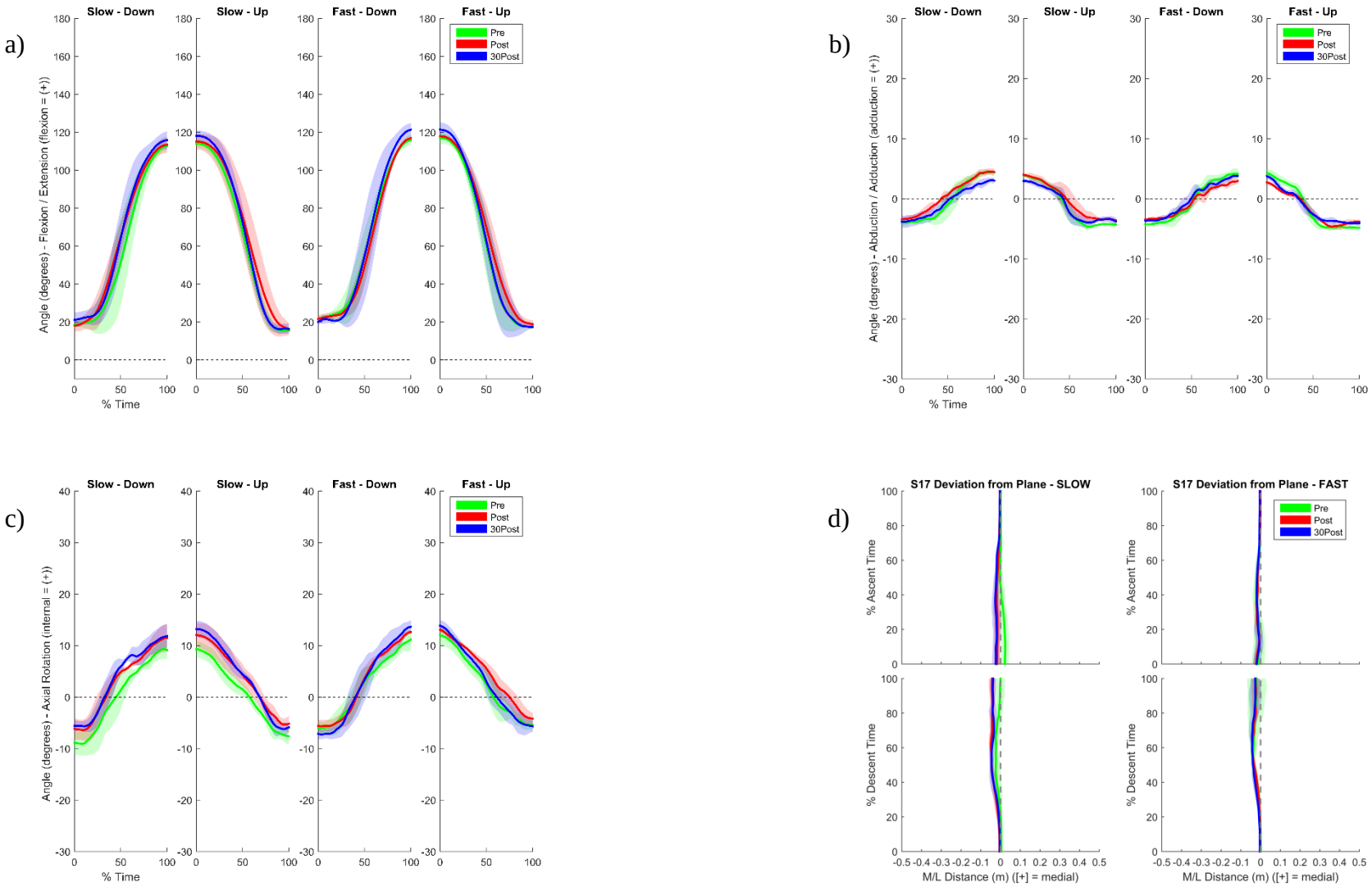


Figure K.12 S17 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

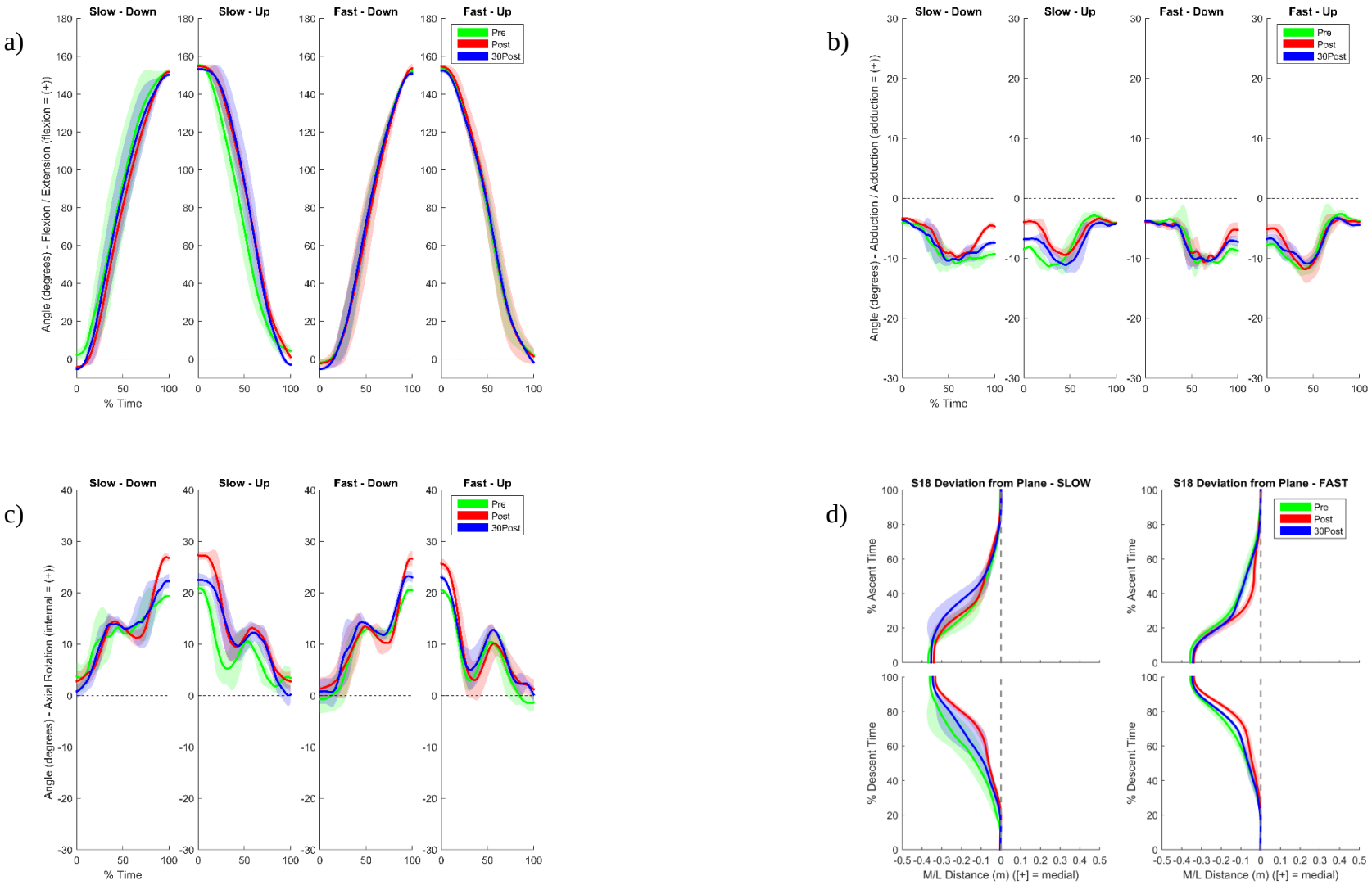


Figure K.13 S18 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

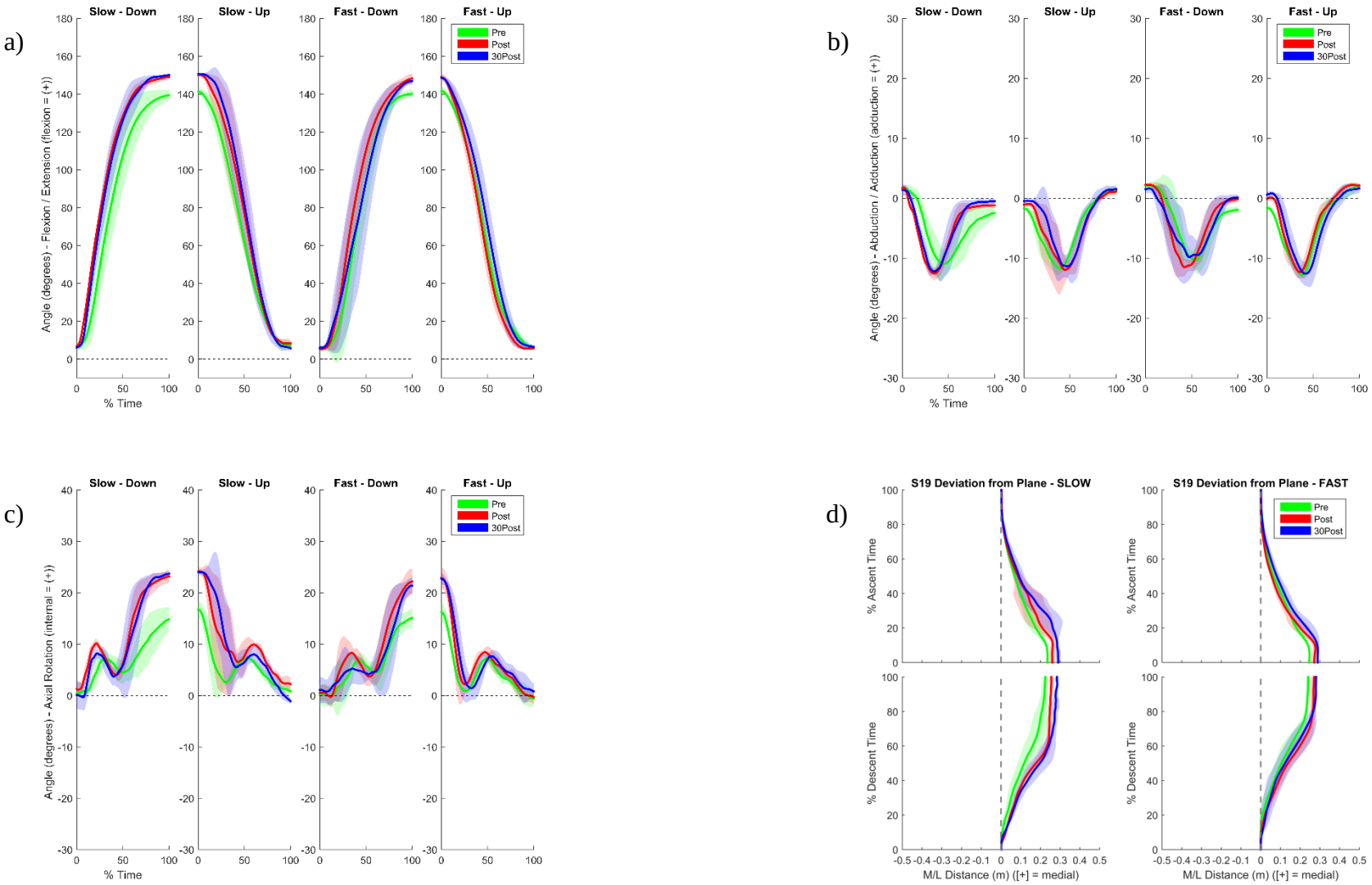


Figure K.14 S19 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

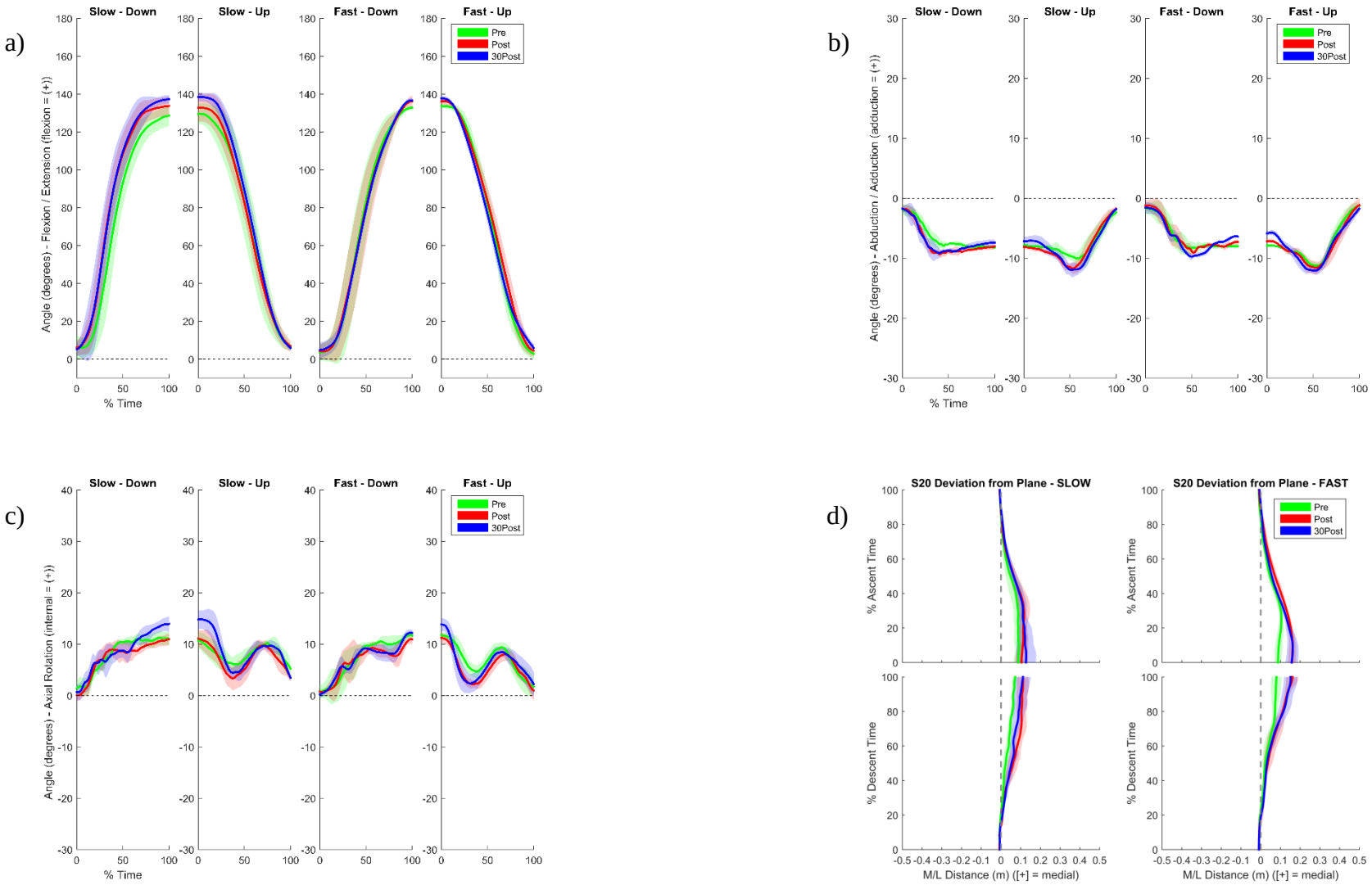


Figure K.15 S20 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

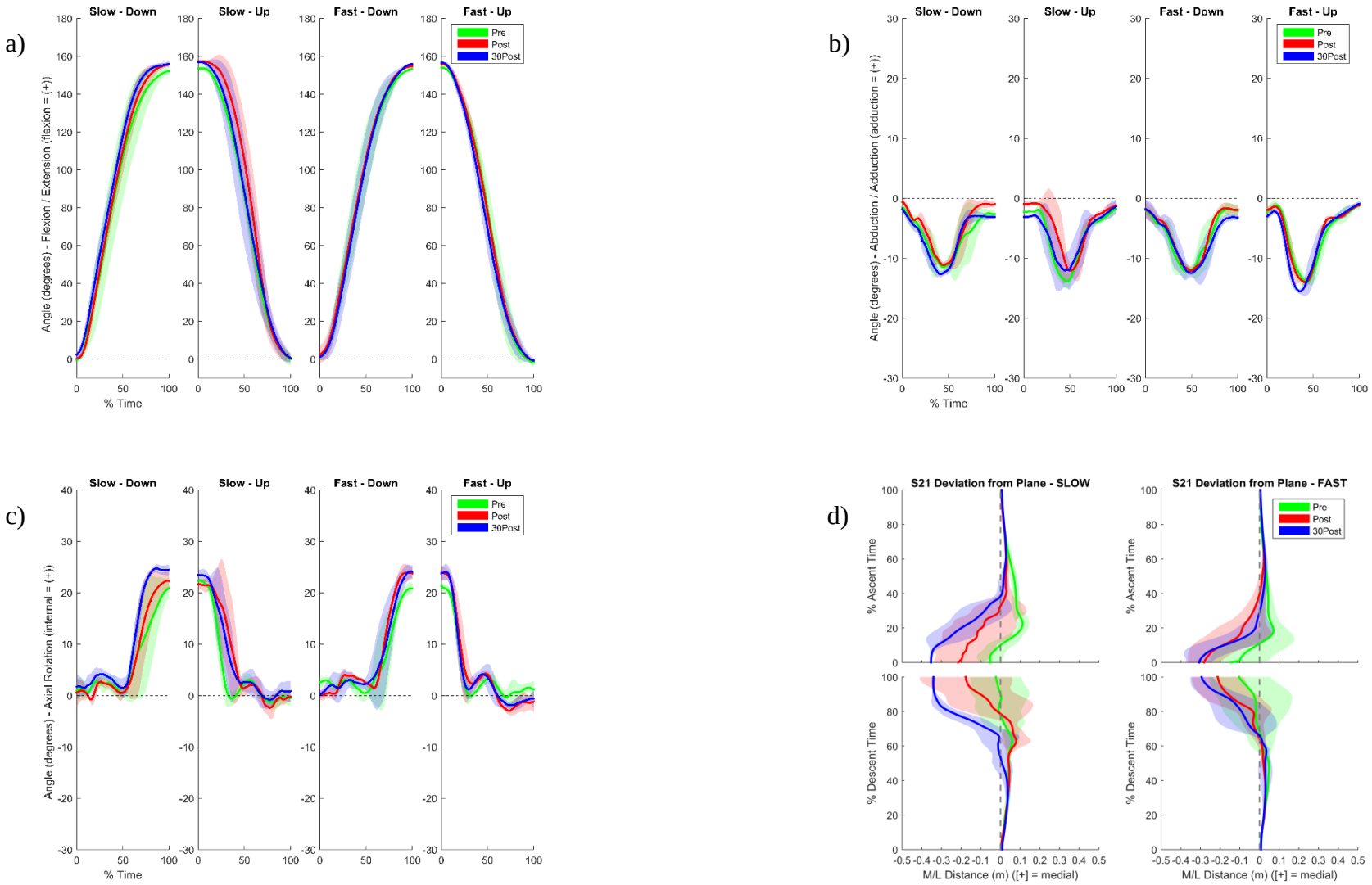


Figure K.16 S21 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

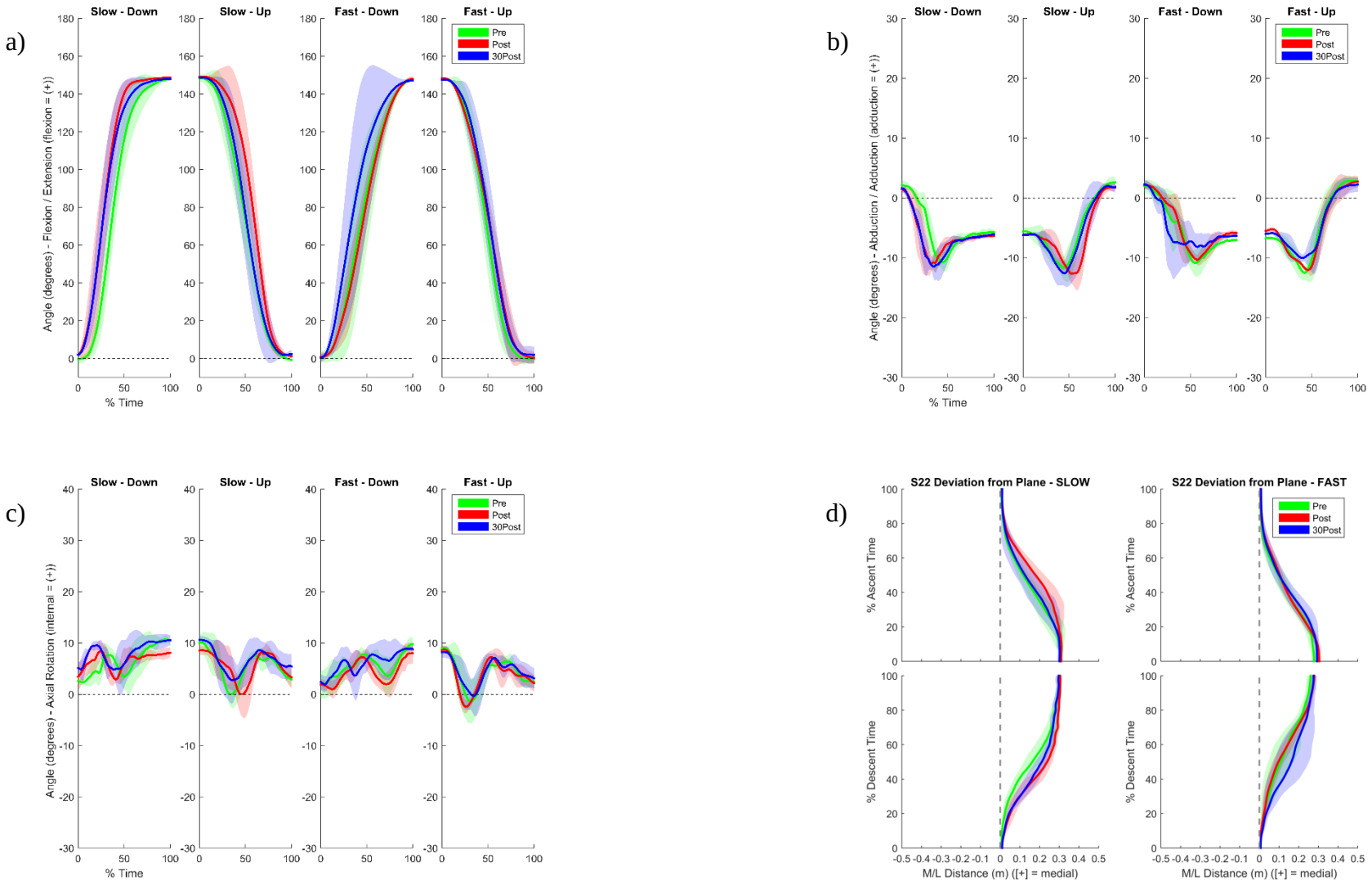


Figure K.17 S22 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

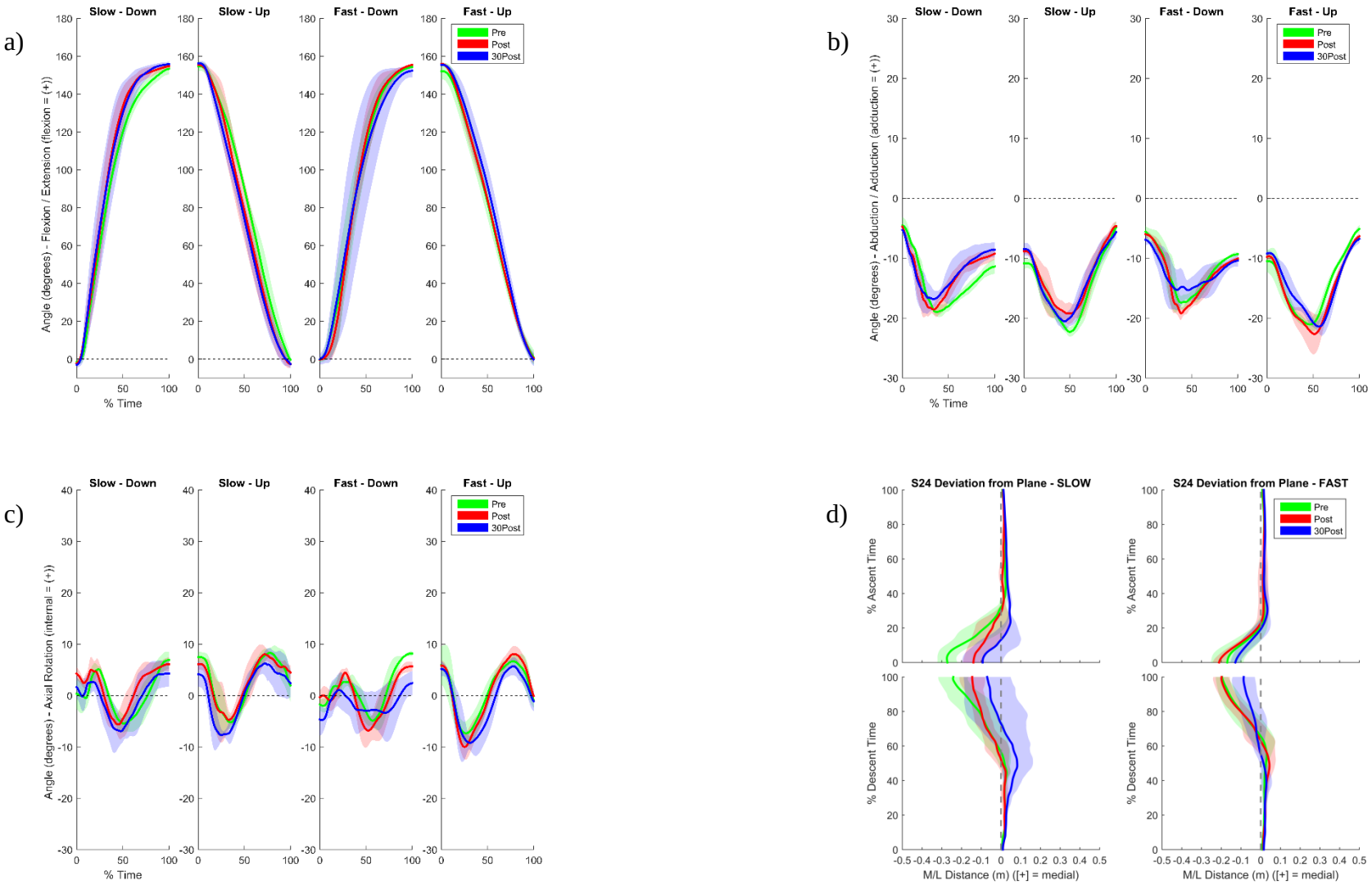


Figure K.18 S24 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

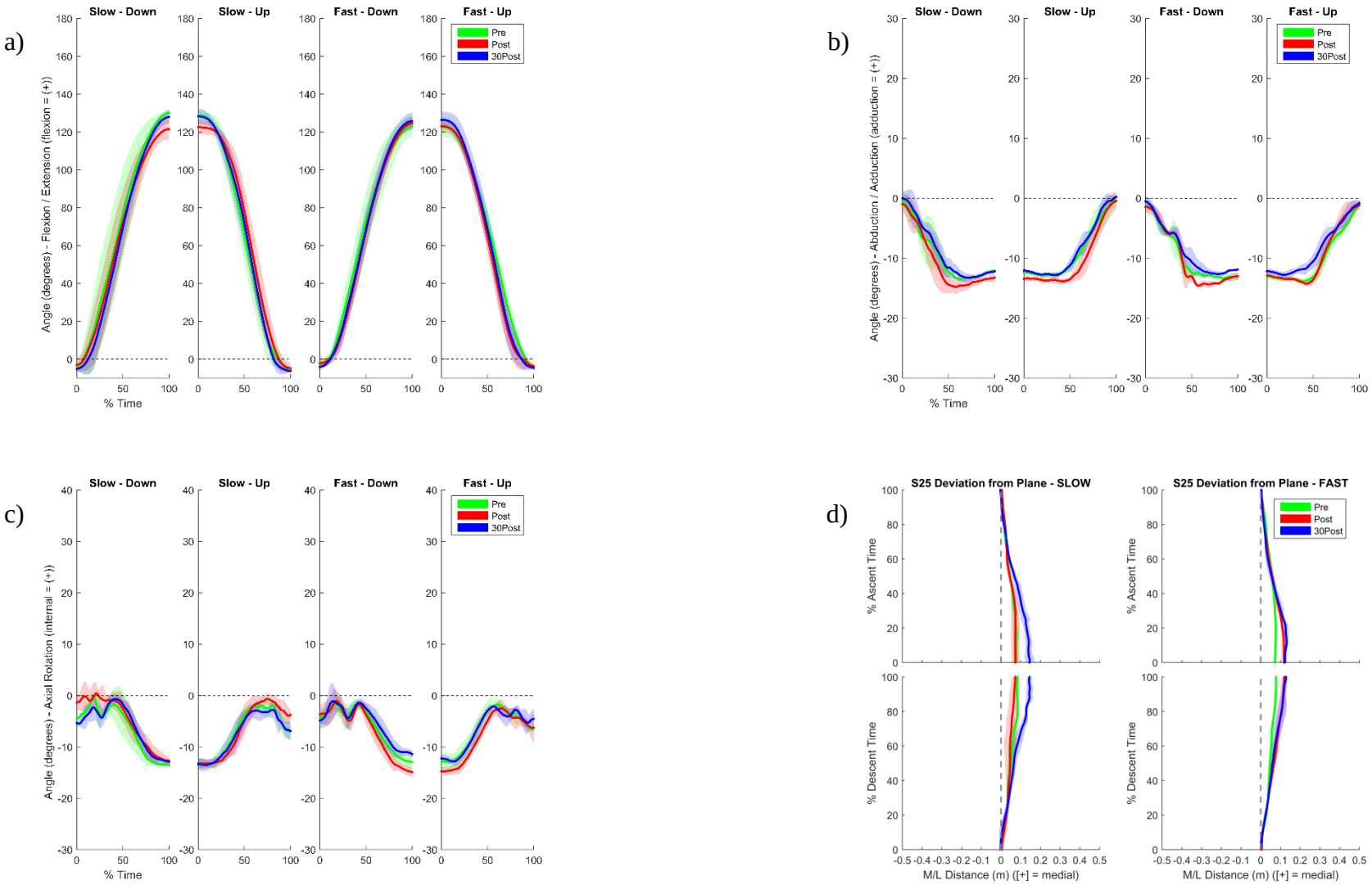


Figure K.19 S25 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

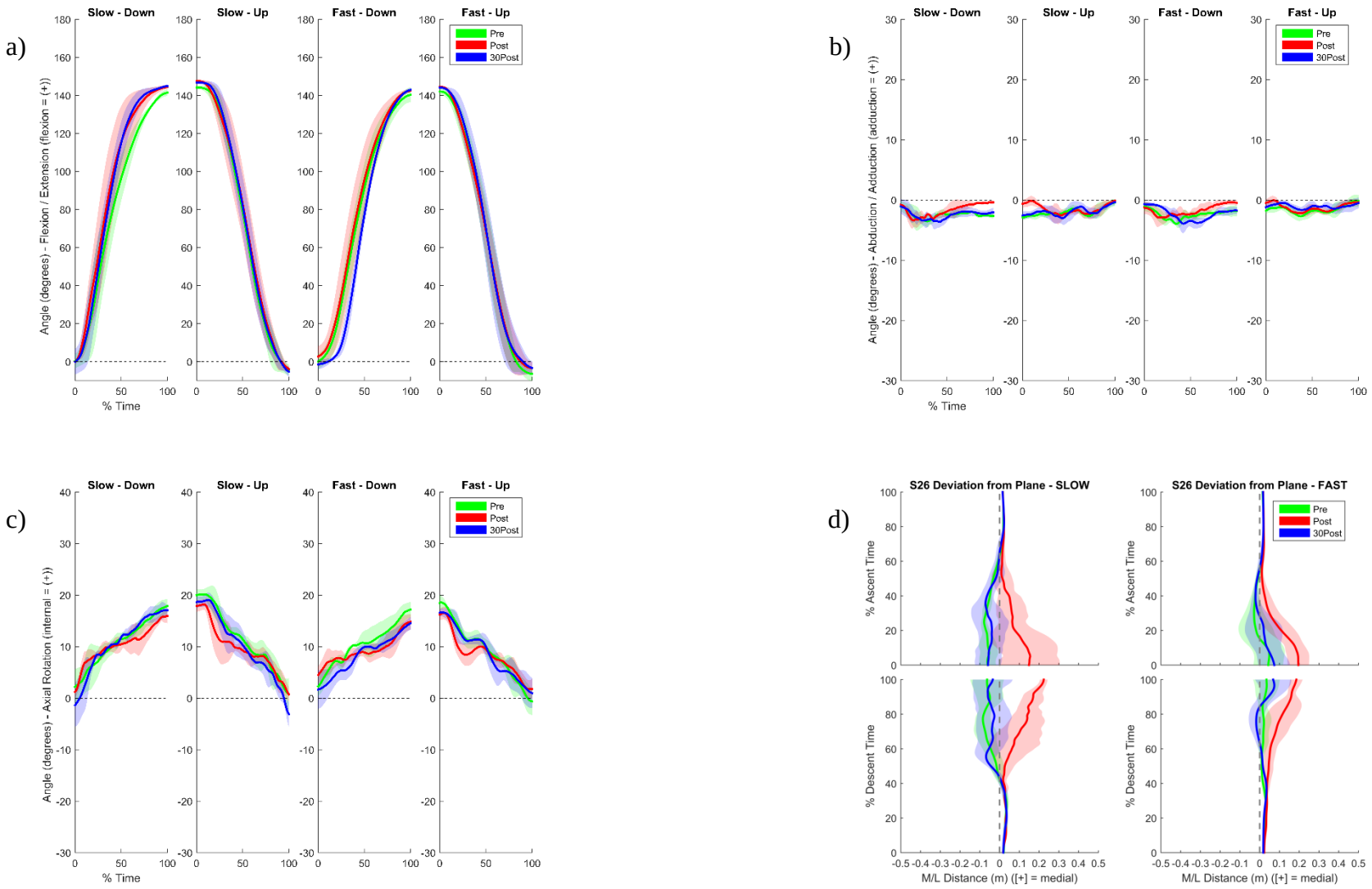


Figure K.20 S26 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

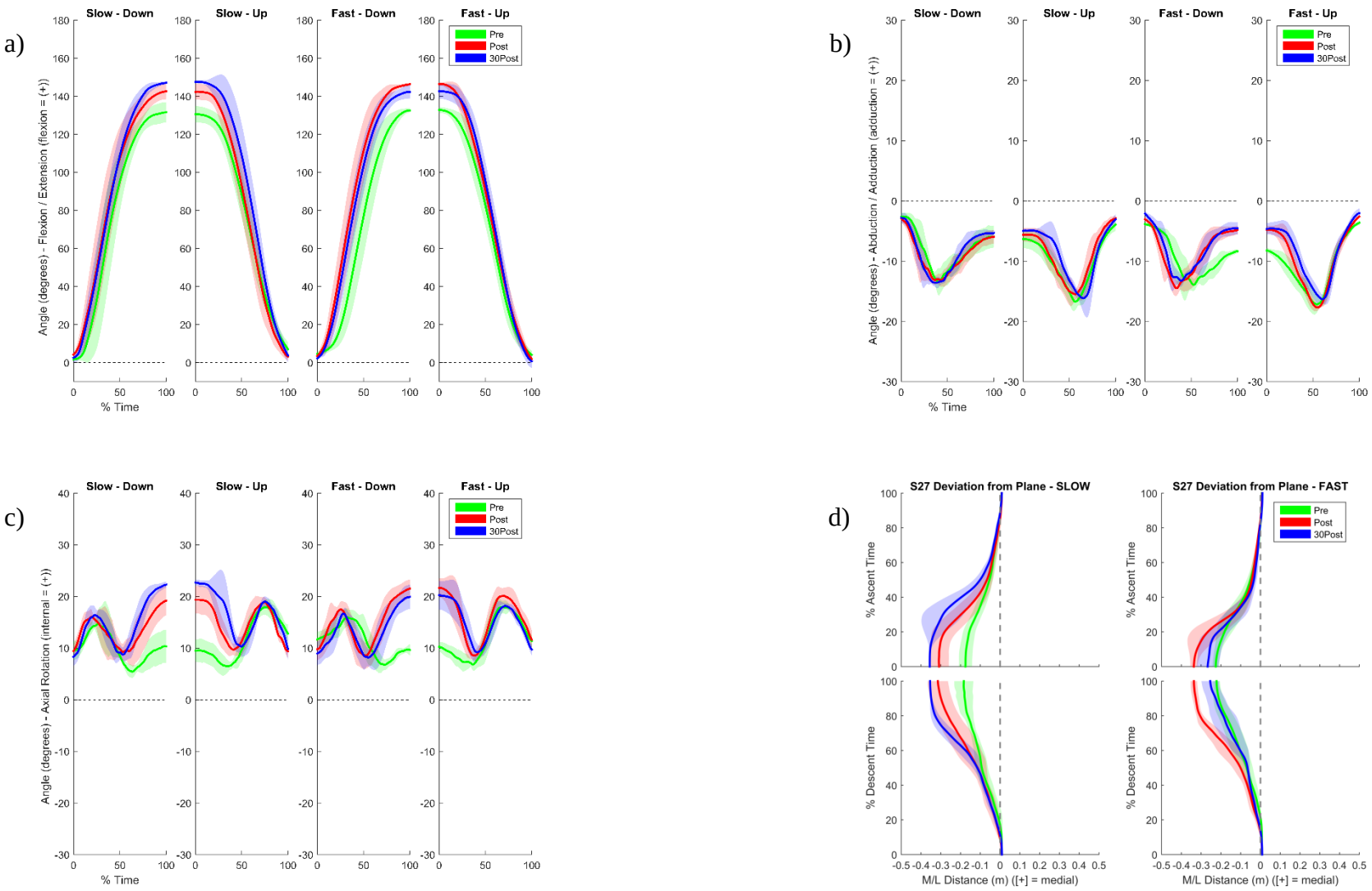


Figure K.21 S27 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

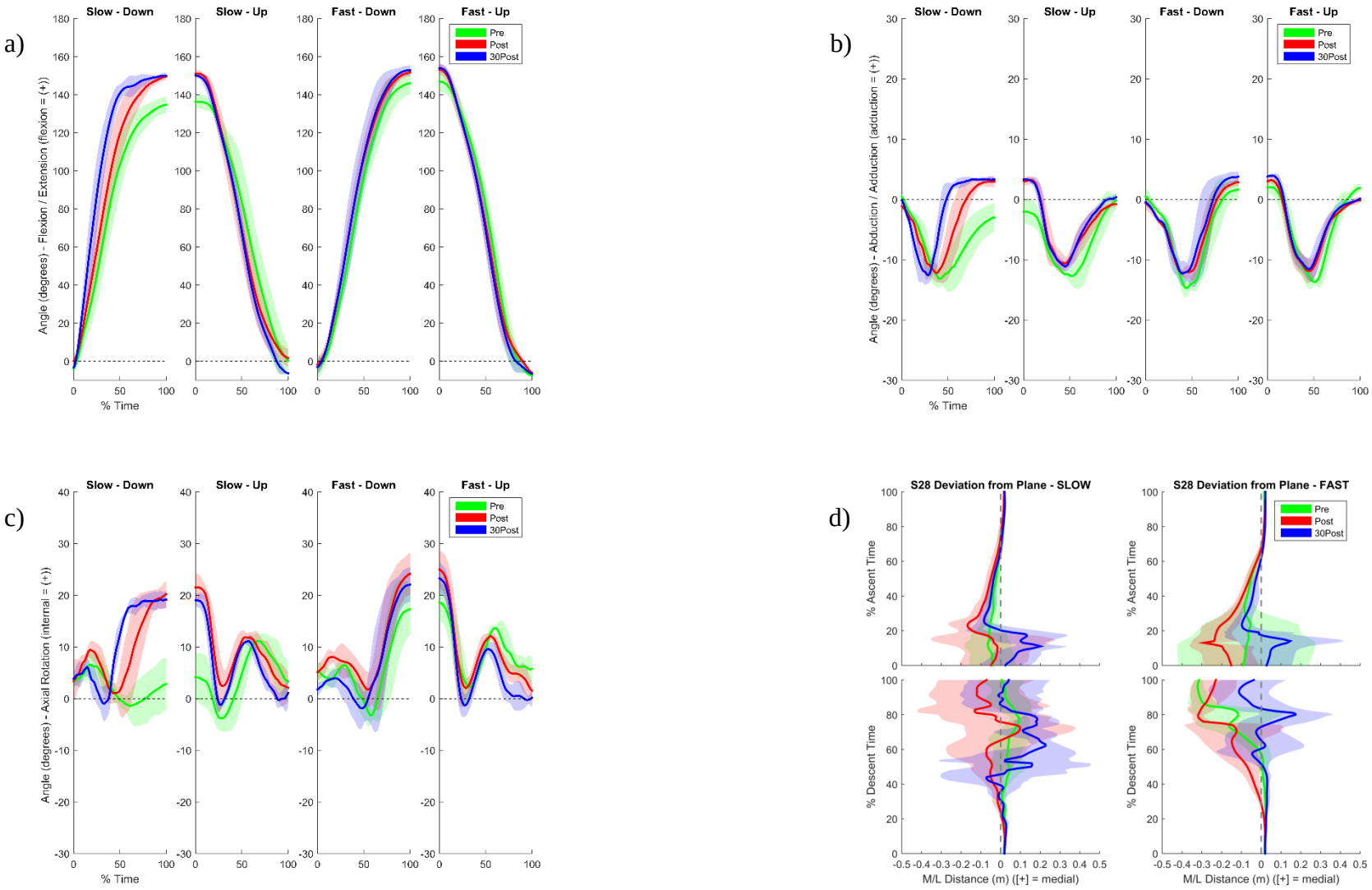


Figure K.22 S28 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

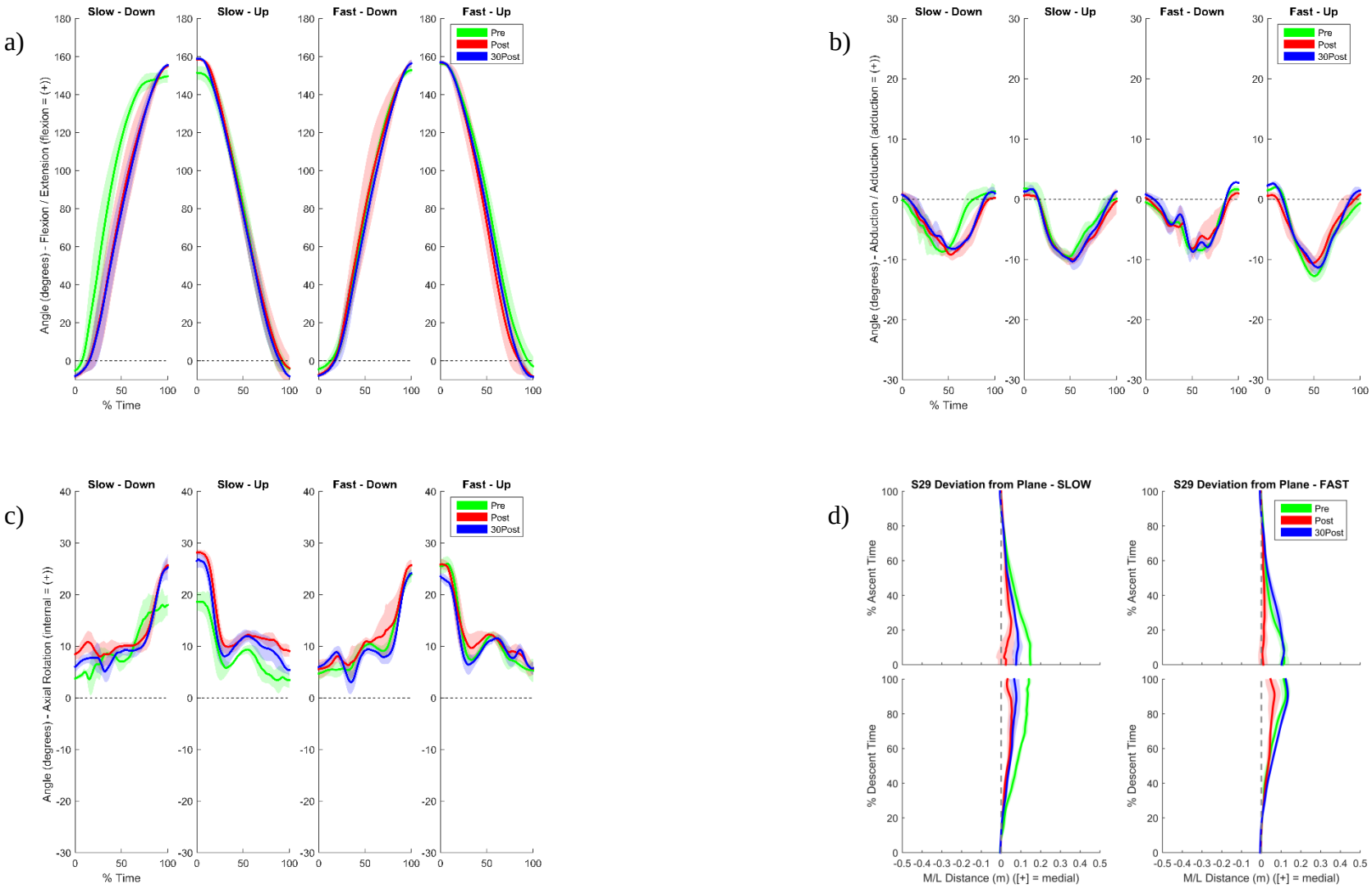


Figure K.23 S29 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

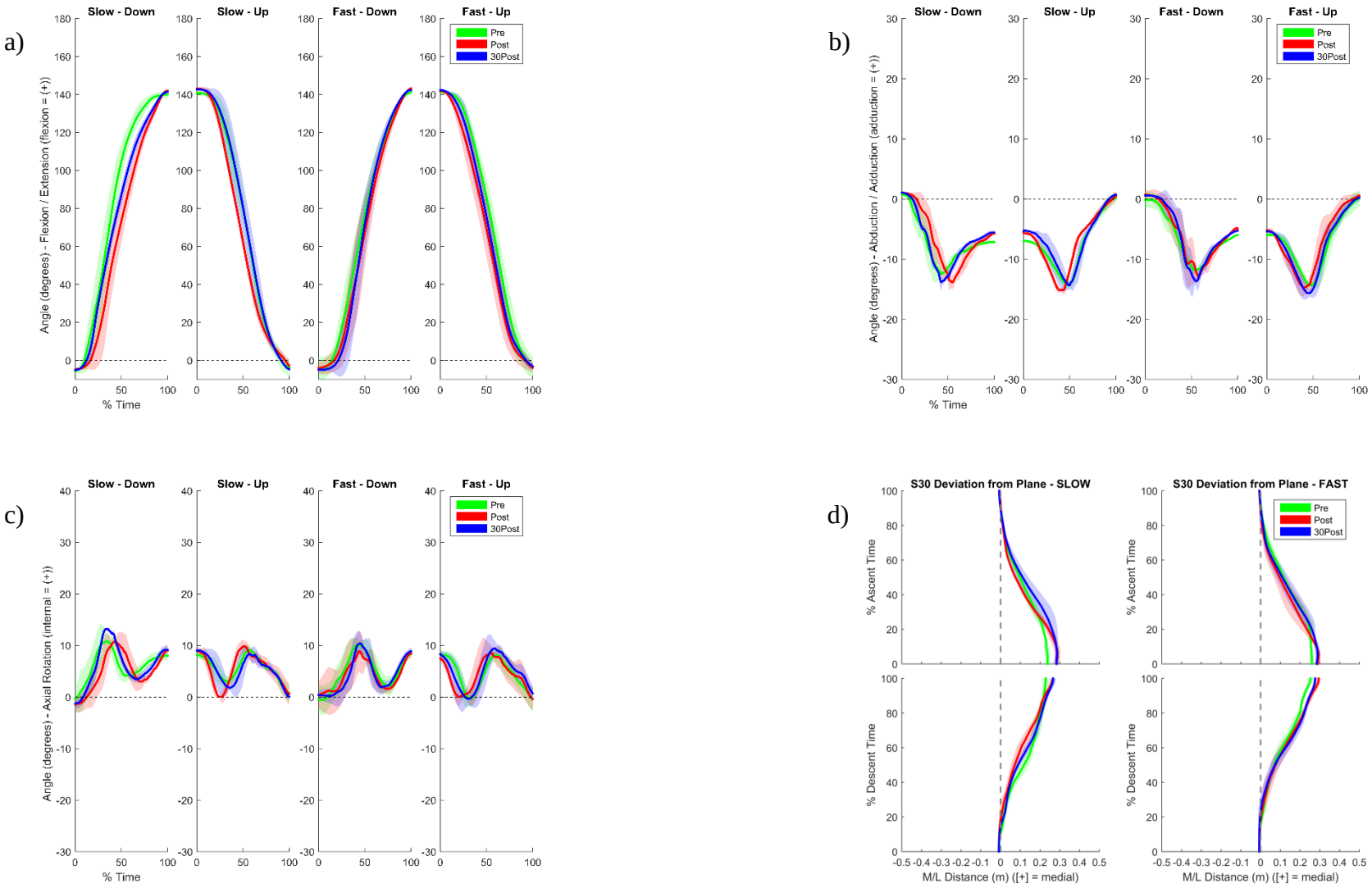


Figure K.24 S30 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

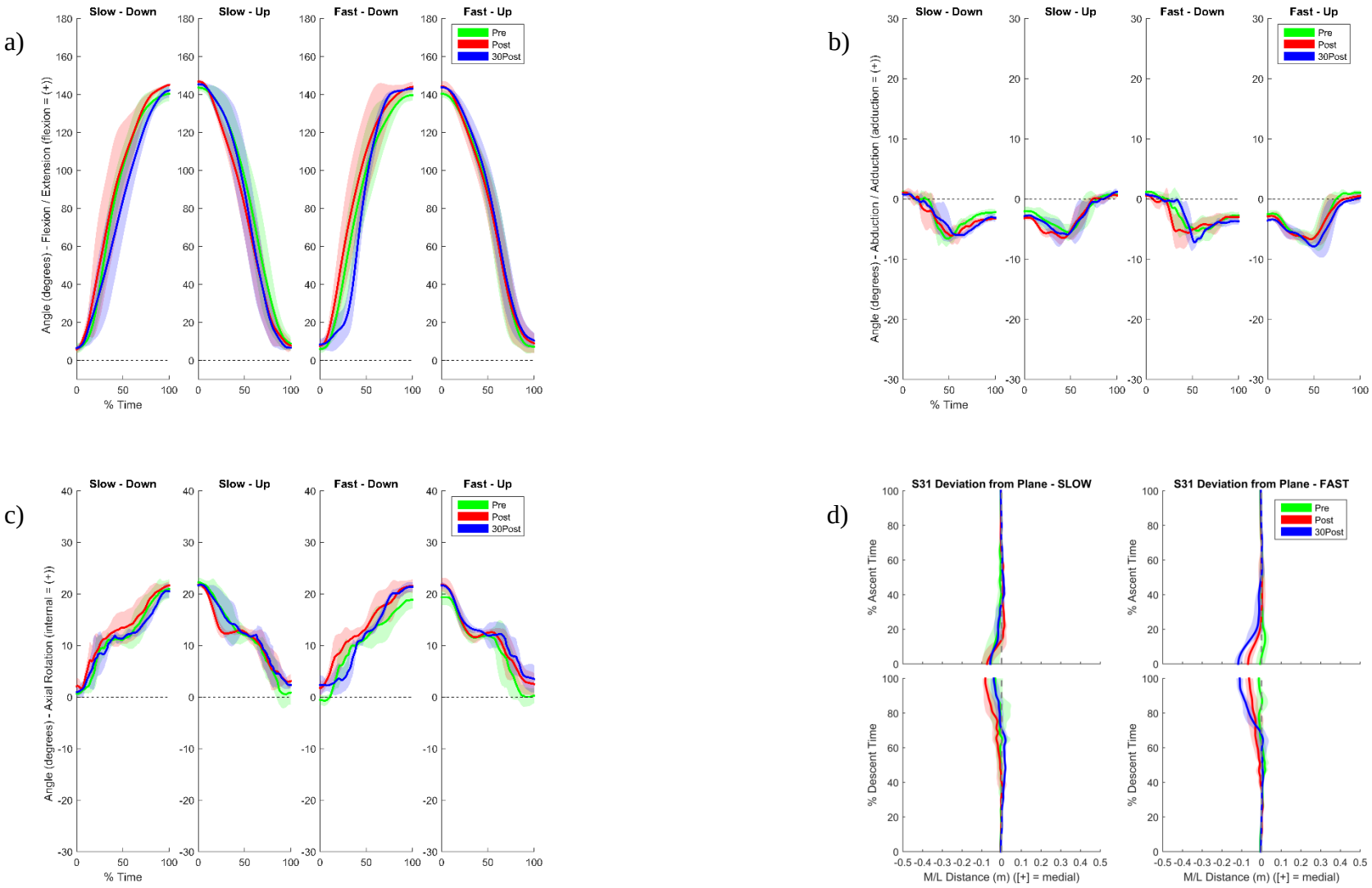


Figure K.25 S31 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

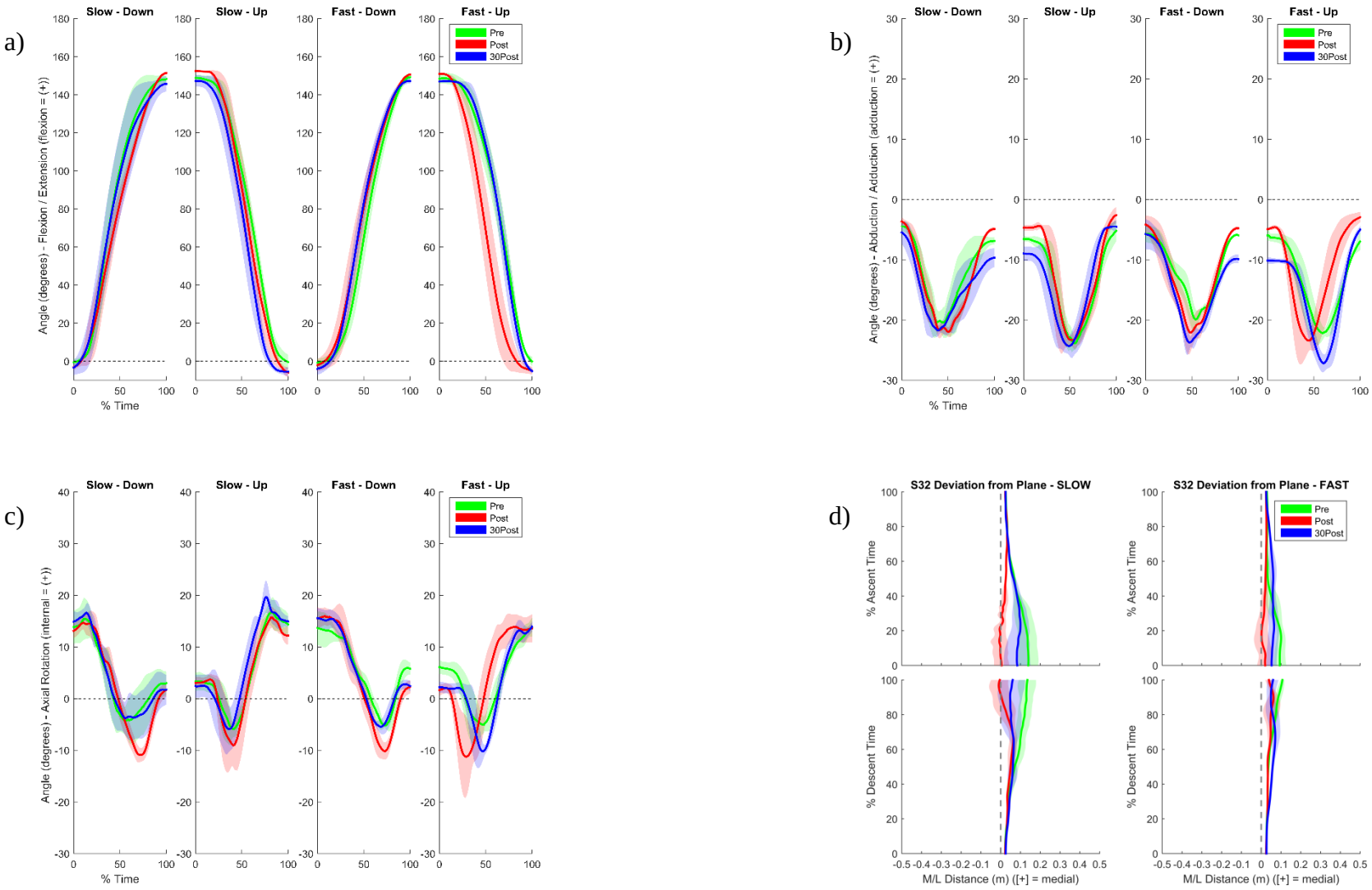


Figure K.26 S32 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

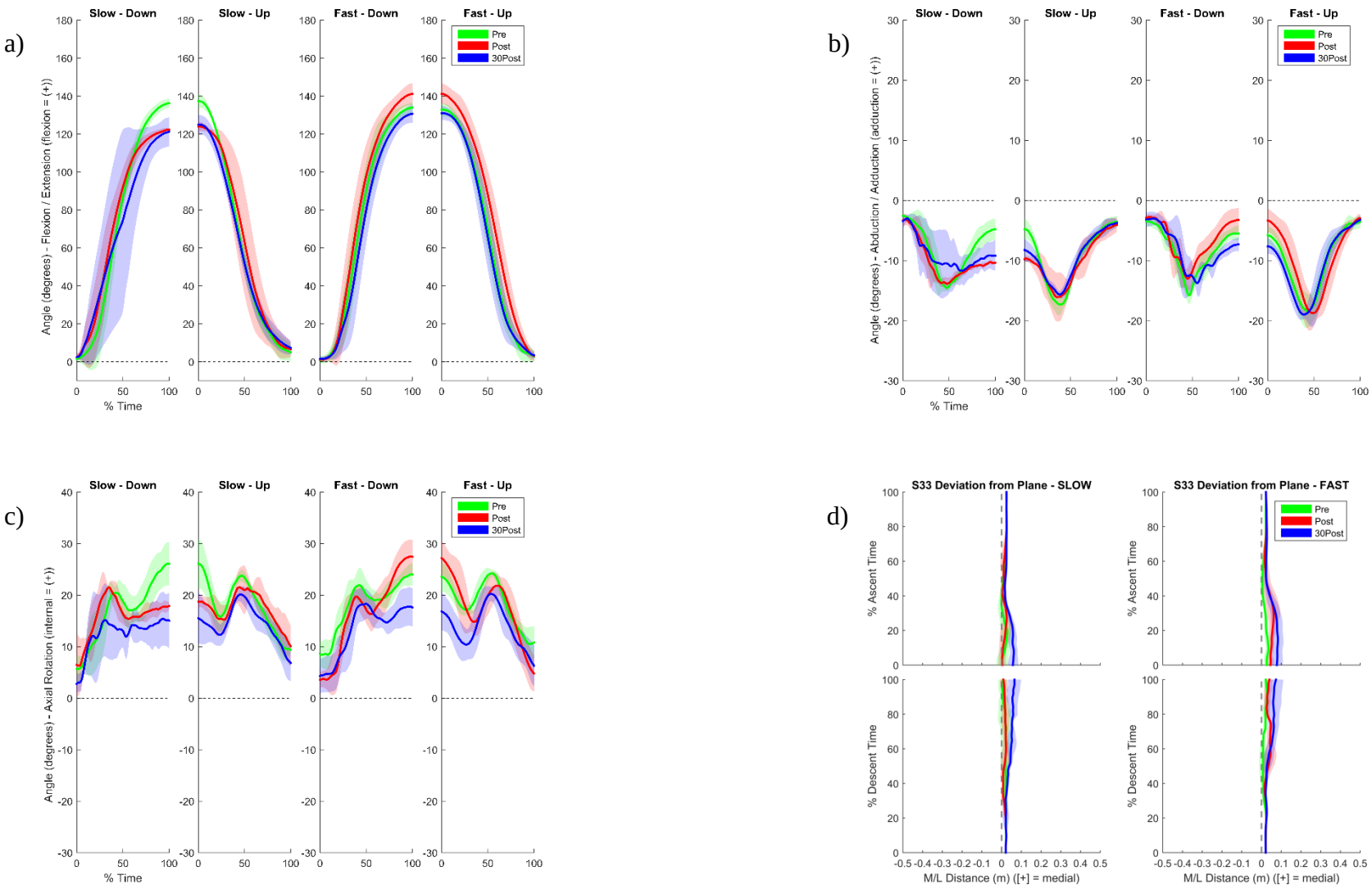


Figure K.27 S33 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

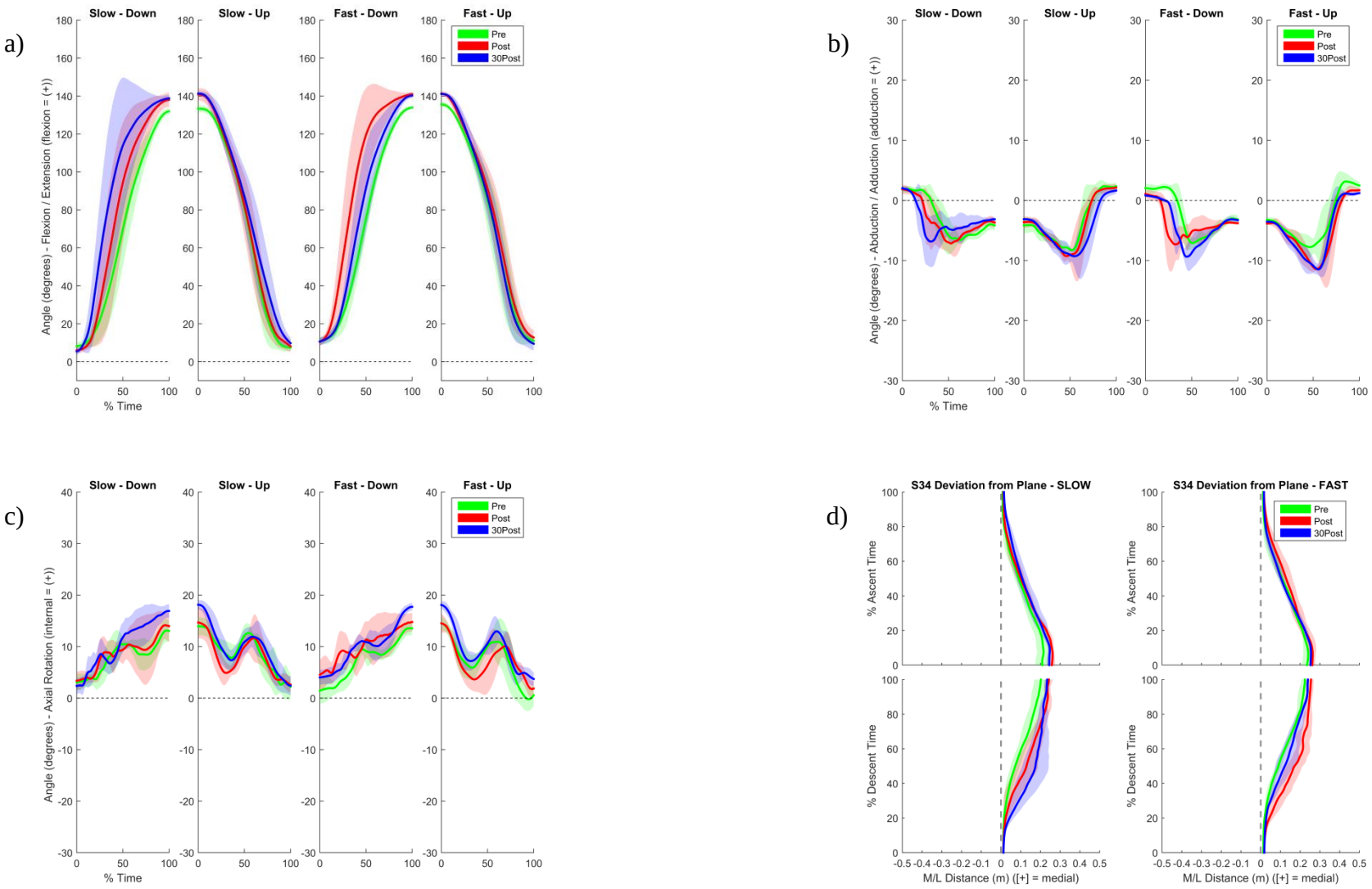


Figure K.28 S34 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

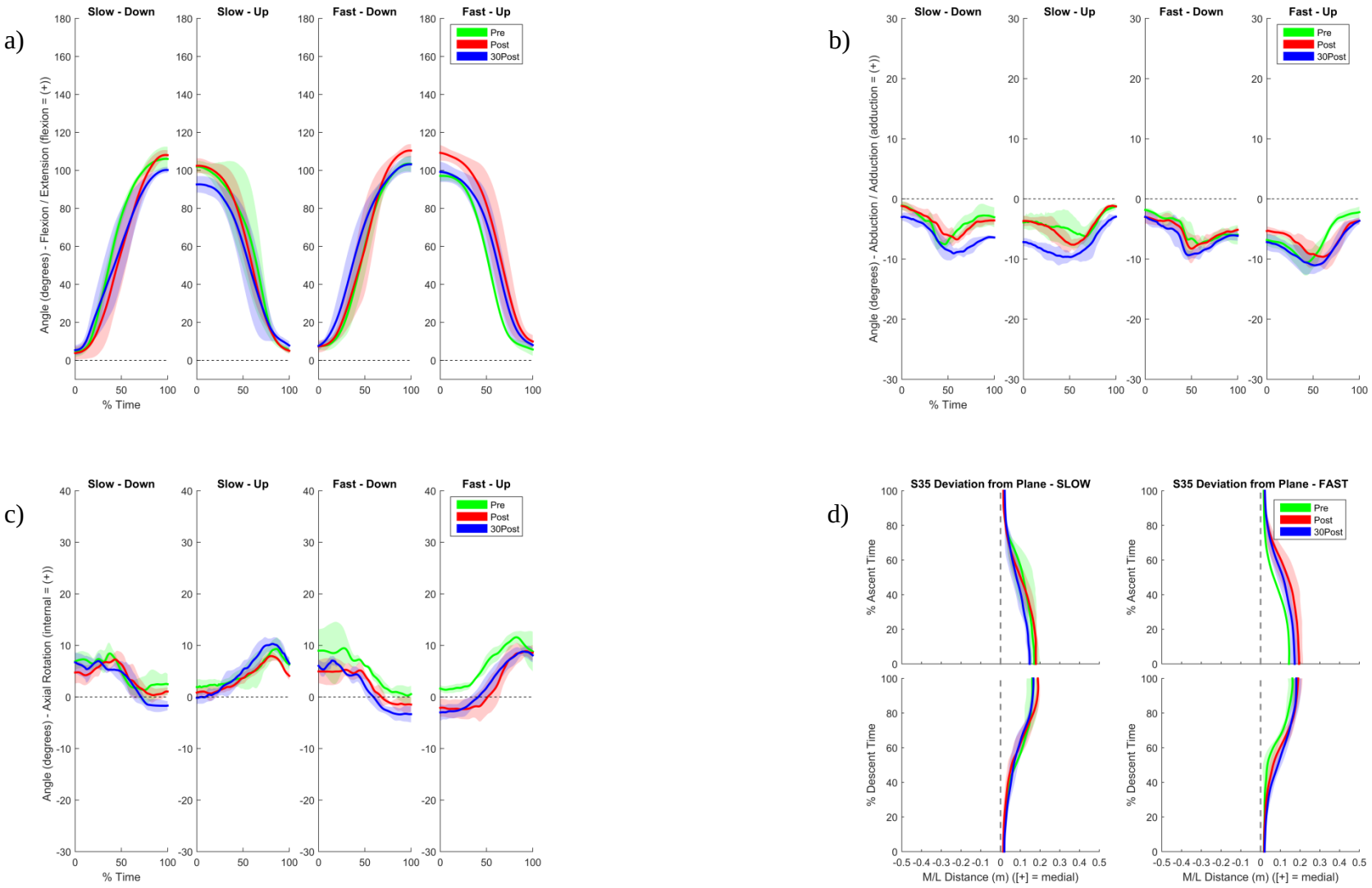


Figure K.29 S35 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

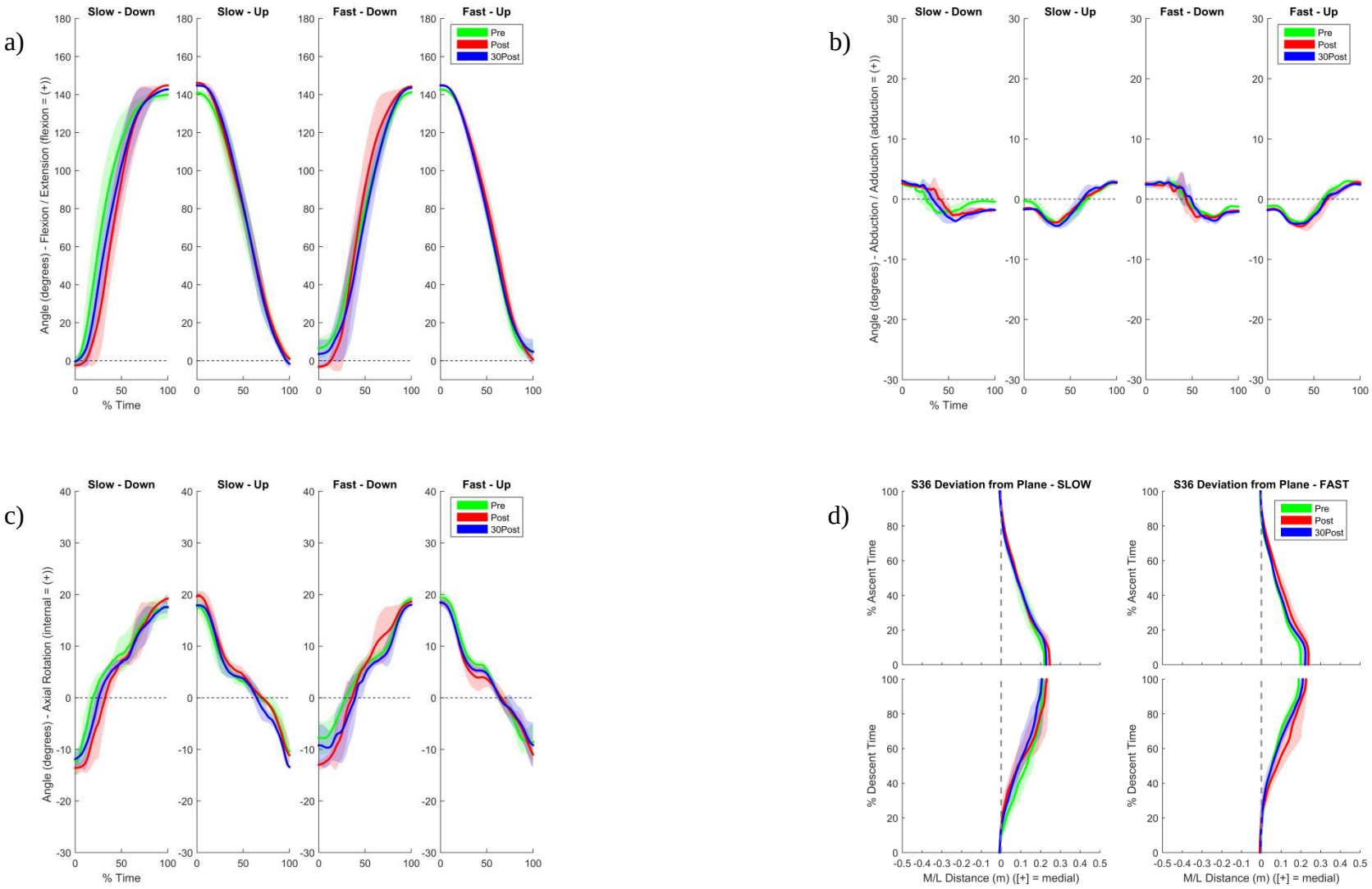


Figure K.30 S36 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

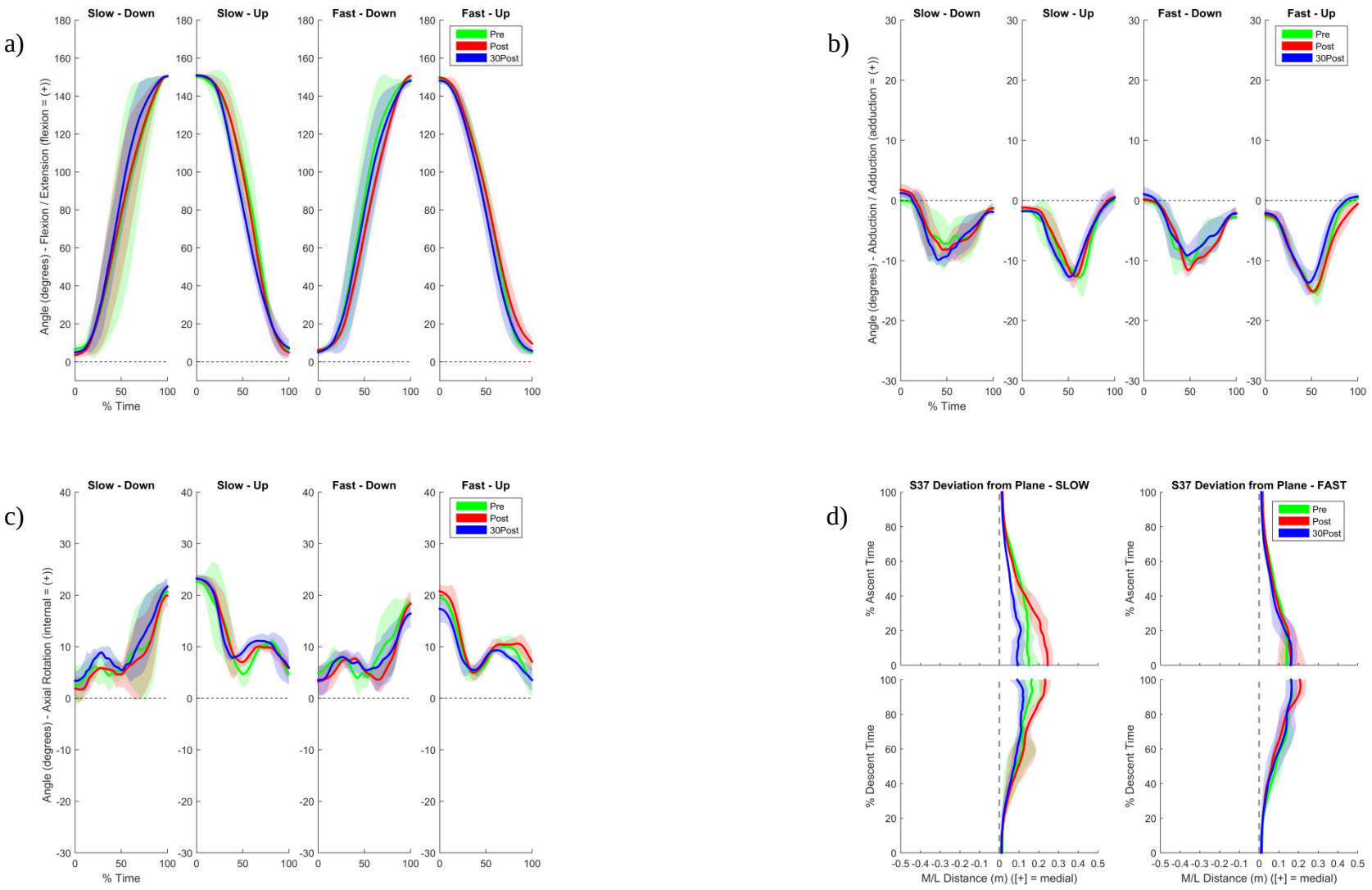


Figure K.31 S37 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

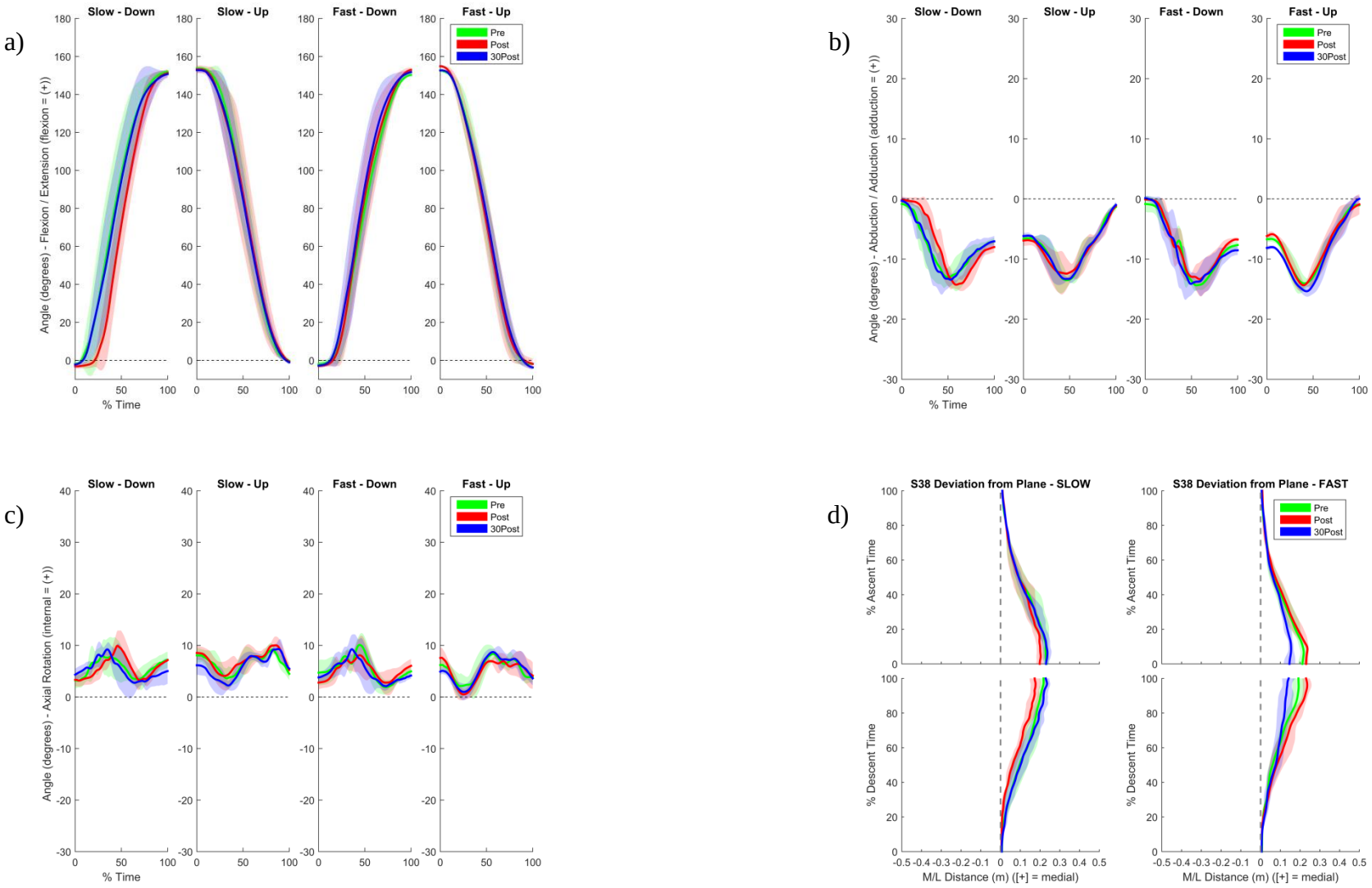


Figure K.32 S38 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

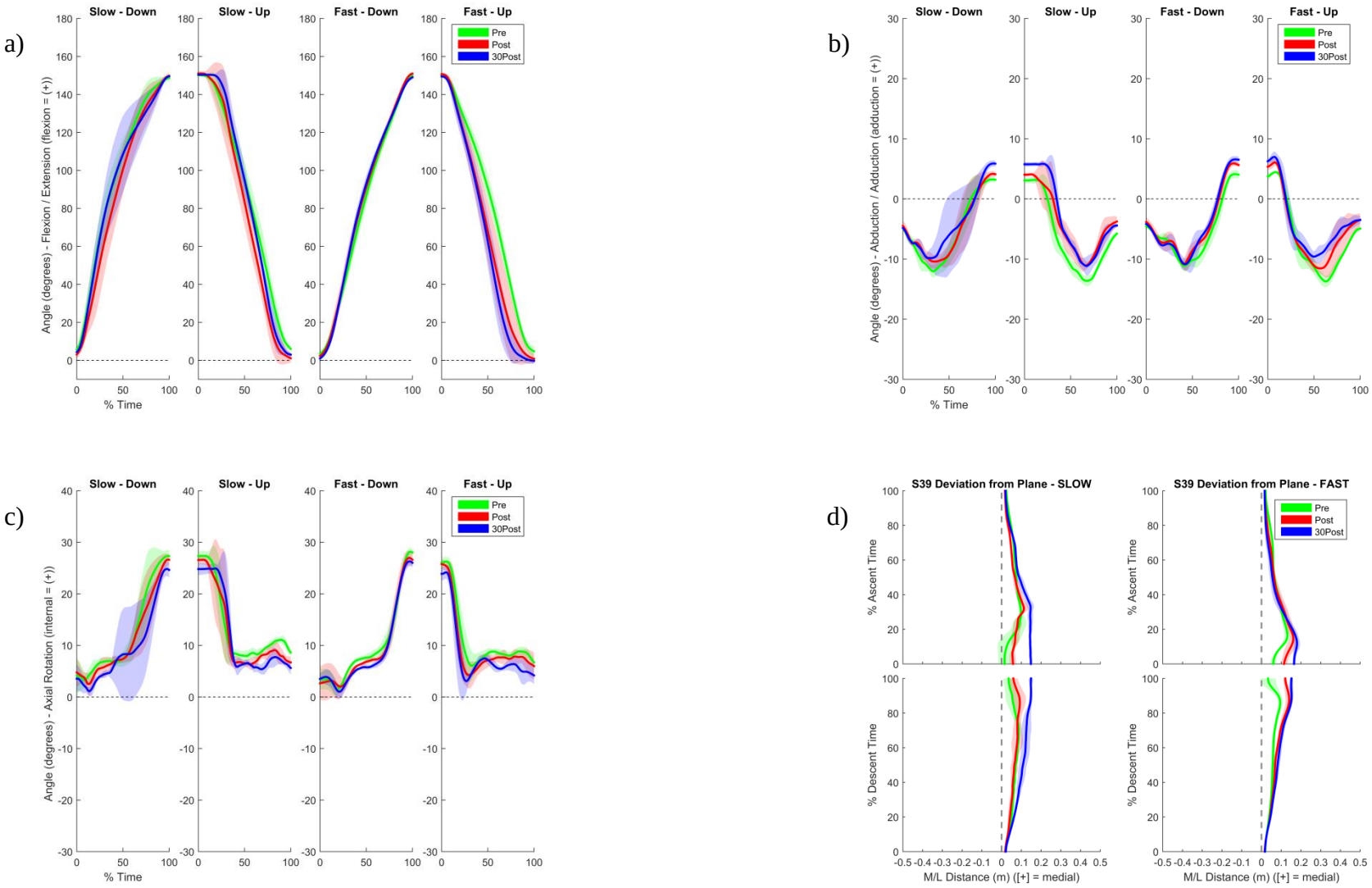


Figure K.33 S39 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

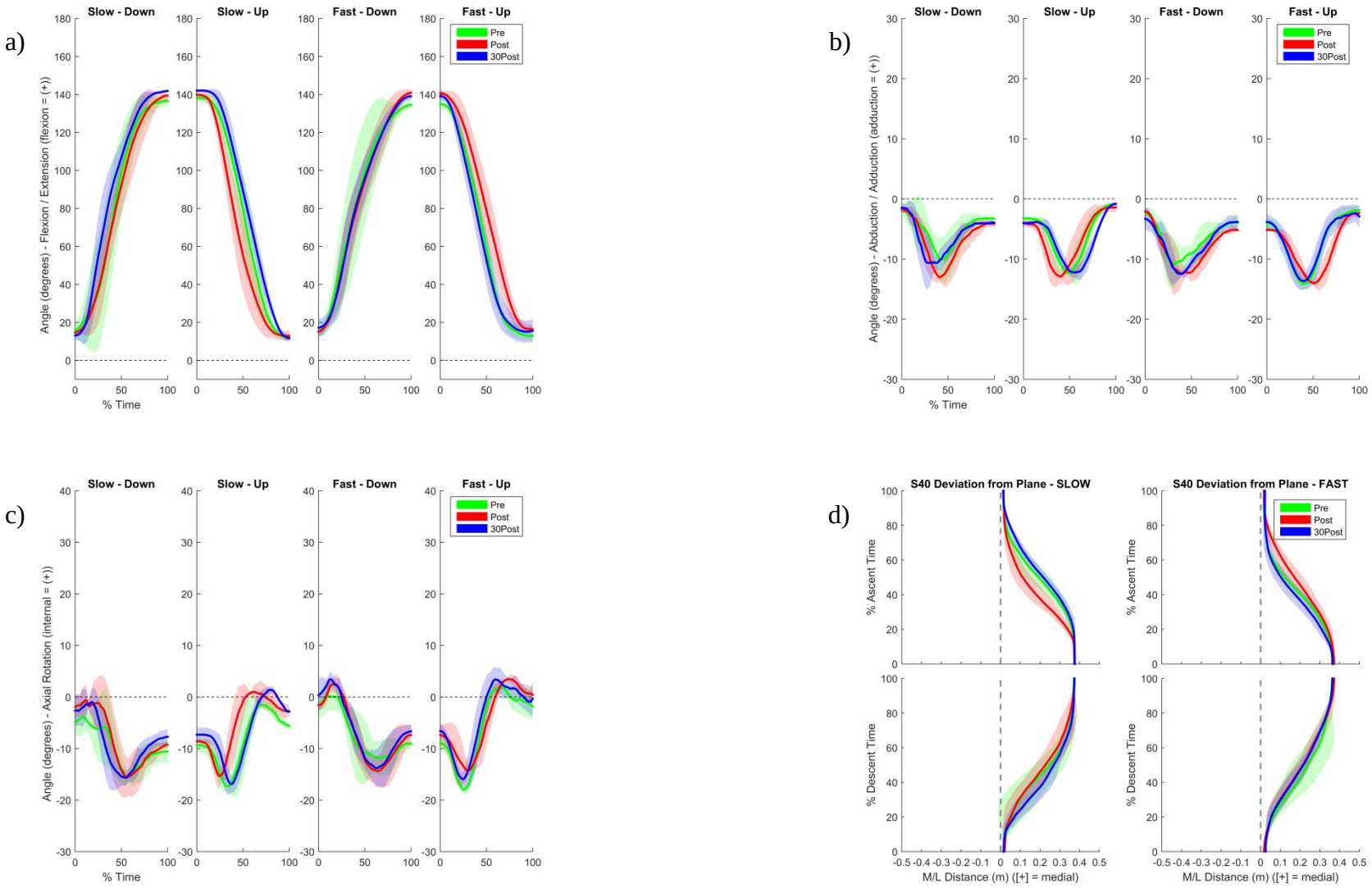


Figure K.34 S40 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

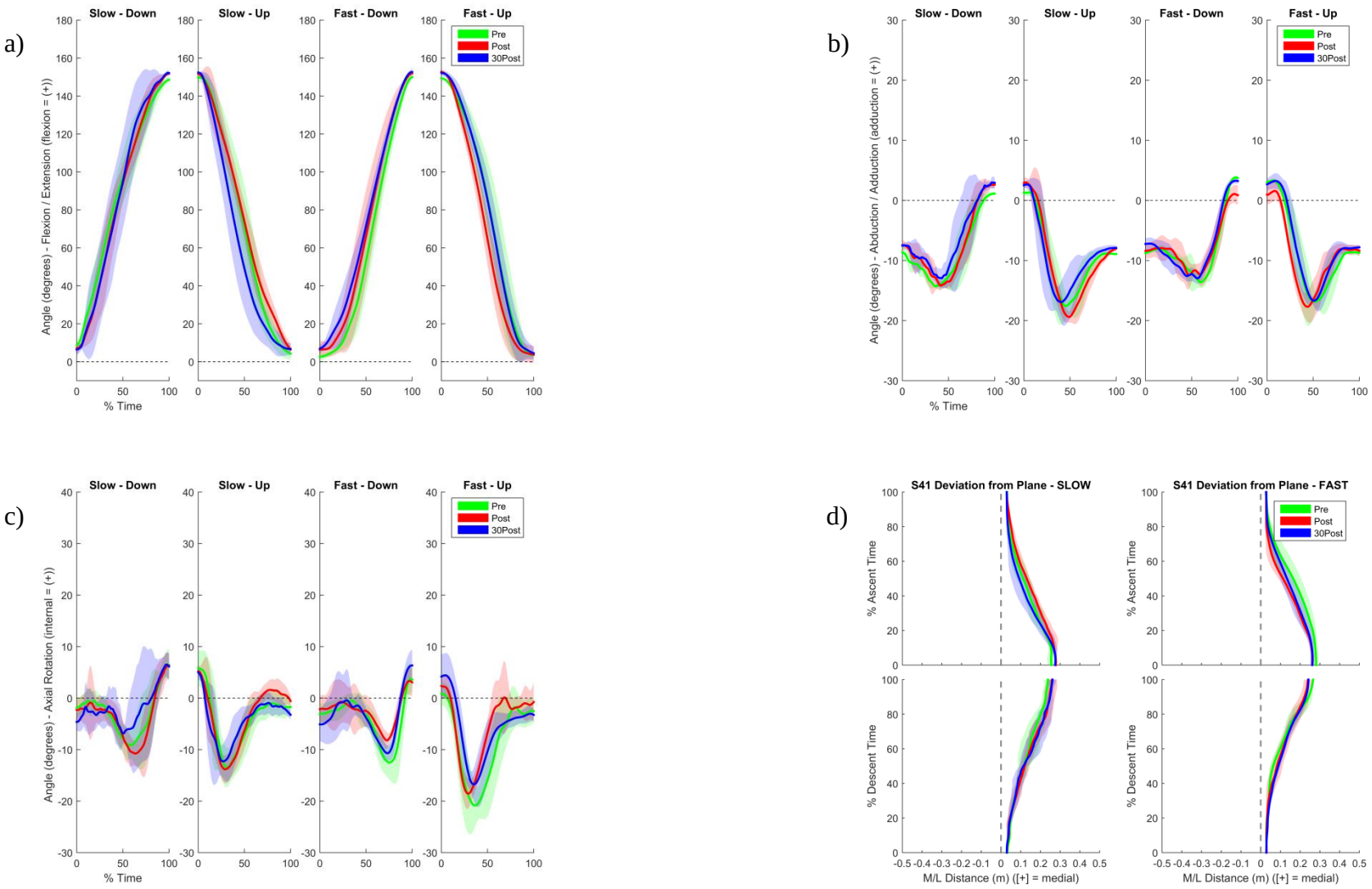


Figure K.35 S41 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

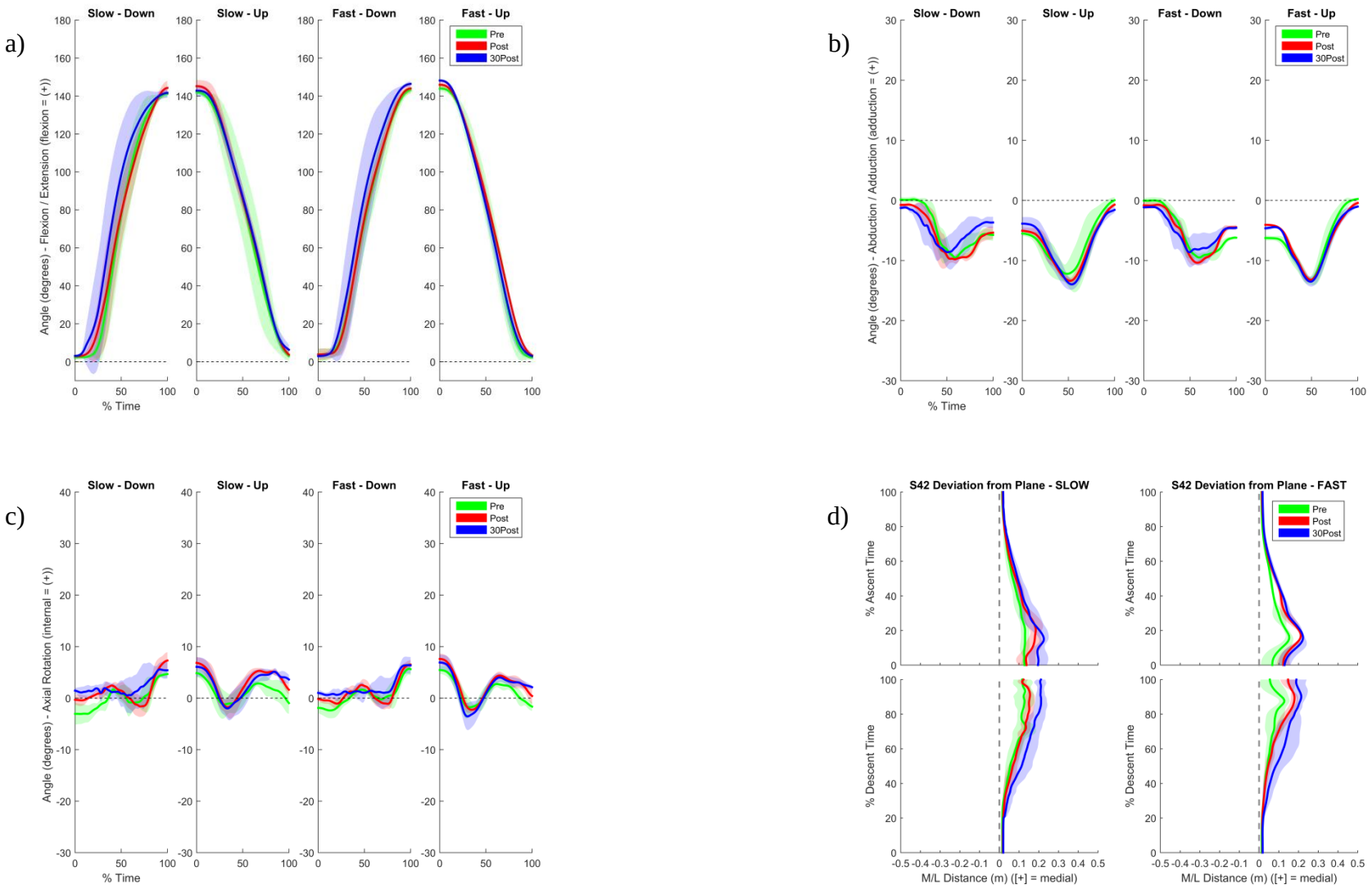


Figure K.36 S42 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

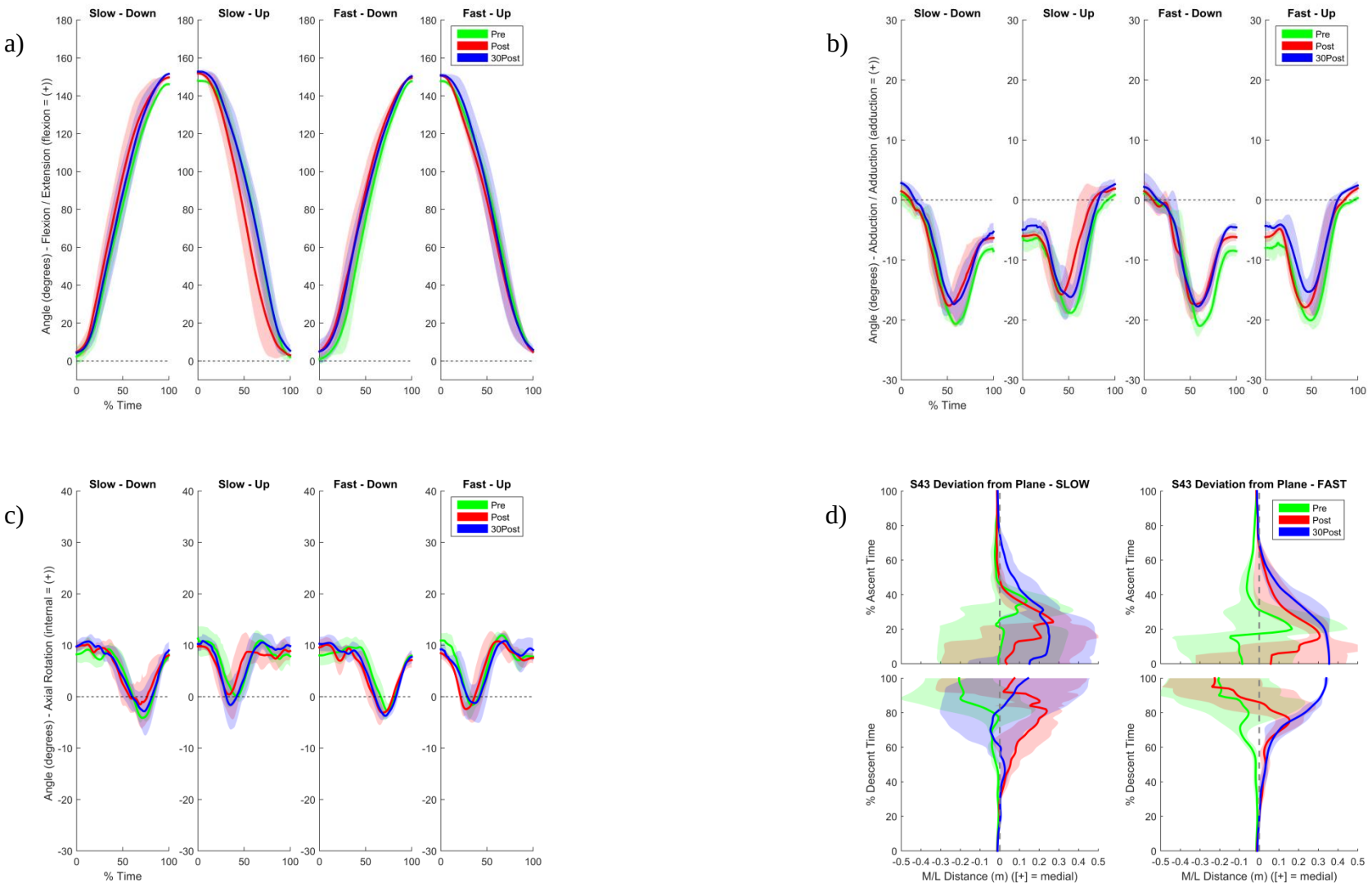


Figure K.37 S43 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

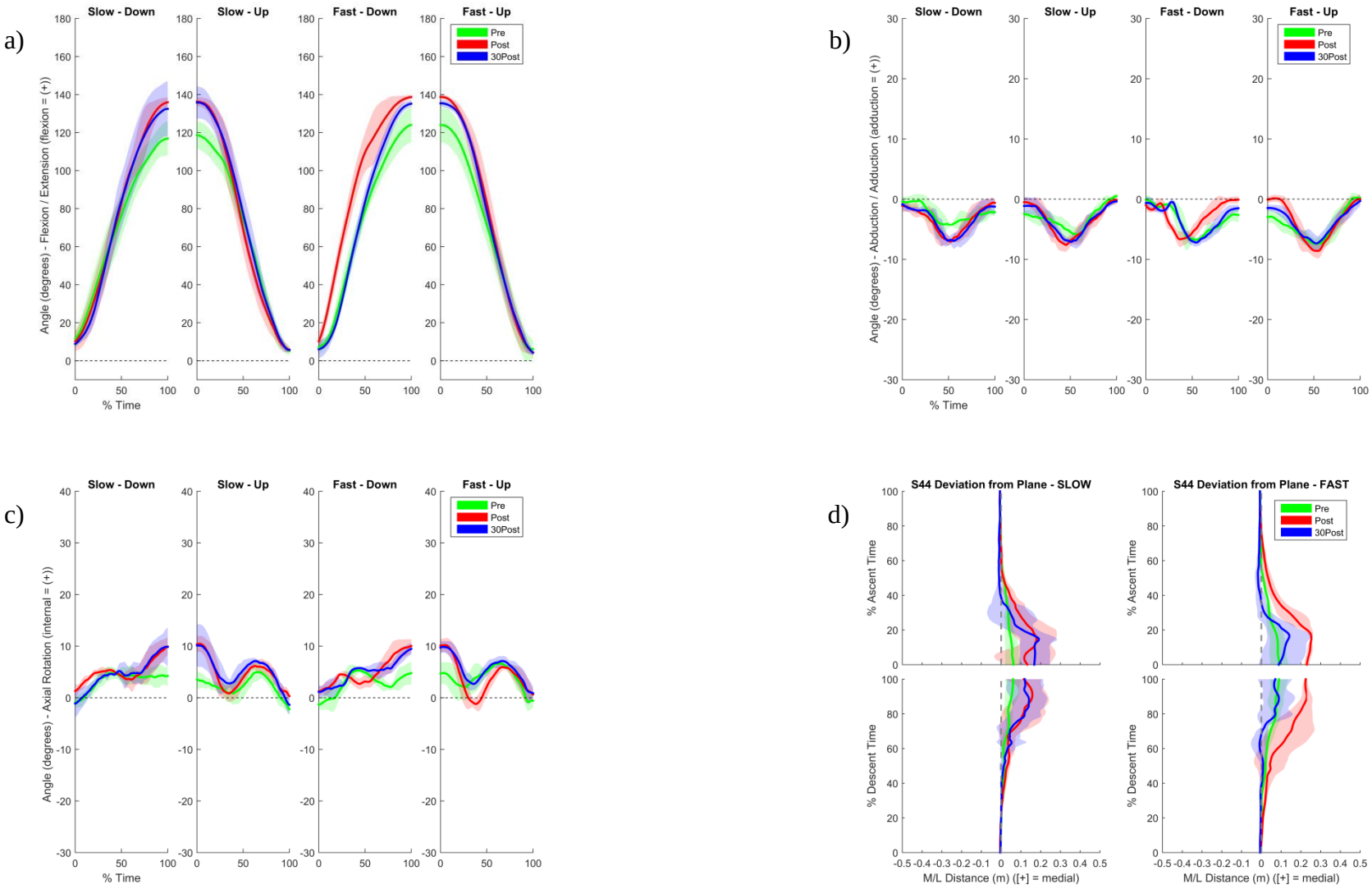


Figure K.38 S44 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

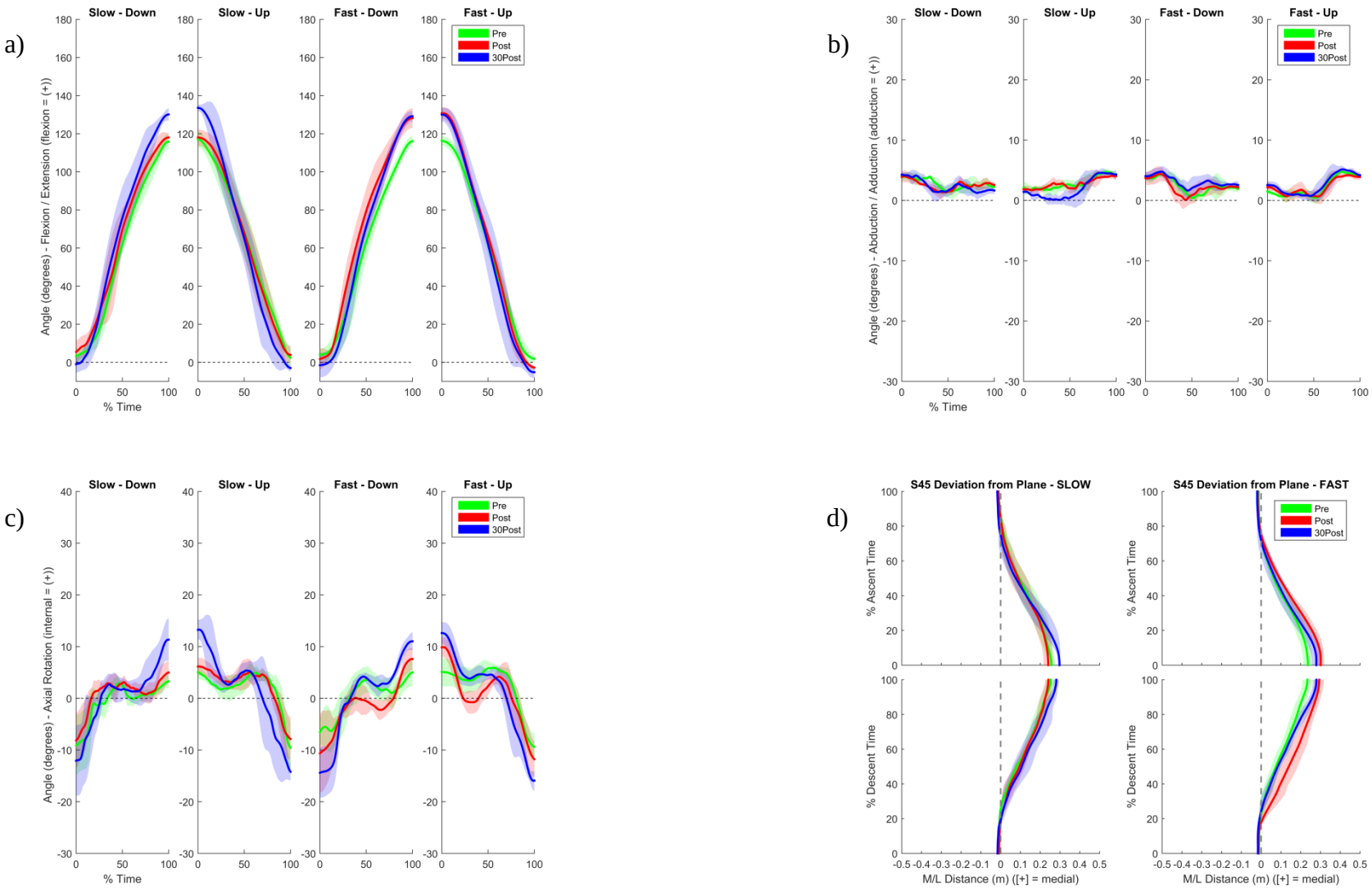


Figure K.39 S45 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

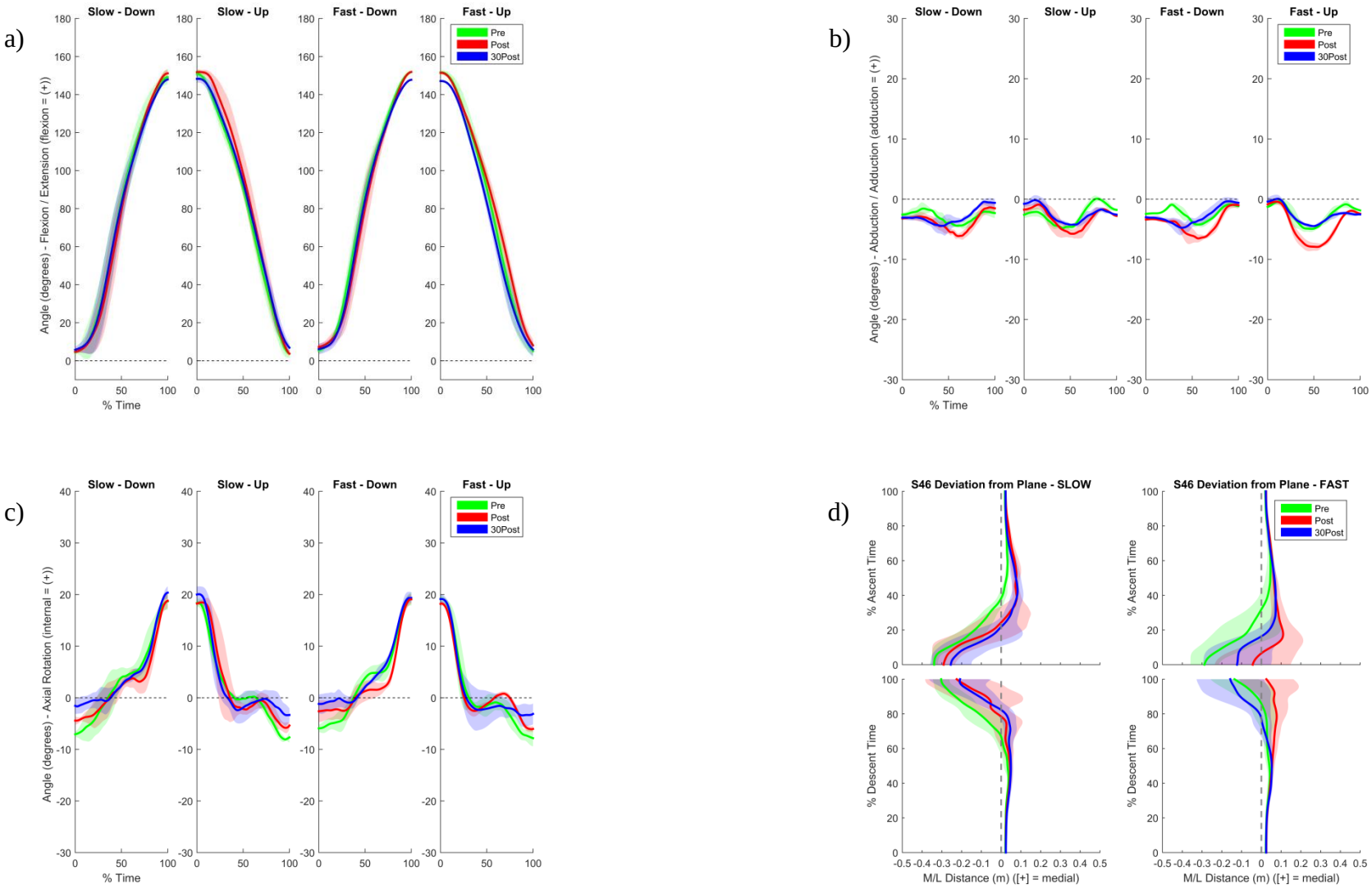


Figure K.40 S46 Frontal plane motion and tri-axial knee joint angles during the squat transition.

a) Flexion angle b) Abduction angle c) Axial rotation d) Knee joint deviation from the body-fixed plane. Shaded error bars indicate ± 1 SD.

Appendix L: Gait Kinetics and Kinematics

Previous work has shown that a 30-minute static kneeling exposure elicits changes in the external knee moments and kinematics during gait. The results from the current study evaluating a dynamic, more occupationally relevant 30-minute kneeling exposure do not support these findings (see Figures L.1 – L.3).

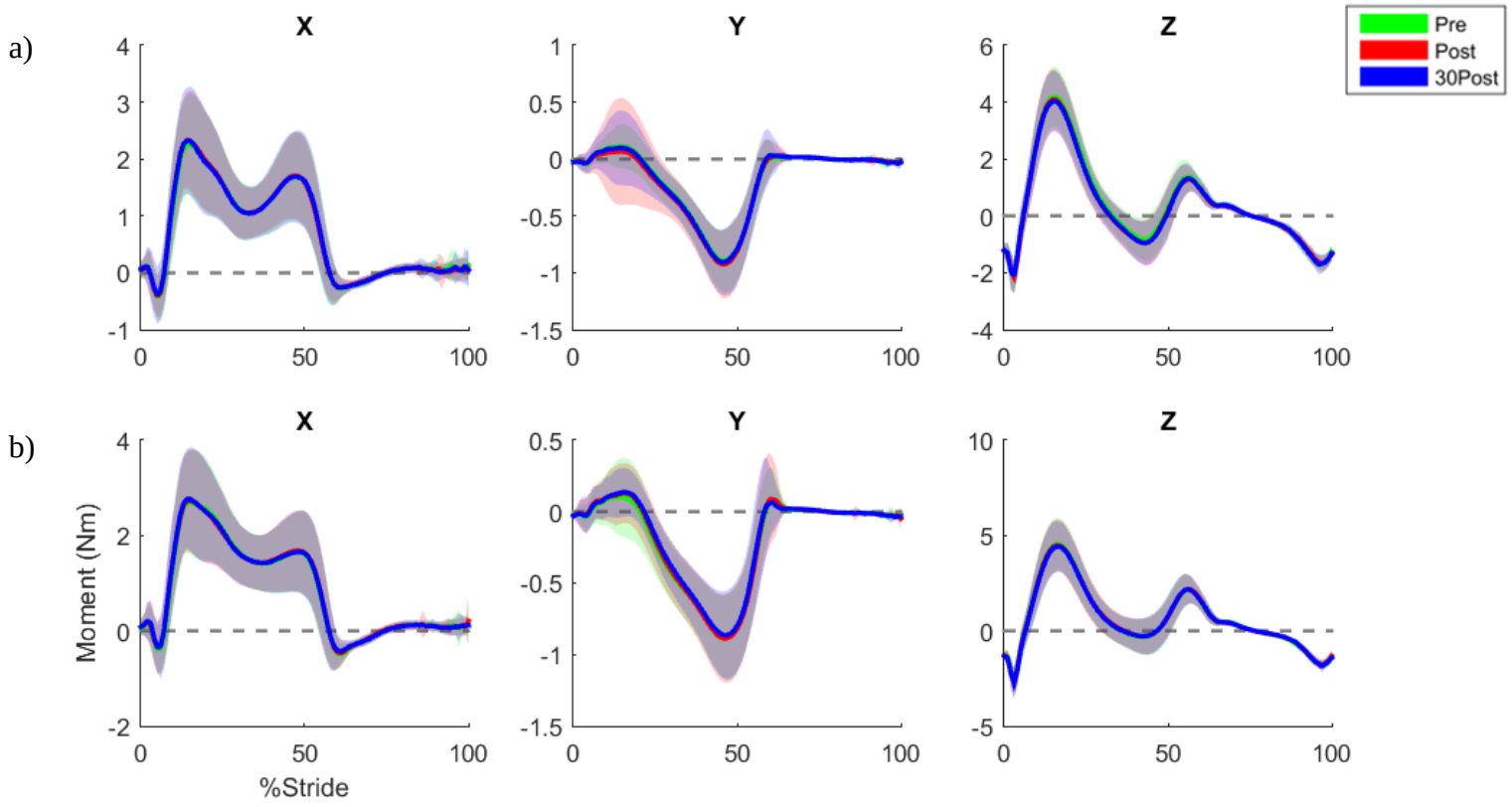


Figure L.1 Knee joint kinetics during gait in both the unloaded (a) and loaded (b) conditions.
NOTE: Moments are external: X: adduction (+), Y: internal rotation (+), Z: Flexion (+). Shaded error bars indicate ± 1 SD.

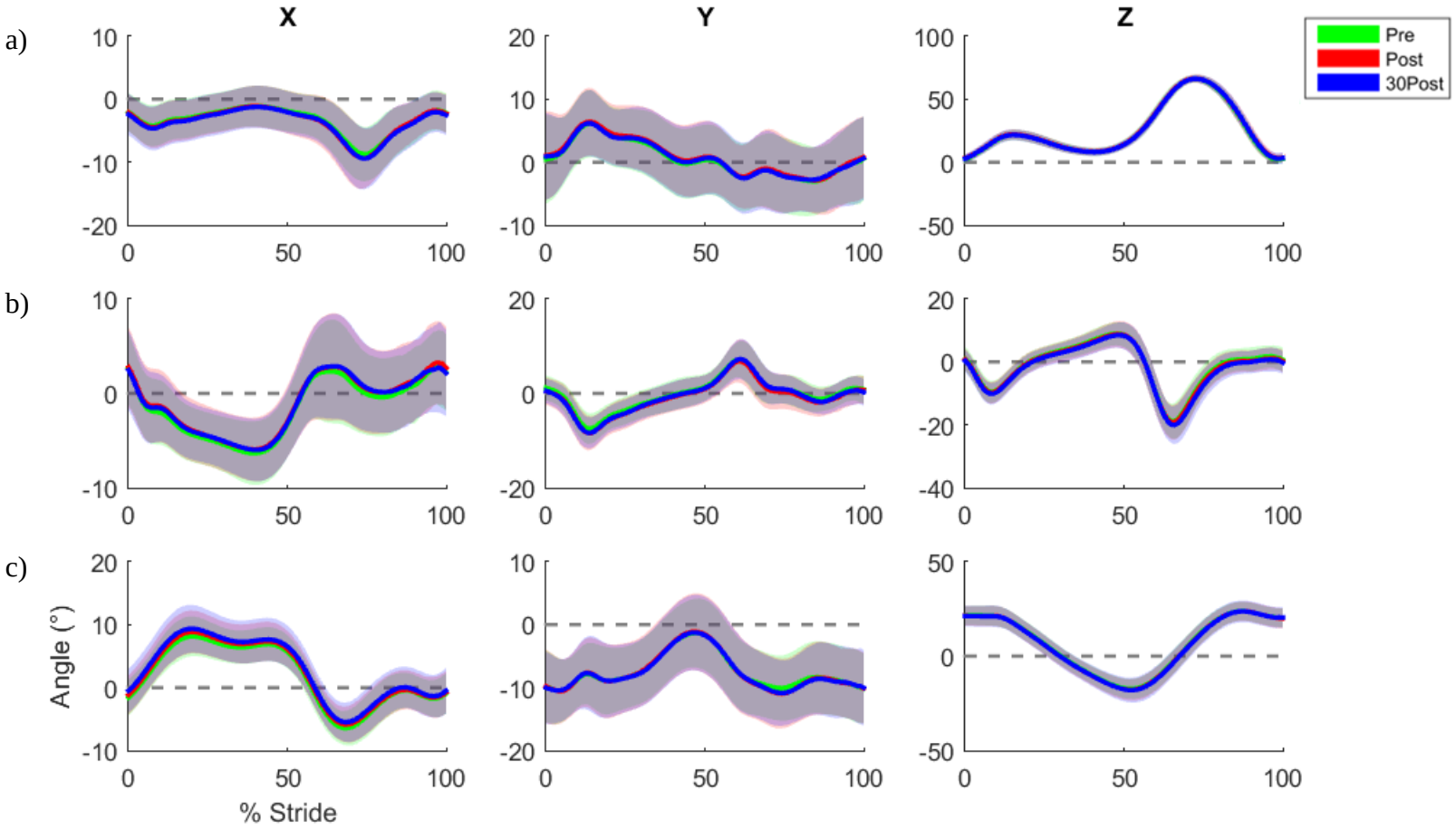


Figure L.2 Joint kinematics during gait in the *unloaded* condition for the knee (a), ankle (b), and hip (c). X: adduction (+), Y: internal rotation (+), Z: flexion (+). Note: for the ankle (b), positive flexion is plantarflexion. Shaded error bars indicate ± 1 SD.

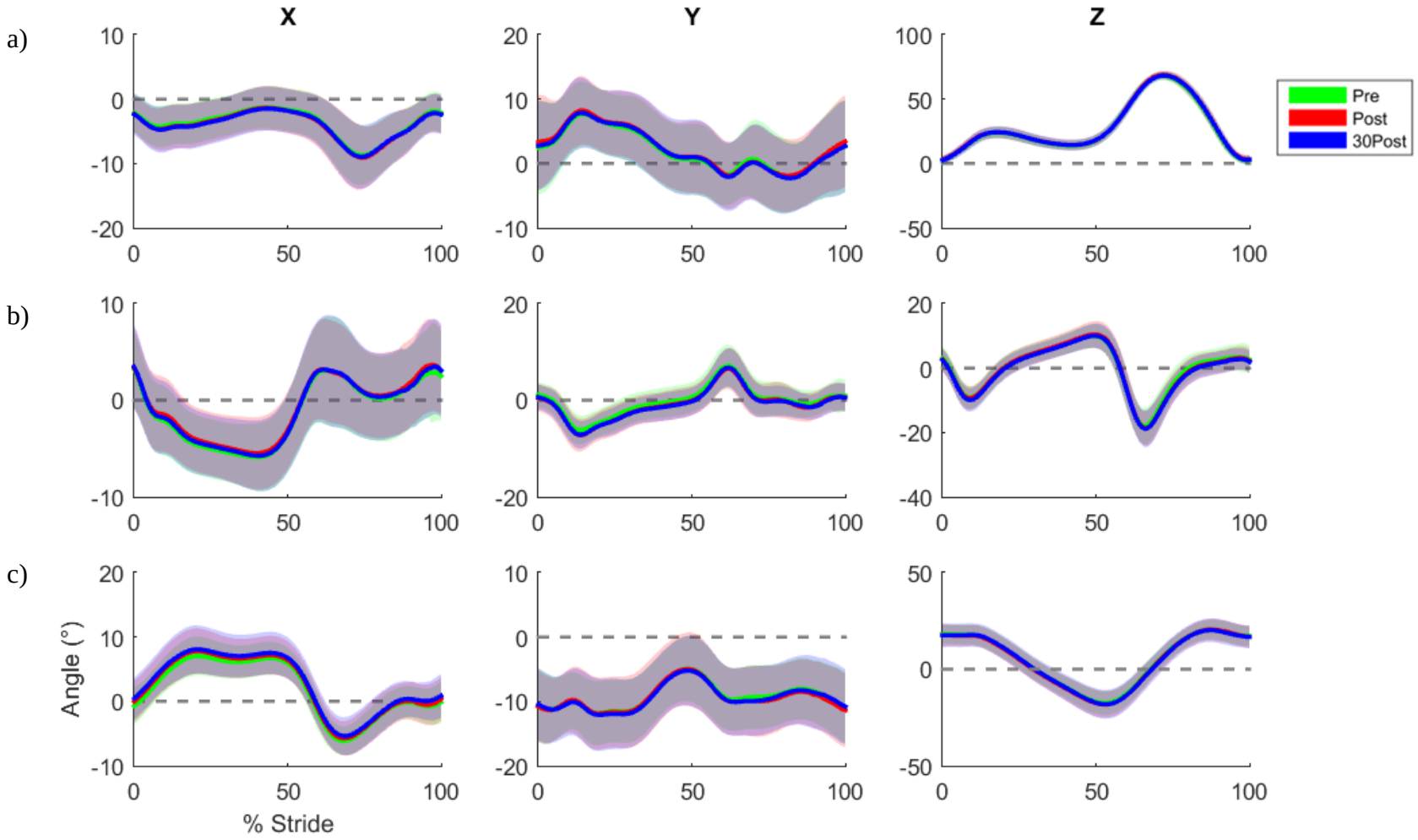


Figure L.3 Joint kinematics during gait in the *loaded* condition for the knee (a), ankle (b), and hip (c).
X: adduction (+), Y: internal rotation (+), Z: flexion (+). Note: for the ankle (b), positive flexion is plantarflexion. Shaded error bars indicate ± 1 SD.